







22101330387











THEORY AND PRACTICE OF MEDICINE



**BY THE SAME AUTHOR.**

Fcap. 8vo, 330 pp., 7s. 6d.

**NOTES ON MATERIA MEDICA AND PHARMACY.**

**\*\* A** concise text-book on Materia Medica and Pharmacy, chiefly intended for students preparing for examination in these subjects, but also useful for reference. The action and doses of drugs are merely mentioned, without any discussion of their therapeutic uses.

LONDON : H. K. LEWIS, 136, GOWER STREET, W.C.

A HANDBOOK  
OF THE  
THEORY AND PRACTICE  
OF  
MEDICINE

BY

FREDERICK T. ROBERTS, M.D., B.Sc., F.R.C.P.,


PROFESSOR OF MATERIA MEDICA AND THERAPEUTICS, AND OF CLINICAL MEDICINE, AT UNIVERSITY  
COLLEGE; PHYSICIAN TO UNIVERSITY COLLEGE HOSPITAL; PHYSICIAN TO BROMPTON HOSPITAL  
FOR CONSUMPTION AND DISEASES OF THE CHEST; EXAMINER IN MEDICINE AT THE ROYAL  
COLLEGE OF SURGEONS; EXAMINER IN MATERIA MEDICA AT THE UNIVERSITY OF  
LONDON, AND AT THE ROYAL COLLEGE OF PHYSICIANS, ETC., ETC.

SIXTH EDITION

LONDON  
H. K. LEWIS, 136 GOWER STREET

1885





Digitized by the Internet Archive  
in 2014

<https://archive.org/details/b20400111>

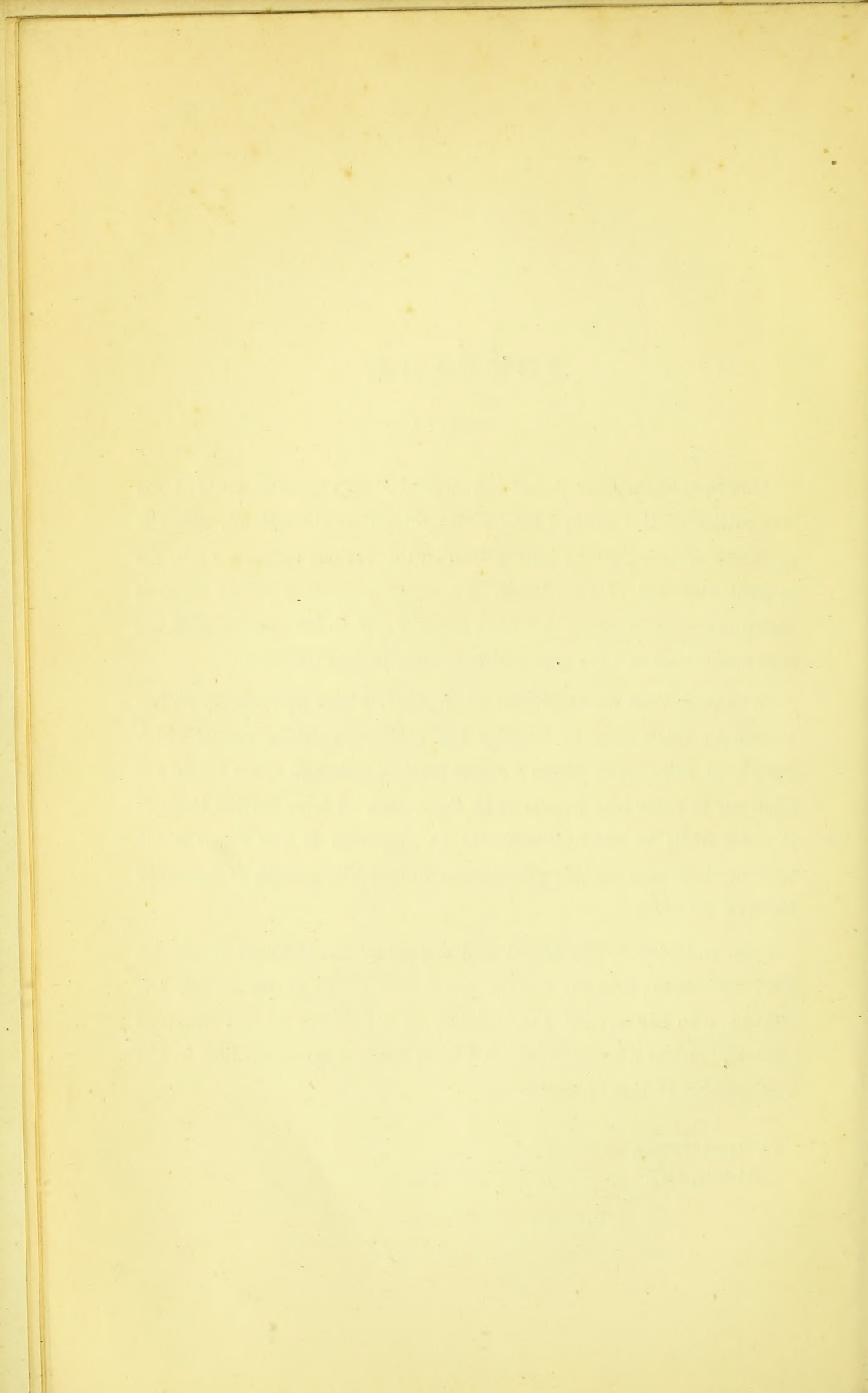
## PREFACE.

---

ALTHOUGH but a short period has elapsed since the publication of the last edition of this work, I have again revised it throughout, with the intention of bringing the information on the various subjects up to the present standard of knowledge. On many important points there is still much controversy, but I have endeavoured to incorporate with the text such views as have appeared to me deserving of notice.

In dealing with the treatment of diseases, I have particularly endeavoured in this edition to mention the principal remedial agents which have been introduced within a recent period, although space would not allow me to enter into details as to their uses. I have further thought it worth while to draw attention in an *Appendix* to two drugs which have attained considerable prominence during the passage of this work through the press.

I am indebted to my friend and colleague, Dr. Mitchell Bruce, for kind assistance; and my thanks are specially due to my friend, Dr. Beevor, who has revised the chapters on "Diseases of the Nervous System," and at whose suggestion I have made some alteration in the arrangement of these diseases.





# CONTENTS.

## SECTION I.

CHAP.	PAGE
I. Introduction—Objects and Methods of Study . . . . .	1
II. Ætiology or Causation of Disease . . . . .	4
III. Symptomatology or Semeiology—Mode of Invasion or Onset, Course, and Duration—Varieties and Types—Complications and Sequelæ—Terminations—Method and Objects of Clinical Examination—Diagnosis—Prognosis . . . . .	10
IV. Treatment or Therapeutics . . . . .	20

## SECTION II.

I. Hyperæmia or Congestion . . . . .	24
II. Dropsy—Hydrops . . . . .	28
III. Hæmorrhage—Hæmophilia—Hæmorrhagic Diathesis . . . . .	35
IV. Inflammation . . . . .	42
V. Alterations in Growth—Hypertrophy—Atrophy . . . . .	61
VI. Degenerations—Fatty—Mineral or Calcareous—Fibroid—Pigmentary—Mucoid—Colloid—Albuminoid or Lardaceous Disease . . . . .	65

## SECTION III.

### I. GENERAL DISEASES.

#### A. IDIOPATHIC FEVERS AND ALLIED DISEASES.

I. Fever or Pyrexia . . . . .	78
II. On Contagion and Epidemics—Hygienic Management of Contagious Fevers—Prevention and Limitation of Epidemics . . . . .	94
III. On the Clinical Investigation of Acute Febrile Diseases—On the Use of the Thermometer . . . . .	106
IV. Simple Continued Fever—Febricula—Synocha . . . . .	113
V. Typhus Fever . . . . .	114
VI. Typhoid or Enteric Fever—Pythogenic Fever—Abdominal Typhus . . . . .	122
VII. Relapsing Fever—Famine Fever . . . . .	135
VIII. Scarletina—Scarlet Fever . . . . .	140
IX. Rubella—Morbilli—Measles . . . . .	148
X. Röteln—Rubella Notha—Epidemic Roseola—German Measles . . . . .	152
XI. Variola—Small-pox . . . . .	154
XII. Vaccinia—Cow-pox . . . . .	165
XIII. Varicella—Chicken-pox . . . . .	169
XIV. Erysipelas—The Rose—St. Anthony's Fire . . . . .	170

CHAP.		PAGE
XV.	Diphtheria . . . . .	175
XVI.	Mumps—Idiopathic Parotitis . . . . .	186
XVII.	Whooping- or Hooping-cough—Pertussis . . . . .	187
XVIII.	Influenza—Epidemic Catarrh . . . . .	191
XIX.	Epidemic or Asiatic Cholera—Cholera Morbus . . . . .	194
XX.	Glanders and Farcy—Equinia . . . . .	204
XXI.	Malignant Pustule—Anthrax—Charbon . . . . .	207
XXII.	Hydrophobia—Rabies . . . . .	210
XXIII.	Dengue—Dandy Fever—Breakbone Fever . . . . .	213
XXIV.	Pestis—Plague . . . . .	214
XXV.	Yellow Fever . . . . .	216
XXVI.	Cerebro-Spinal Fever—Epidemic Cerebro-Spinal Meningitis . . . . .	219
XXVII.	Malarial or Paludal Fevers—On Malarial or Marsh-Miasm—Intermittent Fever or Ague—Remittent Fever . . . . .	222
XXVIII.	On the Diagnosis of Acute Specific Diseases . . . . .	231

B. CONSTITUTIONAL DISEASES.

XXIX.	Rheumatism—Acute Articular Rheumatism—Rheumatic Fever—Chronic Articular Rheumatism—Muscular and Tendinous Rheumatism or Myalgia—Gonorrhœal Rheumatism—Rheumatoid Arthritis . . . . .	240
XXX.	Gout—Podagra . . . . .	253
XXXI.	Scurbutus—Scurvy . . . . .	263
XXXII.	Purpura . . . . .	267
XXXIII.	Rachitis—Rickets . . . . .	269
XXXIV.	Constitutional Syphilis . . . . .	281
XXXV.	Tuberculosis—Scrofulosis . . . . .	287
XXXVI.	Carcinoma—Cancer—Malignant Disease . . . . .	298
XXXVII.	Diabetes Mellitus—Glycosuria—Diabetes Insipidus—Polyuria—Polydipsia . . . . .	303

II. LOCAL DISEASES.

I.	Diseases of the Mouth, Tongue, and Salivary Glands—Stomatitis— Glossitis—Ulcers of the Mouth and Tongue—Symptomatic Parotitis or Parotid Bubo—Chronic Enlargement of the Parotid—Salivation or Ptyalism . . . . .	314
II.	Diseases of the Throat—Acute Inflammatory Affections—Ulcerations— Chronic Affections—Retro-pharyngeal Abscess . . . . .	324
III.	Diseases of the Œsophagus—Acute Œsophagitis—Chronic Diseases of the Œsophagus . . . . .	334
IV.	Diseases of the Respiratory Organs—Clinical Phenomena—Physical Ex- amination—Of the Larynx and Trachea—Of the Chest . . . . .	341
V.	On Respiratory Symptoms—Dyspnoea—Apnoea—Cough—Hæmoptysis . . . . .	369
VI.	Catarrh—Coryza . . . . .	374
VII.	Diseases of the Larynx and Trachea—Acute Laryngitis and Tracheitis— Chronic Laryngitis or Laryngeal Catarrh—Ulcers of the Larynx— Morbid Growths—Œdema Glottidis—Laryngeal Perichondritis— Abscess—Necrosis of the Cartilages—Functional or Nervous Affections . . . . .	376
VIII.	Diseases of the Bronchi—Acute Catarrhal Bronchitis—Chronic Bronchial Catarrh—Plastic or Croupous Bronchitis—Dilatation of the Bronchi— Bronchiectasis . . . . .	393

CHAP.		PAGE
IX.	Pulmonary Congestion—Œdema—Hæmorrhage . . . . .	403
X.	Pneumonia or Inflammation of the Lungs—Acute Croupous Pneumonia —Catarrhal Pneumonia—Broncho-pneumonia—Chronic or Interstitial Pneumonia . . . . .	407
XI.	Gangrene of the Lungs . . . . .	418
XII.	Emphysema of the Lungs—Vesicular—Interlobular or Interstitial . . . . .	420
XIII.	Asthma—Bronchial Asthma—Diaphragmatic Asthma . . . . .	426
XIV.	Atelectasis—Pulmonary Collapse and Compression . . . . .	432
XV.	Phthisis or Pulmonary Consumption—Acute Phthisis—Chronic Phthisis. . . . .	434
XVI.	Morbid Growths in the Lungs—Cancer or Malignant Disease—Syphilitic Disease—Rare Morbid Formations . . . . .	458
XVII.	Diseases of the Pleura—Pleurisy—Hydrothorax—Hæmothorax—Pneu- mothorax—Hydro- and Pyo-pneumothorax . . . . .	462
XVIII.	General Diagnosis of Affections of the Lungs and Pleuræ . . . . .	474
XIX.	Diseases of the Circulatory Organs—Clinical Phenomena connected with Heart, Arteries, and Veins—Physical Examination of the Circulatory Organs . . . . .	478
XX.	Symptoms and Functional Disorders connected with the Heart—Angina Pectoris—Syncope—Disordered Cardiac action—Palpitation . . . . .	504
XXI.	Diseases of the Pericardium—Acute Pericarditis—Chronic Pericarditis— Adherent Pericardium—Hydro-pericardium—Pericardial Hæmorrhage —Pneumo-pericardium . . . . .	512
XXII.	Acute Diseases of the Heart—Acute Endocarditis and Valvulitis— Acute Myo-carditis—Acute Parenchymatous Degeneration . . . . .	519
XXIII.	Chronic Diseases of the Heart—Affections of the Valves and Orifices— Enlargement of the Heart—Hypertrophy—Dilatation—Atrophy—Fatty Diseases—Peculiar Degenerations and New Formations in the Walls of the Heart—Cardiac Aneurism—Rupture of the Heart . . . . .	523
XXIV.	General Diagnosis, Prognosis, and Treatment of Chronic Cardiac Affections . . . . .	540
XXV.	Malformations of the Heart and Great Vessels—Cyanosis—Blue Disease . . . . .	549
XXVI.	Diseases of the Arteries—Thoracic Aneurism . . . . .	551
XXVII.	Mediastinal Tumours . . . . .	560
XXVIII.	On certain Abnormal Conditions of the Blood—Anæmia—Spanæmia— Chlorosis—Pyæmia—Septicæmia . . . . .	561
XXIX.	Thrombosis and Embolism . . . . .	568
XXX.	Diseases of the Abdomen—Physical Examination of the Abdomen . . . . .	575
XXXI.	Diseases of the Peritoneum—Acute Peritonitis—Chronic Peritonitis— Morbid Growths in the Peritoneum—Abdominal Perforations and Ruptures—Ascites . . . . .	580
XXXII.	Diseases of the Stomach and Intestines—Clinical Characters . . . . .	593
XXXIII.	On certain Gastric Symptoms and Functional Disorders—Gastrodynia or Gastralgia—Spasm or Cramp of the Stomach—Vomiting—Hæmate- mesis—Dyspepsia . . . . .	596
XXXIV.	Acute Gastric Catarrh—Acute Gastritis . . . . .	607
XXXV.	Chronic Diseases of the Stomach—Chronic Gastritis or Gastric Catarrh —Ulceration—Cancer—Pyloric Obstruction—Dilatation of the Stomach—Pyloric Incompetence . . . . .	610
XXXVI.	General Diagnosis, Prognosis, and Treatment of Chronic Gastric Affections . . . . .	618
XXXVII.	On certain Intestinal Symptoms and Functional Disorders—Enteralgia— Intestinal Colic—Constipation—Diarrhœa—Melæna . . . . .	625
XXXVIII.	Inflammation of the Intestines—Intestinal or Enteric Catarrh— Enteritis—Duodenitis—Typhlitis and Perityphlitis . . . . .	633
XXXIX.	Dysentery—Bloody Flux . . . . .	637



CHAP.	PAGE
XL. Intestinal New Formations and Ulcerations—Cancer—Tubercle—Tubercular Ulceration—Albuminoid Disease . . . . .	644
XLI. Intestinal Obstruction . . . . .	649
XLII. Intestinal Worms—Helminthiasis—Trichinosis . . . . .	656
XLIII. Diseases of the Liver and its Appendages—Clinical Characters . . . . .	667
XLIV. On Functional Disorders of the Liver—Hepatalgia—Jaundice . . . . .	669
XLV. Congestion or Hyperæmia of the Liver . . . . .	674
XLVI. Acute Inflammatory Diseases of the Liver—Hepatic Abscess—Perihepatitis—Inflammation of the Bile-ducts . . . . .	676
XLVII. Acute Yellow Atrophy . . . . .	681
XLVIII. Chronic Diseases of the Liver—Hypertrophy and Atrophy—Fatty Liver—Albuminoid Liver—Hydatid Tumour of the Liver—Cancerous and other Growths—Cirrhosis of the Liver—Chronic Atrophy—Syphilitic Disease—Tuberculosis—Rickety Liver . . . . .	682
XLIX. Affections of the Gall-bladder . . . . .	696
L. Gall-stones—Biliary Calculi—Cholelithiasis . . . . .	697
LI. General Diagnosis, Prognosis, and Treatment of Chronic Hepatic Affections . . . . .	700
LII. Diseases of the Spleen—Clinical Characters—Congestion or Hyperæmia—Hæmorrhagic Infarction—Splenitis—Hypertrophy—Leucocythæmia—Other Morbid Conditions—General Diagnosis, Prognosis, and Treatment . . . . .	707
LIII. Diseases of the Pancreas . . . . .	717
LIV. Diseases of the Supra-renal Capsules—Addison's Disease . . . . .	720
LV. Abdominal Aneurism . . . . .	724
LVI. Diseases of the Urinary Organs—Clinical Characters—Examination of Urine—Renal Tumour—Distended Bladder . . . . .	726
LVII. On certain Abnormal Conditions of the Urine—Albuminuria—Pyuria—Chyluria—Filaria Sanguinis Hominis—Hæmaturia—Hæmatinuria . . . . .	746
LVIII. Uræmia—Uræmic Poisoning . . . . .	754
LIX. Renal Congestion—Embolism—Infarction . . . . .	756
LX. Suppurative Inflammation in connection with the Kidneys—Suppurative Nephritis or Renal Abscess—Pyelitis—Pyonephrosis—Perinephritis—General Diagnosis, Prognosis, and Treatment . . . . .	757
LXI. Bright's Disease—Acute Bright's Disease—Chronic Bright's Disease . . . . .	761
LXII. On certain Rare Diseases of the Kidneys—Cancerous and other Growths—Tubercle—Parasitic Growths—Cystic Disease—Hydronephrosis . . . . .	777
LXIII. Urinary Calculus and Gravel . . . . .	781
LXIV. Cystitis—Vesical Catarrh . . . . .	785
LXV. Diseases of the Absorbent System . . . . .	786
LXVI. Diseases of the Thyroid Gland—Bronchocele—Goitre—Exophthalmic Goitre . . . . .	795
LXVII. Myxœdema . . . . .	798
LXVIII. Diseases of the Nervous System—Clinical Characters—Physical Examination . . . . .	800
LXIX. On certain Head-symptoms—Headache or Cephalalgia—Vertigo or Giddiness—Menière's Disease . . . . .	811
LXX. Disorders of Consciousness—Delirium—Insensibility—Stupor—Coma . . . . .	815
LXXI. Disorders affecting Sleep—Somnolence—Insomnia—Somnambulism—Somniloquism . . . . .	818
LXXII. Motor Disorders—Spasms—Convulsions—Eclampsia—Motor-Paralysis or Palsy—Paresis—Superficial and Deep Reflexes . . . . .	821
LXXIII. Disorders of Sensation—Sensory Paralysis—Hypæsthesia—Anæsthesia—Hyperæsthesia—Dysæsthesia—Neuralgia—Tic Douloureux—Intercostal Neuralgia—Sciatica . . . . .	834
LXXIV. Aphasia—Aphemia—Amnesia . . . . .	841

CHAP.		PAGE
LXXV.	On Secondary and Trophic Lesions in Nervous Diseases . . . .	844
LXXVI.	On the Localization of Nervous Diseases . . . .	847
LXXVII.	Hemicrania—Migraine or Megrin—Sick-headache . . . .	855
LXXVIII.	Epilepsy—Falling Sickness . . . .	857
LXXIX.	Hysteria and Allied Affections—Hystero-epilepsy . . . .	864
LXXX.	Hypochondriasis . . . .	872
LXXXI.	Chorea—St. Vitus's Dance . . . .	873
LXXXII.	Tetanus—Lock-jaw . . . .	878
LXXXIII.	Tetany . . . .	879
LXXXIV.	Alcoholism—Delirium Tremens—Chronic Alcoholism . . . .	881
LXXXV.	On certain Forms of Metallic poisoning—Lead-poisoning—Saturnism —Mercurial-poisoning . . . .	886
LXXXVI.	Diphtheritic Paralysis . . . .	889
LXXXVII.	Sun-stroke—Insolation—Coup de Soleil . . . .	892
LXXXVIII.	Acute Cerebral Inflammations—Simple or Primary Meningitis—Tuber- cular Meningitis—Acute Hydrocephalus—Rheumatic Meningitis— Acute Cerebritis—Cerebral Abscess . . . .	893
LXXXIX.	Diseases affecting the Cerebral Circulation—Cerebral Congestion or Hyperæmia—Cerebral Anæmia—Cerebral and Meningeal Hæmorrhage —Sanguineous Apoplexy—Cerebral Embolism and Thrombosis— Cerebral Softening . . . .	902
XC.	Chronic Diseases of the Brain and its Membranes—Chronic Meningitis —Chronic Cerebral Softening—Adventitious Growths—Cerebral Tumour—Chronic Hydrocephalus—Hypertrophy . . . .	918
XCI.	Diseases of the Spinal Cord and its Membranes—Neurasthenia Spinalis —Concussion—Acute Spinal Meningitis—Acute Myelitis—Polio- myelitis Anterior Acuta—Infantile Paralysis—Adult Spinal Paralysis —Sub-acuta—Paralysis Ascendens Acuta—Spinal Congestion and Anæmia—Spinal Hæmorrhage or Apoplexy—Chronic Spinal Menin- gitis—Chronic Myelitis—White Softening—Adventitious Growths in the Cord—General Diagnosis, Prognosis, and Treatment . . . .	926
XCII.	Sclerosis of the Nerve-centres—Diffused Cerebral Sclerosis—Locomotor Ataxy—Tabes Dorsalis—Primary Lateral Sclerosis—Spasmodic Spinal Paralysis—Spastic, Paraplegia—Amyotrophic Lateral Sclerosis— Secondary Lateral Sclerosis—Disseminated Sclerosis—General Diag- nosis, Prognosis, and Treatment . . . .	943
XCIII.	On certain Special Nervous Diseases—Progressive Muscular Atrophy —Glosso-labio-laryngeal Paralysis—Writer's Cramp—Pseudo-hyper- trophic Muscular Paralysis—Paralysis Agitans—Spasmodic Wry- neck . . . .	956
XCIV.	Diseases of the Skin . . . .	965





## LIST OF ILLUSTRATIONS.

FIG.	PAGE
1. Amœboid Leucocytes . . . . .	45
2. Pus-corpuscles . . . . .	48
3. Fatty degeneration of striped muscles . . . . .	65
4. Fatty degeneration of nerve-fibres . . . . .	66
5. Liver-cells, infiltrated with oil . . . . .	68
6. Cells in various stages of pigmentary infiltration . . . . .	72
7. Cells undergoing colloid degeneration . . . . .	73
8. Liver-cells infiltrated with amyloid matter . . . . .	75
9. Spirilla of relapsing fever . . . . .	135
10. Spirilla amongst blood-corpuscles . . . . .	136
11. Spores, rods, and filaments of <i>Bacillus anthracis</i> . . . . .	208
12. Gummy growth of liver . . . . .	282
13. Elements from grey tubercle . . . . .	289
14. Giant-cells . . . . .	290
15. Koch's <i>Bacillus tuberculosis</i> in sputum from a case of phthisis . . . . .	294
16. Cells from a mammary cancer . . . . .	299
17. Primary cancer of rectum . . . . .	300
18. Epithelial Carcinoma . . . . .	301
19. Oidium albicans . . . . .	317
20. Cardiogram . . . . .	485
21. Sphygmographic Tracing . . . . .	500
22. Enlarged Sphygmographic Curve . . . . .	500
23. Sarcinæ . . . . .	599
24. Diagram of Intus-susception . . . . .	651
25. <i>Cysticercus cellulosus</i> . . . . .	658
26. <i>Cysticercus</i> with head extruded . . . . .	658
27. <i>Tænia solium</i> . . . . .	659
28. Head and neck of <i>Tænia solium</i> . . . . .	660
29. Circle of hooks . . . . .	660
30. Head of <i>Tænia medio-canellata</i> . . . . .	660
31. Head of <i>Bothriocephalus latus</i> . . . . .	661
32. <i>Bothriocephalus latus</i> . . . . .	661
33. <i>Ascaris lumbricoides</i> . . . . .	661
34. <i>Oxyuris vermicularis</i> . . . . .	662
35. <i>Tricocephalus dispar</i> . . . . .	662
36. Cyst of <i>Trichina spiralis</i> . . . . .	666
37. <i>Trichina spiralis</i> removed from its cyst . . . . .	666
38. Hydatid . . . . .	685
39. Echinococci . . . . .	686
40. Echinococcus viewed transversely . . . . .	686

FIG.	PAGE
41. Circle of hooklets, and separate hooklets . . . . .	686
42. Uric acid crystals . . . . .	738
43. Urates . . . . .	738
44. Oxalate of lime crystals . . . . .	739
45. Crystals of phosphates . . . . .	739
46. Cystine . . . . .	740
47. Epithelium from vagina, bladder, ureter, and kidney . . . . .	741
48. Pus-cells in urine . . . . .	741
49. Blood-corpuscles in urine . . . . .	741
50. Urinary casts . . . . .	743
51. <i>Filaria Sanguinis Hominis</i> . . . . .	748

# THEORY AND PRACTICE OF MEDICINE.

---

## SECTION I.

---

### CHAPTER I.

#### INTRODUCTION.—OBJECTS AND METHODS OF STUDY.

IN order to acquire a satisfactory knowledge of the Science and Art of Medicine, it is essential that the mental faculties should have been previously cultivated and trained, while at the same time the mind is stored with the requisite information, by an adequate study of the fundamental and more scientific subjects which constitute the earlier portion of the medical curriculum. Above all is it necessary that the student should be thoroughly acquainted with the construction of the human body, and the normal composition, structure, and functions of its various fluids, tissues, and organs in health—in short, with Anatomy and Physiology. Having gained this information, he will be materially aided in his further progress, if, in the first place, he understands definitely what Medicine includes, and what he is required to learn; and, secondly, if he sets about acquiring the requisite knowledge and skill according to a systematic, orderly, and scientific method.

It will be expedient at the outset to indicate the general scope of the study of Medicine, and to explain the meaning of its more comprehensive scientific terms which are in common use.

**DISEASE.**—The study of Medicine may be said to comprehend the study of *disease* in all its relationships. Under this term is included every deviation from the normal condition of the body or any portion of it, either as regards its structure, state of nutrition, or the due performance of its functions. It will be readily understood that there is no absolute line of demarcation between *health* and *disease*, these being merely relative terms. If a disease is associated with any evident structural change, it is said to be *organic*; if no such change can be detected by any method of investigation with which we are at present acquainted, it is called *functional*.

Disease requires to be studied from three main aspects, which may be considered under the heads of:—I. PATHOLOGY. II. TREATMENT OR THERAPEUTICS. III. MORBID OR PATHOLOGICAL ANATOMY AND HISTOLOGY.



I. **PATHOLOGY.** In its correct and comprehensive sense *pathology* signifies that branch of Medicine which deals with and explains the origin, causes, nature, and clinical history of the several morbid conditions to which the human frame is liable. It is divided into:—

1. *General pathology*, which treats of what is common to, or bears upon all or a number of diseases; and which includes certain important subjects, such as congestion, hæmorrhage, inflammation, degenerations. 2. *Special pathology*, which is concerned with the various special and individual diseases. The word *pathology* has come to be frequently employed in a more restricted sense than that just indicated, namely, as signifying the explanation of the essential nature of morbid processes, of the characteristic changes associated with the several diseases, and of the pathological causes of symptoms. According to its wider and more correct definition, it will be seen that pathology includes the following subdivisions:—

A. **Ætiology or Causation of Disease**, which treats of the causes of the various maladies, or the influences and agencies by which they are originated.

B. **Symptomatology or Semeiology.** These terms are applied to the science which is concerned with all that pertains to morbid conditions, whether general or local, in their *clinical* relations, that is, as they are represented in the living subject. It has to do therefore with:—1. Their **CLINICAL HISTORY**, including—(a) *mode of onset or invasion*; (b) *symptoms and physical signs or clinical phenomena*; (c) *course, duration, and modes of termination*; (d) *varieties and types*; (e) *complications and sequelæ*. 2. Their **DIAGNOSIS**, which signifies the recognition of the seat and nature of particular diseases, and their discrimination from other morbid states. 3. Their **PROGNOSIS**, or the judgment which is formed as to their future course and ultimate issue.

II. **TREATMENT OR THERAPEUTICS.**—This important branch of Medicine deals with the principles upon which the management of disease is conducted; and the measures and agents which are employed with the view of preventing or curing the different maladies, of modifying their progress, or of relieving their attendant symptoms.

III. **MORBID OR PATHOLOGICAL ANATOMY AND HISTOLOGY.**—The structural and other recognizable changes which are associated with organic diseases come under this head, as revealed by *post-mortem* examination.

An useful and adequate knowledge of Medicine can only be built up by studying the subject in all the particulars just indicated. Before proceeding to study individual diseases, it is highly desirable to have a comprehensive acquaintance with the main facts pertaining to general morbid processes, such as are grouped under *general pathology*. Further, it is essential that all classes of diseases should receive due attention; and that the pathological and clinical relations of different systems and organs to each other should be known. It is a great mistake for the student to confine his observation to any one or more “specialties,” even though he may intend ultimately to practise such; at the same time there are some of the more special departments which at the present day deserve particular attention.

I proceed now to point out the plan according to which Medicine can be learnt most easily and satisfactorily. First, it should be studied as a *Science*, a knowledge of its *theory* being acquired, as taught in books, lectures, etc., in which such information and explanations are given with reference to principles and facts as can be conveyed by these methods of instruction. Secondly, it must be learnt as an *Art*, the *practice* of medicine being studied:—(i.) By personal observation and examination of actual cases of the several diseases, as exemplified in individual patients. (ii.) By proper attention to the different modes of *clinical instruction*, in connection with which allusion may be made to the great advantages to be derived from a thorough training in “case-taking,” under competent supervision. (iii.) By the education of those external senses which are of such constant service in the investigation of diseased conditions, especially touch, sight, and hearing; and by repeated practice in the employment of the special instruments which are available for a similar purpose. (iv.) By the observation and study of morbid changes produced in the organs and tissues, as disclosed on *post-mortem* examination, the more minute alterations being ascertained by chemical and microscopical investigations.

All the methods of study just mentioned are important, though, of course, those of a practical kind are by far the more so, for any one who relies on a mere theoretical knowledge of medicine is absolutely incompetent to enter upon its practice. Still this theoretical knowledge is not to be despised, seeing that it helps materially to clear the way for practical study, and saves a great waste of time and labour, for the student is enabled to comprehend far better what he observes, if he has mastered at least the elements of the subject, and the more information he possesses before beginning his practical work, the more likely is he to derive full and permanent benefit therefrom.

The usual fields for the study of illustrative cases of the several diseases are the wards of a hospital, and the different out-patient departments: each has its advantages and should be duly attended, the former affording illustrations of the acute or more serious chronic affections, the latter of the more common ailments met with in ordinary practice, and of the minor or more localized disorders not usually admitted into hospitals. If practicable, however, it is very desirable that patients should also be visited at their own homes, so that they may be seen surrounded by those conditions which form part of their experience in every-day life. There are certain diseases, moreover, a practical acquaintance with which is of the greatest consequence, which generally can only be studied in this way, namely, the majority of the *acute specific fevers*, which, with a few exceptions, are not admitted into general hospitals, on account of their infectious nature, and the opportunity is afforded only to a small number of observing them in the special hospitals set apart for these complaints. Though it is requisite, if possible, that examples of *all* forms of disease should be seen, however rarely they may be met with, yet those which are the more common, and which are likely to come frequently under notice in the course of practice, should receive the chief attention, and of these the student cannot observe too many cases, in order to become familiar with all the important facts pertaining to them.

## CHAPTER II.

## ÆTIOLOGY OR CAUSATION OF DISEASE.

THIS is a subject of the utmost importance, and demands the most attentive consideration, for an adequate acquaintance with Ætiology is of great value in several respects. It often gives material aid in diagnosis; it not uncommonly throws a light upon prognosis, as the same morbid condition may differ much in its gravity according to its cause; in the majority of individual cases it affords most useful indications as regards treatment; and, above all, it enables the practitioner to adopt preventive measures against the development or extension of many diseases, and thus to preserve the health of the general community.

A knowledge of Ætiology implies not only a general knowledge of the causes which are capable of producing disease; but also a more particular acquaintance with the relation between special causes and special diseases, and, so far as this can be obtained, an intelligent comprehension of the manner in which the various influences produce their injurious effects.

It will be readily understood that the same cause may produce many and very different diseases, and *vice versa*; but with regard to the various classes of disorders, it will be found that each is brought about most commonly by a certain limited number of causes, which act more or less directly on the organ or part affected. This remark may be illustrated by the relation which exists between "taking cold" and lung-affections; between improper food and drink and derangements of the alimentary canal; or between excessive mental labour and brain-diseases. Further, the particular causes which give rise to special affections of each organ are still more limited in their range. Some diseases can only be originated by one, definite, specific cause, and to such the term *specific* is applied.

Numerous terms have been employed to classify causes, often with much ambiguity of meaning. Only those which are practically useful will be alluded to here. The primary division generally made is into *proximate* and *remote*. The *proximate* cause is really the actual morbid condition of any organ or structure upon which the symptoms present depend, and it is synonymous with *pathological* cause. *Remote* causes are further divided into *predisposing* and *exciting*, some of the latter being named *determining*.

*Predisposing* causes include those influences which bring about a condition of the system generally, or of some organ or part, rendering it more prone to become the seat of disease. *Exciting* causes comprehend the immediate and direct agencies by which the different morbid changes are produced. The term *predisposition* is used to express the state favourable to the action of an exciting cause, and the individual in whom it exists is said to be *predisposed*. It is not to be supposed, however, that there is a distinct and definite limit between the two classes of causes just mentioned; what may only predispose at one time may excite at another, and especially is this true when several



deleterious influences act together and for a long period. Further, a so-called predisposing cause may only render one organ more liable to be attacked with a certain disease than another; for example, age exercises a marked influence as regards the seat of tubercle or cancer.

For the purpose of pointing out the more common causes of disease somewhat in detail, it will be convenient to classify them into:—1. INTRINSIC, or those depending upon the individual, in whom they are either *inherent* or *acquired*. 2. EXTRINSIC, or those due to accidental, and chiefly external influences. So far as this is practicable, an endeavour will be made to indicate which may be ranked as predisposing, and which as exciting causes.

1. INTRINSIC. *a. Age*.—A number of diseases are more prone to occur at certain periods of life than at others; while some morbid conditions tend to affect different organs at different ages, or even to be limited to special tissues in the same organ. This may often be explained by the nutritive and functional activity of the system generally, or of certain organs, being much greater at one time of life than at another, and therefore the liability either to general or local disease is more marked. Changes of structure also, in the direction of decay, not unfrequently account for the predisposition due to age, as, for example, degeneration of the vessels in advanced life rendering them brittle, and thus leading to cerebral hæmorrhage. Young children and persons advanced in years are very subject to ailments of various kinds.

*b. Sex*.—Females are more prone to certain affections than males, and *vice versâ*; while some diseases are necessarily limited to one or other sex. This depends on the difference in the conditions of certain organs in the two classes of persons, and in the length of outlets (*e.g.*, the urethra); on the functions peculiar to each sex; on the dissimilarity in habits, occupation, etc.; on the difference in constitutional strength and vigour: or on certain peculiarities in the nervous system, women being much more sensitive and excitable, and therefore more liable to various nervous disorders. The proportion of deaths is greater among males than females.

*c. General or Constitutional Condition. State of Health, etc.* A state of general debility, whether congenital or acquired, predisposes to many diseases. Possibly the opposite condition of robustness and vigour may increase the liability to other affections. The condition of the blood has also much influence, plethoric or anæmic individuals being predisposed to many complaints. Previous diseases, especially those of an acute nature, frequently predispose to or excite others, *e.g.*, the various fevers, hooping-cough, lung-affections, rheumatism, and syphilis. Certain symptoms, such as cough, particularly if neglected, may be productive of serious mischief. Habitual neglect in attending to the natural functions, especially those connected with the alimentary canal, very commonly leads to injurious results. The existence of morbid changes in certain organs or tissues may readily induce or predispose to further lesions in them, or may give rise to diseased conditions in other parts. Thus a fatty or calcified state of the arteries renders them liable to be easily ruptured; cardiac diseases often excite lung-affections, and *vice versâ*: or one disease of the lung or heart may originate another. Other causes of disease which may be alluded to under this head are direct loss of blood; excessive or long-continued



discharges; and the sudden suppression of an habitual discharge, of a chronic skin-disease, or of some local development of a constitutional disorder, such as gout.

*d. Temperament.*—Four principal temperaments are described, namely, the *sanguineous*, *lymphatic*, *bilious*, and *nervous*, and each is supposed to indicate a susceptibility to certain particular diseases, though the statements made on this matter are very contradictory, and are often without any foundation in fact. Speaking generally, the *sanguineous* temperament is believed to predispose to fevers of a sthenic type, active congestion or hæmorrhage, and acute inflammations; the *lymphatic* to passive congestion, dropsy, low inflammations, and certain constitutional affections; the *nervous* to various disorders of the nervous system; and the *bilious* to digestive and hepatic derangements.

*e. Idiosyncrasy.*—Some individuals are affected injuriously by certain agencies, which do not at all influence others in the same way. This is the case with articles of diet, as fish or mushrooms; or medicines, such as iodide of potassium or quinine. To this individual peculiarity the term “idiosyncrasy” is applied, and it probably has an influence in predisposing to some diseases.

*f. Hereditary predisposition.*—Several diseases are supposed to be capable of transmission from parent to offspring, and with regard to some of them this belief is unquestionably true, but the evidence is by no means so clear or reliable with respect to others. It must be borne in mind that members of different generations may be exposed to the same extrinsic causes of disease, and this may account for the fact that certain affections seem to run through families. Those maladies or general morbid conditions usually considered to be hereditary include:—

(i.) Certain constitutional or blood-diseases, namely, gout, rheumatism, scrofula, tuberculosis, cancer, syphilis, and the hæmorrhagic diathesis.

(ii.) Some affections of the nervous system, namely, epilepsy, chorea, insanity, hypochondriasis, neuralgia, apoplexy, paralysis.

(iii.) Physical deformities, as well as deficiencies in connection with the special senses, such as blindness or deafness.

(iv.) Early degenerations, either local or general, which are evidenced by degeneration of the vessels, fatty changes in organs, loss of the elasticity of the skin, premature greyness or baldness, loss of teeth and other signs of decay.

(v.) Some skin-diseases, especially psoriasis and lepra.

(vi.) Emphysema and asthma (?).

(vii.) Gravel and urinary calculus.

(viii.) Diabetes.

(ix.) Hæmorrhoids (?).

The morbid conditions observed in different generations need not be identical, but may be merely allied in their nature. This particularly applies to the nervous diseases above mentioned, and to the degenerations. For instance, there may be epilepsy in one generation and insanity in the next. Again, some vicious habit in the parent may lead to disease in the offspring; thus, intemperance may undoubtedly originate certain nervous disorders. In some cases a constitutional disease in the parent, such as syphilis, may only cause the offspring to be weakly and delicate.

The transmitted disease may be actually developed in the foetus in

*utero*, being then termed *congenital*; it may appear spontaneously at some period or other after birth; or it may lie dormant until brought out by an exciting cause. In some cases it is supposed to pass over one generation and appear in the next, this being called "Atavism."

Hereditary tendency to disease may unquestionably be intensified by intermarriage of those suffering from the same affection, for example, phthisis; and also by the marriage of those who are closely related, very young, or of very unequal ages.

The hereditary character of diseases is sometimes revealed by their development at an earlier age than is usual, of which gout is an instance. Families occasionally seem remarkably prone to be attacked by certain affections, and to have them with great severity, of which the infectious fevers afford illustrations.

*g. Race.*—There can be no doubt respecting the influence of race in increasing the liability to certain diseases, and *vice versâ*. This is exemplified in the greater proneness of white than black people to suffer from malarial fevers. The prevalence of some diseases among particular races may, however, be explained by their habits, mode of living, and place of abode.

## 2. EXTRINSIC. *a. Causes depending on surrounding conditions.*

(i.) *Atmosphere.*—The air breathed must necessarily influence greatly the state of health, and it does so in the following ways. It may be impure, because it is not sufficiently changed by proper ventilation, and therefore contains an undue amount of the products of respiration and combustion. Or it may be mixed with gases not usually present, such as those which emanate from sewers or decomposing animal or vegetable matters, or those which are given off in connection with many manufactories. Suspended impurities are also often present, for example, dust, cotton, hair, wool, unconsumed carbon, fragments of metals, arsenic, and, probably, living organisms. The atmosphere is frequently the vehicle for the transmission of specific poisons. The degree of moisture of the air is highly important, excess or deficiency in this respect often producing very injurious consequences. Possibly its electrical condition, or the amount of ozone in it, may have some influence. And, lastly, the degree of pressure of the atmosphere certainly affects the health, of which we have a familiar illustration in the consequences which sometimes follow the ascent of a high mountain. Atmospheric influences may act either as predisposing or exciting causes of disease.

(ii.) *Temperature.*—Excessive or long-continued heat or cold, whether acting upon the general system or applied locally, is most injurious. A sudden change from one extreme to the other often causes serious mischief, and so does exposure to cold winds. Even in warm weather a "chill" may give rise to disease, and this is frequently due to the patient's own carelessness, for instance, neglecting to change wet clothes, or remaining exposed to a draught when heated and perspiring.

(iii.) *Amount of light and insolation.*—Those who reside or spend a considerable part of their time where little or no sun-light enters, are unquestionably below par as regards health. The amount and kind of artificial light employed has also some influence in the causation of disease.

(iv.) *Soil.*—The chief modes in which the soil exercises its influence upon the body are by the amount of vegetable matter which it contains

susceptible of decomposition; by its degree of, and permeability to moisture; by its effect on the heat and light of the sun, whether absorptive or reflective; and by its chemical composition, which affects that of the water and air in the neighbourhood. Wherever there is an accumulation of vegetable substances, with sufficient moisture and a certain temperature, as is the case in marshy districts, malarial affections are almost always prevalent. Clayey soils are very moist and cold. Most sandy and gravelly soils are healthy, unless they contain vegetable matter. Those in which there is a large proportion of lime and magnesia are believed to originate goitre and renal calculus. The breaking up of soil is often attended with evil consequences.

(v.) *Sewage*.—This is a very common source of disease, both on account of the deleterious gases given off from it, and the decomposing organic matter of which it consists. In certain cases it contains specific agents in the causation of disease, and promotes their development, or renders them more virulent. Sewage materials or the gases which emanate from them are particularly hurtful when mixed with water which is used for drinking purposes.

**b. Causes due to the social condition and habits of the individual, and to certain other accidental influences.**

(i.) *Food*.—This may be deficient in quantity or of improper quality, either habitually or only temporarily, and may thus promote or induce disease, especially in children. On the other hand the diet may be excessive, or too rich in quality. Irregularity as regards meals, the habit of bolting food, or insufficient mastication from any cause are often very injurious.

(ii.) *Drink*.—Intemperance in the use of *alcoholic stimulants* is a fertile source of disease, and it is always well to bear this in mind when investigating any doubtful case. Spirits do most harm, especially if taken at frequent intervals, strong or only slightly diluted, and on an empty stomach. It must be borne in mind also that many of the compounds sold as beer, wines, and spirits, contain highly noxious adulterations. Water, or the want of it, is a very prolific cause of disease. When this element is insufficiently supplied for cleanliness and other purposes, serious results often ensue. The habit of drinking large quantities of water, especially during meals, frequently does harm. Again, water may be the direct means of conveying various morbid agents into the system, such as noxious gases, certain salts, poisonous metals, the ova of worms, animal organic matters, particularly those contained in the excrements, vegetable matters in a state of decomposition, and specific poisons. Tea-drinking in excess is a common cause of troublesome symptoms, and its injurious effects are familiar in all classes of society. Milk may do harm if decomposed or adulterated, and it has been definitely proved to be not unfrequently the medium by which certain specific poisons enter the system.

(iii.) Certain *habits*, such as smoking or snuff-taking in excess; the use of narcotics, especially opium, morphia, or chloral hydrate; and excessive indulgence in hot condiments, not uncommonly injure the health seriously.

(iv.) *Clothing*.—This may be insufficient, either habitually or only from time to time; or certain regions may be inadequately protected. Thus infants and young children are frequently completely exposed about the lower part of the body, and no doubt "take cold" as a consequence. The chest is also in many persons insufficiently



covered. On the other hand individuals are not uncommonly over-clad, especially children. Clothing may also do harm by being too tight and exerting pressure, as in the case of those who wear tight stays or belts. The habit of neglecting to change wet clothes is very dangerous.

(v.) *Want of cleanliness*, domestic or personal, often induces disease. Cutaneous affections may arise from the repeated contact with the skin of various substances of an irritating character.

(vi.) *Amount of labour and exercise*.—Many persons suffer as the result of excessive and prolonged labour or exertion, whether carried on habitually or only at intervals. On the contrary a much larger number are injured by leading a sedentary life, and taking no exercise. Various occupations furnish instances of both these hygienic errors, though they are often voluntarily indulged in apart from occupation, especially the leading of a sedentary existence.

(vii.) *Mental causes*.—Among these may be specially mentioned excessive intellectual effort or study, particularly if combined with deficient sleep or mental anxiety; and all violent or depressing emotions, such as grief, sudden joy, deep anxiety of mind, or severe and sudden fright. They may either predispose to or excite diseases, especially those connected with the nervous system.

(viii.) *Mechanical causes*.—These constitute a very important class, especially in exciting or determining some morbid condition, owing to the direct injury or irritation which they originate. Mechanical causes chiefly include external violence, long-continued pressure, excessive use of a part, over-exertion and straining, prolonged maintenance of a fixed position, and the irritation of foreign bodies. Among the last may be particularly mentioned calculi, accumulations of fæces, parasitic animals and plants, and particles inhaled into the respiratory organs. Occupations often act injuriously in one or more of the ways above indicated. A mechanical cause sometimes leads to the local development of a constitutional disease; thus pressure or injury may determine the formation of cancer in a particular organ.

(ix.) *Causes connected with the sexual functions*.—Venereal excesses, masturbation, and too early or frequent sexual excitement, unquestionably often give rise to serious mischief.

The elementary causes which have thus far been considered are usually more or less combined in any individual case. On an extensive scale their influence is evident in the diversities which are observed as to the general state of health of communities, and as to the particular diseases which prevail in civilized and uncivilized countries; in different nations, and in the same country under varying modifications of government, civilization, religion, etc.; in large towns and country places, as well as in different towns or districts and in different parts of these; and in mountainous regions and low confined valleys. Some of them also explain the influence which *climate, season, occupation*, and other so-called causes of disease exercise upon the general health, and upon the development of many special complaints.

**SPECIAL CAUSES OF DISEASE.**—There are certain agents in the causation of disease which have not yet been considered, and which require separate notice. They are chiefly of the nature of poisons of various kinds, and produce effects which are more or less definite and constant.

1. **Chemical poisonous substances, chiefly inorganic.**—The effects of various chemical poisons upon the system are sufficiently



obvious, and require no comment. It is necessary, however, to call special attention to the fact that some of these may gain an entrance into the body in connection with the occupation of an individual or in some other way, without being directly administered as poisons or medicines. This is seen in the deleterious effects, local or general, which are produced by lead, mercury, phosphorus, arsenic, copper, gold, and other substances. With respect to arsenic, it is important to observe that one of its compounds may be given off as a fine powder from certain papers used for papering rooms, being afterwards inhaled along with the atmosphere in which it floats, thus giving rise to symptoms of poisoning.

2. **Causes originating in the vegetable kingdom.**—(i.) Many of the ordinary poisons are derived from this source, such as opium. (ii.) *Parasitic plants* growing in various structures of the body are frequent causes of disease, especially of skin-affections. The presence of certain minute fungi (*sarcinæ*) in the stomach is supposed occasionally to excite vomiting. (iii.) *Decomposing vegetable matter* often does much harm. It is particularly injurious by producing *malarial* or *miasmatic poisons*, so prevalent in marshy districts. These give rise mainly to ague and remittent fevers, but also to certain nervous disorders and other complaints. This subject will be discussed in detail in a later portion of this work. (iv.) It is believed by many that *contagion* is due to low vegetable organisms.

3. **Causes originating in the animal kingdom.**—(i.) Certain animals are venomous, and are capable of inflicting poisoned wounds, for example, serpents. (ii.) Some animals are poisonous if taken internally, such as cantharides. (iii.) *Parasites* derived from the animal kingdom very commonly set up morbid conditions. The various intestinal worms, and the external parasites which infest the skin afford illustrations. (iv.) *Specific contagious poisons.*—A number of very important diseases originate from the entrance into the system of certain specific poisons, which are transmitted from some other animal to man, or from one human being to another, for instance, hydrophobia, small-pox, scarlatina, syphilis. These will call for special and detailed consideration hereafter.

4. **Causes originating within the system.**—Some morbid conditions are due to the presence of a poison in the blood, which has been generated within the body, as the result of perversion of the functions of digestion, assimilation, and nutrition. Gout furnishes an illustration, and once developed the malady may be hereditarily transmitted.

---

## CHAPTER III.

### SYMPTOMATOLOGY OR SEMEIOLOGY.

A SYMPTOM may be defined as any phenomenon which in the living subject gives evidence of the existence of a diseased condition. It is necessary to explain certain terms which are usually employed to indicate the nature of symptoms. 1. *General or Constitutional* and *Local*.—These imply respectively that the symptoms are referred to the

entire system; or only to some particular part. 2. *Objective and Subjective*.—The former include all phenomena which are evident to the senses of the observer, *e.g.*, redness or swelling; the latter those which are only subjectively felt by the patient, *e.g.*, pain or numbness. 3. *Direct or idiopathic and Indirect*.—These terms signify respectively, symptoms which are immediately associated with the diseased part; and those connected with some part remote from the seat of mischief, the latter in some cases being called *sympathetic*. Thus vomiting is often present as a sympathetic symptom during the passage of a renal calculus. 4. *Premonitory or Precursory*.—Before the actual development of a disease, symptoms may be present indicating more or less clearly what is about to happen, and to such the above terms are applied. 5. *Diagnostic, Prognostic, and Therapeutic*.—These words sufficiently explain their several meanings, namely, as representing those symptoms which indicate the nature of a disease, its prognosis, or its treatment. *Pathognomonic* is the term applied to such symptoms as belong to one particular disease and no other, which are therefore absolutely characteristic of this affection. Much confusion has arisen in the use of the word *sign*. Frequently it is employed as synonymous with *symptom*, but this is incorrect, for a *sign* really means a symptom which points to the nature of a disease; in short, it is a *diagnostic* or *pathognomonic* symptom. *Physical signs* strictly speaking include all *objective symptoms*, but by some writers only such of these as are elicited by certain special methods of *physical examination* are recognized as *physical signs*.

In the majority of cases a number of symptoms are grouped together, general and local, subjective and objective, but sometimes one becomes so prominent as to be popularly looked upon as the disease itself, while it gives the main indication for treatment, *e.g.*, dropsy, jaundice, or hæmorrhage. Under such circumstances, however, it is imperative that the actual pathological cause of the phenomenon should be carefully sought for, and the nature of the prominent symptom often affords a useful hint as to the direction in which this inquiry should be conducted.

It is very desirable that a knowledge should be acquired of the *clinical phenomena* which pertain to each organ or system of the body, before the study of its individual diseases is entered upon. In this work, therefore, the description of the diseases of the several organs will be preceded by an outline of the prominent symptoms and signs which have to be looked for as indications that they are in a morbid condition.

Having offered these general observations, it will now be expedient to consider briefly each of the more important particulars relating to the symptomatology of disease.

## I. MODE OF INVASION OR ONSET, COURSE, AND DURATION.

The varieties which may be met with in these particulars are as follows:—1. The invasion of an illness may be quite *sudden*, as often happens in the case of apoplexy, syncope, and many forms of hæmorrhage, the subsequent course necessarily differing in different instances, a rapidly fatal termination being not uncommon. 2. Frequently a disease is *acute*, coming on rapidly, though often preceded by premonitory symptoms; being severe in its character; and of brief, or at any rate, limited duration. Many acute affections run a tolerably definite

course ordinarily—for instance, the eruptive fevers and pneumonia; but irregularities are frequently observed, owing to disturbing influences, and several diseases of this class exhibit distinct *varieties* in their progress. When the onset is less rapid, and the symptoms are less intense, the illness is said to be *sub-acute*. 3. The great majority of complaints are *chronic*, the symptoms setting in gradually and not being severe, while the progress is slow and protracted. A chronic disease may, however, be the sequel of an acute attack, or such an attack is often the cause of a fatal termination in chronic cases. 4. Some diseases are characterized by *periodical* exacerbations, which come on at regular or irregular intervals, the patient being comparatively or even quite well in the meantime. Such complaints are chronic in their progress, but acute or sudden as regards the onset and intensity of the attacks, having a remittent or an intermittent course. Epilepsy, ague, and asthma will serve as illustrations.

## II. VARIETIES AND TYPES.

Many diseases are liable to present more or less evident deviations from their ordinary clinical course, and these in some instances are so distinct and characteristic as to be termed *varieties*. A few diseases exhibit well-marked *types*.

## III. COMPLICATIONS AND SEQUELÆ.

*Complications* include morbid conditions which arise during the course of a disease, but which do not usually form part of its clinical history. They may be due to the same cause; or be the direct result of the primary affection; or supervene as accidental and independent events. *Sequelæ* are those morbid states which remain after, or are developed subsequently to the apparent cure of various affections. Complications and sequelæ are particularly observed in connection with acute diseases, such as fevers. It is very necessary to be familiar with those which are liable to arise in the several disorders, in order to be prepared for them, and to take measures with the view of preventing their occurrence.

## IV. TERMINATIONS.

From a clinical point of view a case may terminate in:—1. *Complete recovery*, which is usually gradually established, the patient passing through a period of convalescence of longer or shorter duration, but may be suddenly or very rapidly brought about. 2. *Incomplete recovery*, either a condition of impaired general health remaining, or some organ or part being permanently altered in its structure or functions: in fact, a *chronic* state of disease remaining behind. 3. *Death*, which event may take place suddenly, rapidly, or slowly. As a rule it is a complex process, the functions of all the chief vital organs being more or less involved; but often the signs of approaching dissolution are associated more especially either with the heart, respiratory organs, or brain. Death beginning at the heart is said to be by *syncope*, and it may be due either to a want of a proper supply of blood to the heart—*anaemia*; or to a loss of contractile power in this organ, from mechanical interference with its action, structural changes in its walls,



or nervous disturbance—*asthenia*. In some instances, such as when death results from starvation, these two modes are combined. Death commencing in the respiratory organs is said to be by *suffocation* or *asphyxia*. This may depend upon the air inspired being unfit to aërate the blood, or, for various reasons, not entering the lungs in sufficient quantity—*apnoea*; or upon a stoppage of the flow of blood through these organs, as happens when a clot suddenly obstructs the pulmonary artery. Death beginning at the brain is said to be by *coma*, being characterized by a primary state of stupor or insensibility, which, however, is soon followed by interference with the respiratory and circulatory functions. These modes of death are merely mentioned at present, as their characteristic phenomena will demand a full description when treating of the diseases of the several organs.

#### V. METHOD AND OBJECTS OF CLINICAL EXAMINATION.

It is extremely important that the practitioner of medicine should be thoroughly conversant with the mode of conducting the clinical examination of patients, and that he should be able to carry this out in an intelligent and systematic manner. In order to acquire this ability, the student should realize that he needs considerable training and practice in “case-taking,” a mode of clinical instruction to which he ought to devote earnest and diligent attention, taking notes of a variety of cases, and writing commentaries upon them. It is very desirable that some definite plan of procedure in this investigation should be adopted and impressed firmly upon the memory, so that it may be followed without effort on the part of the observer. The plans recommended by various writers differ only in minor details, and that of which I now propose to give an outline will answer every necessary purpose. I will endeavour to point out at the same time the object and meaning of the different enquiries which are made, as it is very important to have clear views on this matter.

1. Having noted down the patient's name, age, sex, race (if peculiar), whether married or single, and the date of admission into hospital or when first seen, enquiry should be made with regard to *residence, social position, occupation, habits, and mode of living*, with special reference to *food and drink, clothing, and cleanliness*. Thus information is obtained as to the **general history**.

2. The **family history** should then be taken, in order to ascertain if any hereditary tendency to disease exists. It may be necessary not only to enquire about particular diseases, but also with reference to the habitual state of health, the ages at which the death of members of the family has occurred, and other matters which individual cases might suggest. Of course, it is of primary consequence to investigate with regard to parents, brothers and sisters, and children; but grandparents, and collateral relatives, such as uncles, aunts, and cousins, must not be neglected, should there be any reason to believe that some hereditary taint exists.

3. The **previous state of health** of the patient is next to be investigated, and the occurrence of particular diseases noted. Among those maladies which it is particularly necessary to keep in mind are the acute specific fevers, rheumatic fever, pulmonary affections, and syphilis in certain cases. It is always well to ascertain how the chief functions



are habitually performed, especially those connected with the digestive organs, and, in females, the menstrual functions.

4. Coming then to the **present illness**, enquiry must be made as to whether it can be traced to any definite cause: and as to its *clinical history*, including its duration, so as to determine whether it is acute or chronic, in the former case it being requisite to ascertain as nearly as possible the exact date of its commencement; its mode of invasion; the chief symptoms complained of; and their progress up to the time at which the patient comes under observation.

It will be evident that the chief object of the investigation thus far is to find out what causes have been influential in bringing about the morbid condition present, whether revealed in the general history, family history, previous health, or in the history of the origin of the present illness. No case ought to be looked upon as properly investigated, until every effort has been made to trace the mischief to its source, and, in order to arrive at a satisfactory conclusion on this matter, it will be obvious that it is essential to have as correct and concise a knowledge as possible of the ordinary causes which are likely to give rise to disease of each organ or system, as well as of the relation between particular causes and particular diseases. The degree of difficulty in making out the ætiology of a case varies very considerably; in some instances it is evident at once, and one or more causes may be definitely and positively fixed upon; in others very careful and prolonged enquiry has to be made, and then, perhaps, without any substantial result. And here it must be remarked that much caution is often needed in accepting the statements of patients, and this applies especially to the account they give of their habits, family history, and previous illnesses. Many mislead from ignorance, but it must also not be forgotten that patients who indulge in vicious habits, such as abuse of alcoholic stimulants, or who have suffered from venereal diseases, not unfrequently try to conceal these facts.

Not only is the enquiry up to this point useful in determining the ætiology of a case, but, further, the information obtained is often of material aid in diagnosis.

5. The next and most important step is to take the **present state**, that is, to submit the patient to a personal examination, and note the existing clinical phenomena. Here again it is necessary to impress upon students the extreme desirability of adopting a systematic course in conducting this examination. This not only materially facilitates the process of arriving at a diagnosis, but, not uncommonly, it is absolutely essential in order to come to a correct conclusion as to the seat and nature of a disease. When learning how to "take cases," it is desirable to go through a tolerably complete investigation of every organ and system of importance in the body, first noting down any general symptoms which may be complained of or observed.

The methods by which the required information is obtained are by *intelligent and orderly questioning* of the patient, or of friends or others who are in a position to render such information, should the patient be very young, or mentally or physically unfit to be interrogated; and by *objective or physical examination*. The former method reveals subjective, the latter objective symptoms and *physical signs*. The term *physical examination* is somewhat ambiguously employed; properly it is synonymous with *objective examination*, and should include all modes of investigation in which the external senses of the observer are brought into use; by

some, however, it is confined to certain special methods, such as those adopted in the exploration of the chest or abdomen. In conducting these methods most important aid is derived from the employment of various instruments, such as the stethoscope, laryngoscope, ophthalmoscope, thermometer, and microscope; and from the use of chemical tests.

It will be at once evident that what is ascertained by objective examination is generally far more helpful to the practitioner, and can be much more implicitly relied upon, than what is gathered from the statements of patients or others, and therefore this method ought never to be neglected, if it can be brought to bear in any way upon the investigation of a case. With regard to the subjective sensations of patients, although, of course, they should always be taken into account and duly weighed, both for diagnosis and treatment, yet the description of them must be received with a certain degree of caution, otherwise very serious mistakes may be made. They are liable to be exaggerated or misrepresented, and it must not be forgotten also that symptoms may point to one part when some other is actually the seat of mischief. There is one rule which should be invariably followed by the practitioner, namely:—to test the statements of patients, whenever this is practicable, by *personal* observation, so as to verify them or otherwise, and to find out as much as possible for himself. This remark may be illustrated by such symptoms as spitting of blood, vomiting, local redness or swelling, about which patients frequently give very unreliable accounts. The special modes of *physical examination* demand most careful study, as in a considerable proportion of cases one or other of them constitutes the only means by which a correct diagnosis can possibly be attained. In order to carry them out satisfactorily, much *personal* practice is needed, during which the several external senses are trained and educated, and the use of the various instruments is learnt, while at the same time a knowledge is acquired of the nature and meaning of the *physical signs* which are observed or elicited. It must be remarked that in subjecting patients to physical examination, regard must always be paid to their condition, for they may be too exhausted or otherwise incapacitated for undergoing the process, and might therefore be seriously injured if proper care were not exercised.

6. Having thus concluded the clinical examination of the patient, and methodically recorded the facts elicited, the student should then **write a commentary** upon the case, summing up its leading ætiological and clinical features, and giving his views as to its diagnosis, pathology, prognosis, and appropriate treatment. In this way the mind is trained for going through that process of reasoning which is always supposed to be performed when an opinion is being formed on these matters, but which too often receives very inadequate attention, or is even ignored altogether.

7. If possible, it is very useful to watch the case in its further **course** up to its **termination**, noting the progress of the symptoms, as well as any new phenomena which may arise, including **complications** and **sequelæ**. Should the termination be fatal, the **post-mortem examination** ought to be conducted in the same systematic manner as the clinical investigation, a record being kept of the condition of the various organs and structures.

Of course, it is not to be expected that those engaged in ordinary practice can submit every patient to the complete routine examination



just described, nor, indeed, is this required in the majority of cases; much of the general and family history will be known in many instances, and an experienced observer can usually obtain a speedy clue as to the probable seat of disease, and can frame and direct his enquiries accordingly. At the same time every practitioner ought to be thoroughly competent to carry out a full and searching investigation, should this be needed; while it is most important to avoid falling into the habit of conducting the enquiry in a careless and superficial manner, and every case should receive as attentive a consideration as its nature seems to demand.

A few concluding hints as to the mode of examining patients when rapidity of diagnosis is desirable, may be of assistance to young practitioners, as well as to advanced students, especially those who may have to undergo an examination in clinical medicine.

1. Whenever a patient is first seen, the observer should always be looking out for any sign of disease which may be evident on a superficial and cursory examination. Much information is often thus gained, and important clinical phenomena may be at once noticed, which give most useful indications as to the seat or nature of the existing malady. The chief points to which attention is to be directed are as follows:—  
*a.* The **general aspect and condition**. This may reveal, for example, the “typhoid state”; corpulence or emaciation; malnutrition of the muscles; a peculiar temperament; agedness; or striking evidences of some constitutional disease, such as cancer or scrofula. *b.* The **posture or gait**, which may indicate great debility or helplessness; prostration; difficulty in breathing; restlessness; or immobility on account of pain, paralysis, &c. *c.* The **countenance**. This deserves careful study, for it often affords most instructive information. Thus we may observe an *abnormal colour*, such as the pallor of anæmia, or of syncope or shock; the peculiar tint of chlorosis; the malar flush of “hectic fever”; the redness and turgidity of plethora; the dusky or livid hue of some cardiac and pulmonary diseases; the white and pasty appearance characteristic of certain forms of kidney disease; or the yellow colour of jaundice. There may also be noted puffiness, especially about the eyelids; a bloated condition of the face; or enlargement of the small blood-vessels. The *expression* is also frequently very characteristic, indicating, for instance, serious illness, pain, anxiety, or morbid indifference, but especially as pointing to certain forms of insanity and other nervous disorders, such as mania, melancholia, imbecility, epilepsy, hysteria, or delirium tremens. Again, the *features* may give evidence of paralysis or of muscular twitchings or convulsions, as in chorea or epilepsy; squinting or alteration in the size of the pupils may also be observed. *d.* The **state of the skin and superficial structures** generally. Here may be noticed an abnormal colour; the presence of eruptions; undue dryness or moisture of the skin; excessive or deficient temperature; subcutaneous dropsy, either general or local. *e.* **Symptoms referable to special organs**. In many cases one or more characteristic symptoms, pointing to a certain organ or system, may be obvious at once. Thus there may be signs of dyspnoea; phenomena referable to the nervous system, such as delirium, unconsciousness, various forms of spasmodic movements, or paralysis; or vomiting and other symptoms pointing to the stomach. It must not be forgotten that the senses of *smell* and *hearing* may afford valuable assistance in this general examination. For instance, the breath may have some characteristic odour, such as that of

alcoholism, uræmia, or gangrene of the lungs; or the attention may be drawn to the larynx or trachea by the sound produced during the act of breathing.

2. Having completed this general survey, the next point is to ascertain how long the patient has been ill, so as to determine whether the malady is acute or chronic. Then enquiry should be made as to the prominent symptoms of which the patient complains, or which have been noted by others, and in the majority of cases attention will thus be directed to some particular region or organ, which is the seat of mischief. Should this not be sufficiently clear, a few leading questions may be put, so as to try to find out whether the malady seems to be localized in any part, or if it belongs to the class of general diseases, in the latter case the further investigation being guided by the information which has thus been elicited.

3. Should the clinical phenomena which are noted seem to point to some local morbid condition, investigation should then be directed first to the organ or part apparently involved, careful and complete enquiry being made with reference to the symptoms and signs known to be associated with it, of course employing *physical examination* whenever this can render any service. Then it must be borne in mind that many of the organs and systems in the body have a material influence upon each other in disease as in health, and therefore it is requisite in the next place to find out the condition of those organs which are most nearly related to that which is found to be primarily affected. After this a few questions may be asked, so as to find out the state of the other important systems of the body, even though no symptoms are complained of having any reference to them. Especially is it useful to pay attention to the digestive, respiratory, and circulatory organs, and in females to find out whether menstruation is normal. It is a good rule always to look at the tongue; to feel the pulse; to submit the lungs, heart, and vessels at least to a cursory physical examination; and to *test the urine*, particularly if the case under investigation is involved in any obscurity.

The discovery of the seat and nature of the malady from which a patient is suffering often affords valuable suggestions as to the points to be principally attended to in the history, in order to ascertain the ætiological facts bearing upon the case. In this way not only may the diagnosis be rendered more certain and clear, but most useful indications for treatment are frequently brought to light.

## VI. DIAGNOSIS.

It cannot be too often insisted upon, that to endeavour to arrive at a *satisfactory diagnosis* ought to be the first duty of a medical practitioner in every case which is brought under his observation and treatment. Now it is necessary that those who are studying medicine should understand at the outset what a *satisfactory diagnosis* means. It implies a complete, exact, and comprehensive knowledge of the case under consideration, as regards the seat, extent, origin, and nature of *all* existing morbid conditions. Of course such a diagnosis is not always attainable, but it is what should be conscientiously aimed at, and, if the observer has acquired the knowledge indicated in the previous chapters, and conducts his investigation with sufficient care



and thoroughness, a tolerably correct opinion can generally be formed. It is not an uncommon error to be content with merely ascertaining the chief symptoms present, perhaps giving a name to the group, such as dyspepsia; or to fix upon one prominent symptom, *e.g.*, ascites or jaundice, and to call that *the disease*, while no attempt is made to interpret the meaning of the phenomena which are observed, or to find out the pathological conditions upon which they depend. Again, when one or other of the organs is found to be structurally diseased, it often happens that very inadequate attention is paid to the determination of the precise locality, extent, and nature of the existing lesions, while the possibility of other organs being implicated is very liable to be overlooked, and thus the diagnosis is by no means so thorough and exact as it ought to be.

In attempting to form a diagnosis, a process of mental reasoning should be gone through, which needs to be more or less elaborate in different cases, according to their degree of difficulty, the facts elicited being passed in review, and certain conclusions founded upon them. The questions to be decided may be thus stated:—

1. Whether there is anything wrong at all?—for it must be borne in mind that not a few persons complain when there is no actual disease, especially among those who belong to the class of malingerers.
2. Should there be indications that the patient is really ill, it is requisite to determine:—*a.* Whether the ailment is *acute* or *chronic*? *b.* Does it belong to the class of *general* diseases, and, if so, what is its nature? *c.* Is the mischief localized in one or more of the organs of the body, or in some particular tissue? *d.* Should this be the case, is there merely functional disorder, or can any positive organic and structural change be detected? The seat, extent, and nature of all morbid conditions should then be made out as accurately as possible. It must not be forgotten that *local* lesions are frequently found accompanying so-called *general* diseases, such as the various fevers, and a diagnosis would be anything but complete in such affections, unless every care had been taken to ascertain whether any local mischief existed.

The exact mode of arriving at a diagnosis differs in different cases, while the degree of difficulty experienced in coming to a correct conclusion is necessarily very variable. In some instances we can make a *direct* diagnosis speedily and confidently, some combination of clinical phenomena, or some one or more pathognomonic symptoms clearly revealing the nature of the malady. In others the diagnosis has to be more or less *differential*, diseases which resemble each other being called up in the mind, and discriminated from each other. This is a less simple and easy process, and a very careful consideration of all the data which are available for assisting at forming a diagnosis is often required. These data, when the patient is first seen, are:—

1. The account which is given of the general history, family history, and previous health.
2. The history of the present illness, as to its duration, probable cause, mode of invasion, and progress.
3. The actual clinical phenomena observed, especially those of an objective character.

Even after the fullest consideration of all these points, it is sometimes impossible to come to any, or to more than a very doubtful conclusion. Under these circumstances it is exceedingly important not to form a hasty opinion, but to learn to wait and see what assistance the course of events may render. This rule is especially to be

attended to in cases of acute febrile diseases, otherwise very serious mistakes are liable to be made. The further elements in connection with the progress of a case which may aid diagnosis are:—4. Its clinical course, duration, and termination. 5. The phenomena observed on repeated examination under various conditions. 6. The results of treatment. In some obscure cases a diagnosis can only be made by *exclusion*, that is, by proving the absence of all diseases which might give rise to the symptoms observed, except one, the presence of which is therefore rather a matter of probability, than actually established by any positive signs. Occasionally it is quite impossible to come to any conclusion as to the nature of the malady from which a patient may be suffering.

In order to render the process of arriving at a diagnosis more easy and rapid, as well as to ensure greater certainty in the opinion formed, the following points are worthy of the attention of students. They should make themselves familiar with the more characteristic clinical signs of, at least, the *ordinary* individual diseases, that is with those symptoms which are diagnostic or pathognomonic. Then it is very useful to be able, speedily and without difficulty, to call up in the memory the complaints which have to be discriminated from each other in any case which comes under notice. Therefore it is necessary to bear in mind what affections resemble each other; to have the chief diseases of the several organs arranged under the two groups of acute and chronic; and to be quite familiar with the pathological causes to which any very prominent symptom may be due, such as dropsy or jaundice. Lastly, in making a diagnosis it must not be forgotten that irregularities and deviations from the usual clinical course of diseases are often met with in practice; that many affections present distinct and well-marked varieties; and that some serious complaints are liable to arise very insidiously, not being attended with any prominent clinical phenomena.

## VII. PROGNOSIS.

To “give a prognosis” is often a matter of considerable difficulty, and in many cases it involves an amount of knowledge and tact such as can only be acquired by prolonged experience. Here it is only practicable to give a few general hints bearing upon the subject. In the first place it is requisite to understand what questions have to be determined in forming a prognosis, and to try to realize distinctly which of these apply more particularly to any individual case, before offering an opinion concerning it. These questions have reference to the progress, ultimate issue, and duration of the case, and the following include the most important:—1. Whether the disease is more or less likely or certain to terminate in death or recovery; or to continue for an indefinite period as a permanent and incurable malady, but without causing any danger to life? 2. In case of death, may this event be expected to take place suddenly or slowly, and in what way will it probably be brought about? 3. If the patient recovers, will the cure be complete, or is there a danger of some morbid condition being left behind, either a state of general ill-health or some local organic lesion remaining? 4. What will be the probable duration of the complaint? 5. What events are liable to happen in its course, such as changes in symptoms, development of new symptoms, critical phenomena, the occurrence of

complications, &c. ? 6. Does its presence render the patient more amenable to other affections ; or, on the other hand, does it afford protection against certain maladies ? 7. May not slight symptoms observed be but signs and warnings of some more serious mischief which is likely to happen ? For example, numbness, tingling, slight local paralysis, and other apparently trivial nervous phenomena may be premonitory of some grave organic lesion in the brain.

Of course it will be impossible to give a reliable prognosis, unless a due knowledge has been acquired of the various points bearing upon this matter, in connection with each several disease, such as whether it is dangerous to life ; its rate of mortality ; usual modes of termination ; ordinary duration ; unfavourable symptoms ; complications and sequelæ ; and how it is influenced by accidental circumstances, whether dependent upon the patient or due to external conditions. It is always important to be cautious in offering any opinion as to prognosis, to give the matter due consideration, and to avoid anything like rashness or thoughtlessness. If there are good reasons for coming to a certain and definite conclusion, this ought to be stated confidently, and not with apparent doubt and hesitation. On the other hand, when the prognosis is questionable, it is a great mistake to give a positive opinion, but the state of affairs should be made as clear and explicit as possible to those interested, and the probabilities as to the result of the case pointed out, as well as the dangers which are liable to arise. It is better in doubtful cases to err in the direction of giving too hopeful an opinion rather than the opposite, especially with regard to acute diseases, as this often encourages perseverance in treatment upon which the issue may materially depend. Particular care is necessary in speaking to patients themselves about the prognosis, and it should be a rule to make it appear to them as favourable as possible, due regard being paid to any dangers against which they need to be warned. At the same time the friends should be fully informed as to the exact condition of things in every case in which the prognosis is at all grave. In a hopeless case, should the patient desire a positive opinion, it is the duty of the practitioner to give all the information required.

---

#### CHAPTER IV.

#### TREATMENT OR THERAPEUTICS.

THE ultimate and most important object of the study of Medicine, in a practical point of view, is to learn how to cure, relieve, or prevent the various maladies to which the human frame is liable. It needs to be particularly enforced at the present day that treatment may be made efficacious in accomplishing most beneficial results, if conducted properly and according to true principles. It must further be added, however, that in order to become competent to carry out treatment in a satisfactory manner much experience is required, as well as the constant exercise of intelligent and independent observation and thought. There is always a danger of falling into a mere routine treatment of particular diseases ; or of relying too implicitly on the experience and teaching of



others. These mistakes should be avoided, and each individual case ought to be considered on its own merits, for even the same disease may require very different management under different circumstances, and therefore the practitioner should be able to bring his own knowledge and common-sense to bear upon the matter, and to use his discretion in varying the measures employed. Before commencing treatment an endeavour should always be made to realize distinctly what it is intended to accomplish by its aid; and what indications are afforded as to the measures which require to be adopted.

It is requisite to make a few general remarks upon the *objects, indications, and methods* of treatment.

The *objects* which have to be kept in view in treatment may be stated as follows:—1. To **cure** the patient as speedily and completely as possible. This is termed *curative treatment*, but though it is a reality, it is applicable to only a limited number of diseases. 2. To **guide the progress** of a malady towards a favourable termination, when this is not directly curable, but must run a certain course, the objects being to avert death, and prevent permanent injury to health. This is termed *expectant treatment*, which may be illustrated by the treatment of many cases of the ordinary fevers, and it is very important that it should be duly recognized, as a great deal of mischief is often done by meddlesome interference, it being far better to let many diseases take their natural course, merely watching their progress, and only adopting active measures when circumstances seem to require them. 3. To **prolong life**, and render the condition of the patient as comfortable as possible, should a fatal termination be inevitable. 4. To **remove or relieve symptoms**. The result may be merely *palliative*, or sometimes in a sense *curative*. For instance, the removal of ascites and other forms of dropsy may practically be regarded as a cure, in so far that the patient may be able for many years to follow the ordinary avocations of life, although the organic disease upon which the dropsy depends is permanent. It is entirely wrong in principle merely to direct treatment to symptoms, or to attempt to relieve them at the expense of the general disease. At the same time they frequently need particular attention, and in some cases nothing further can be done than to endeavour to mitigate them. 5. To **prevent diseases**. *Preventive or prophylactic treatment* is of the deepest importance. It includes attention to the general health of an individual, so as to obviate any tendency to disease; the prevention of the extension of a disease in the same person or to other individuals, and the guarding against possible complications; the warding off of habitual attacks, such as those of acute dyspepsia, asthma, or epileptic fits; and the rooting out of various maladies, especially of constitutional disorders from the members of a family, and of contagious diseases from the midst of communities. In some instances all that can be done in the way of prevention is to warn patients against actions which may give rise to injurious consequences, and to ward off everything that might prove hurtful to them.

The *indications* for treatment are derived from—1. The nature and seat of the disease. 2. The causes which have led to it. 3. The personal conditions and surrounding circumstances of the patient. 4. The symptoms present, which may not only call for the adoption of certain measures, but may *contra-indicate* a line of treatment which would otherwise be followed. 5. The state of the system generally, and of the chief organs of the body. The condition of the lungs,

heart, and kidneys often influences treatment materially, which is one important reason for making it a rule always to examine these organs.

It will be found that there are certain indications to be recognized in the treatment of affections of each organ or system, common to them as a class, as, for example, those of the lungs, heart, or digestive organs. These should always be borne in mind, and in the subsequent chapters an endeavour will be made to generalize as much as possible the principles of treatment applicable to the diseases of the several organs.

The general *methods* of treatment may be summarized under the following headings :—

1. **Therapeutic**, which implies more particularly the *administration of medicines*, and there is no question but that by their proper employment much good may be done. There are a few drugs which undoubtedly exert a *specific curative* action upon certain diseases, and it is to be hoped that, as the result of the investigations which are now being made with regard to the action of medicines, many more *specifics* may be discovered. For the large majority of complaints, however, no curative medicine is known, and it is particularly necessary at the present time strongly to warn those entering upon the practice of medicine against believing in the so-called “specifics” for those diseases, such as phthisis, for which, from their very nature, no “specific” can ever be discovered. By using remedies in different doses and in various combinations, according to scientific and rational principles, we can modify materially the course of many affections, as well as exercise an important influence upon symptoms. It must not be forgotten that there are other modes of administering medicines besides by the mouth, especially by subcutaneous or intra-venous injections; by means of baths, inunction, or endermic applications; by enemata; and by inhalations.

2. **Diet and general hygiene**.—It is very necessary to bring this fact into special prominence, as it is apt to be frequently forgotten, namely, that *treatment does not consist solely in the administration of medicines*. In not a few cases these are not required at all, or they hold a very secondary place in point of importance, while their beneficial action may be almost invariably assisted more or less by paying due regard to the measures to be now mentioned. Attention to *diet* is often of the greatest consequence, proper directions being given, not only as to the nature of the food and drink, but also as to its quantity, the intervals at which it should be taken, and other matters which individual cases might suggest. The use of *alcoholic stimulants* always demands the utmost care and consideration. They ought never to be recommended in an off-hand manner, or unless it is felt that they are really required; while as definite instructions as possible should be given with regard to the kind and amount of stimulant which should be taken, and other particulars, especially when it is deemed advisable to order spirits. It will often be found necessary to limit the consumption of stimulants, as habitual indulgence to excess is a common cause of ill-health. It is also essential always to keep in mind various matters connected with *general hygiene*, as these frequently need to be looked into, and have an important influence in treatment, such as the place of residence of the patient, with its surrounding conditions; habits of life; occupation; clothing; the kind and amount of exercise;

and the necessity for change of air or climate. It should be remembered that it is the duty of the practitioner to be prepared, if required, and especially in cases of acute febrile diseases, to attend personally to questions pertaining to diet and hygiene, so as to ensure that his instructions are duly carried out. Thus it is often advisable to examine food, such as beef-tea, and to see that it is of the proper kind and properly made; also to look to the conditions of the sick-room, especially as to ventilation and cleanliness, temperature, the state of the bed, and the removal of excessive curtains or carpets. If a nurse is needed, the practitioner should ascertain that she is competent and reliable, as upon her skill and attention the ultimate issue of many cases turns.

3. **Local and external applications.**—These are often most serviceable in treatment, such as hot fomentations or poultices, cold applications, baths, liniments, ointments, lotions, blisters, sinapisms, plasters, mechanical appliances, electricity and galvanism, and gargles. In this connection may also be mentioned the employment of friction, shampooing, kneading, passive movements, and similar measures, which are sometimes very useful.

4. **Operations** are not uncommonly required even in medical practice, for instance, venesection or the local removal of blood, paracentesis, acupuncture, the use of the aspirateur, or tracheotomy. When any such operation is clearly indicated, there ought to be no unnecessary delay or hesitation in having recourse to it.



## SECTION II.

In the present section it is proposed to consider certain morbid conditions which are included under GENERAL PATHOLOGY, of which it is most desirable to have a comprehensive knowledge before studying them in connection with special organs or tissues.

## CHAPTER I.

## HYPERÆMIA OR CONGESTION.

**HYPERÆMIA** OR **CONGESTION** signifies the presence of an excessive amount of blood in a part. According to its cause, and to the vessels in which the accumulation chiefly occurs, the hyperæmia is said to be:—**I. Active or Arterial: II. Mechanical or Venous: III. Passive or Capillary.** Each of these forms requires separate consideration.

**I. Active or Arterial.—Determination of blood.**—In this form the arteries are chiefly implicated, being dilated, while there is an increased afflux of blood through them, though usually too much passes out by the veins as well, and the circulation is accelerated.

**ÆTIOLOGY.**—1. Weakening or paralysis of the muscular coat of the arteries is the usual cause of active congestion, in consequence of which they yield to the normal pressure of the blood. It is well known that this coat is under the control of vaso-motor nerve-fibres, which pass from the spinal cord, through the sympathetic, and these exercise an important influence on certain pathological processes, including active congestion. This vaso-motor paralysis may be produced:—*a.* By direct lesion of the spinal cord in experiments, or as the result of injury or disease. *b.* By compression of, or injury to the sympathetic trunk, of which the congestion resulting from the pressure of an aneurism upon the sympathetic nerve in the neck is an example; or by section of vaso-motor nerves in any part of their course. *c.* By reflex irritation through the sensory nerves, such as the congestion induced by friction, or by the application of a mustard poultice, heat, or cold to the skin; or that resulting from the excessive use or increased activity of an organ, such as the eye or mammary gland. Under the same category may be mentioned the congestion which often attends severe neuralgia, although this has also been attributed to irritation of supposed vasodilator nerves. Probably many cases of congestion of internal organs originate in this way. It may further account for the active congestion in the vicinity of morbid growths; and may be the first step towards inflammation. *d.* By causes acting through the brain, which may be illustrated by emotional blushing, and by the effects of certain drugs and poisons. Some of these, however, seem to have a direct effect in paralyzing vaso-tonic nerves.

2. The rapid withdrawal of external support from arteries may cause them to be dilated, and thus lead to active congestion. This is seen in the effects following the application of a cupping-glass to the surface,

by which the pressure of the atmosphere is removed; or those produced by the rapid removal of ascitic or pleuritic effusions.

3. The internal pressure on the vessels may be increased, either from an augmented force on the part of the heart, which principally affects the structures supplied by the arteries given off from the arch of the aorta; or from some channels being narrowed or obliterated, the blood having therefore to find its way along those which are pervious, consequently distending them unduly. One of the best illustrations of the latter is the "collateral circulation" which is speedily set up when a main artery is tied, or when it is suddenly blocked up in any way. Internal congestions are also often due to this cause, as when they follow exposure to cold, which leads to contraction of the small vessels of the skin, and thus the blood is driven inwards. If the walls of the arteries are weakened from any cause, and are deficient in tone, they are more liable to yield and to become dilated.

**SYMPTOMS AND EFFECTS.**—The objective signs of active congestion are more or less bright redness, with turgescence, and increased temperature. Pulsation in the arteries is increased, and may be visible; while secretions are often more profuse or altered in quality. Ultimately the vessels may be so distended as to transude serum, or even to rupture and give rise to hæmorrhage. The subjective sensations are usually those of heat, fulness, and throbbing; the functions of an actively congested organ are often materially and seriously interfered with, as, for example, those of the nerve-centres or lungs. If the congestion continues for a long period, it may lead to permanent hypertrophy or to induration of tissues; the arteries may also become persistently dilated and thickened.

**II. Mechanical or Venous.**—Here there is no excess of blood entering a part, but a difficulty is experienced in its passage through the veins, which therefore, as well as the capillaries, become unduly filled with dark blood, moving slowly and languidly. This is a very important form of congestion, and often leads to troublesome symptoms calling for the attention of the physician.

**ÆTIOLOGY.**—1. Some mechanical obstacle to the circulation of the blood through the veins is the most important cause of this form of congestion. This may be seated in the heart, thus affecting the entire systemic or pulmonary circulation, or both, according to the precise locality of the impediment. Or it may only involve some special vein or system of veins, such as the portal, or those of a limb, the obstruction being correspondingly localized. The cause of the impediment may be either something within the vessels, such as a clot; constriction from morbid changes in their coats; or external pressure upon them by a tumour, ligature, fibrous thickening, or other conditions. It may be mentioned under this head that incompetence of the valves of a vein, from any cause, materially aids in maintaining venous congestion.

2. Parts which are in a dependent position are very prone to become venously congested from the mere influence of gravitation, and this is especially apt to occur if the tissues are relaxed and yielding, or if the *vis a tergo* is deficient. Illustrations of this mode of origin are presented in the congestion of the veins of the legs which follows prolonged standing; and in the formation of hæmorrhoids as the result of sedentary occupations.

3. A diminution in the *vis a tergo* often either itself gives rise to mechanical hyperæmia, or assists other agencies in its production. The

heart may be weakened in its action, or the arteries may be impaired as to their elasticity and contractility, owing to degeneration or other causes, and thus the forces which carry on the circulation are inadequate to drive the blood through the veins. This is especially observed in old people.

**SYMPTOMS AND EFFECTS.**—The objective signs of venous congestion, when the condition is superficial, are redness of a dull, dusky, purplish, or livid hue, with frequently evident distension of the capillaries and veins, the latter being often knotted; increase in bulk of the part affected; and not uncommonly a lowering of temperature. After a time the watery part of the blood transudes in excess, containing some of its solid constituents in solution, and thus *dropsy* is produced, which gives rise to still greater enlargement, with a feeling of softness and pitting on pressure. In some cases a fibrinous material is exuded, imparting to the structures implicated a firm and brawny feel, as may be observed after obstruction of the veins of the leg in phlegmasia dolens. When the kidneys are the seat of venous congestion albumen is often present in the urine; in connection with mucous surfaces the same condition leads to a watery flux; while in serous membranes it leads to dropsical effusions.

If the congestion is still more intense the colouring matter of the blood passes out; or the corpuscles migrate through the walls of the vessels into the surrounding tissues; or finally the vessels themselves give way and hæmorrhage occurs, as is observed in some cases of varicose veins, and in the bleeding into the stomach or intestines which may follow obstruction of the portal vein. This event is especially liable to happen if the vessels are weakened, and the bleeding may take place into the substance of organs as well as on free surfaces.

Should the congestion be very great, rapidly produced, or long-continued, it will lead to serious interference with nutrition, which may end in ulceration or gangrene. Occasionally a *thrombus* or clot is formed in connection with a congested vein, for example, in the portal vein in cases of cirrhosis of the liver. With regard to organs or tissues, mechanical congestion causes them in course of time to become enlarged or thickened, while a fibroid material is formed, giving rise to induration, stiffness, and loss of elastic and contractile properties. Changes in colour are also not uncommonly noticed, to grey, brown, or black, due to alterations in the blood-pigments. Ultimately organs may become much contracted and diminished in size, indurated, and seriously disorganized as regards their structure.

The subjective symptoms associated with venous congestion will necessarily vary according to the part affected. Generally there is a sense of weight or dull heavy uneasiness, while the functions of the organ or structure involved are impaired to a greater or less degree. External parts which are the seat of venous congestion often feel cold and numb.

**III. Passive or Capillary.**—Many include *passive* under mechanical congestion, but there is a distinction between them, though they are often associated. In passive congestion the capillaries are mainly involved, the circulation being languid in these vessels, owing to a disturbance of the vital and nutritive relations existing between the elementary tissues and the blood. The conditions of a part thus congested are very similar to those observed in mechanical congestion. Atrophy of, and degenerative changes in, the tissues are liable to occur;



while they become prone to low and asthenic forms of inflammation, tending to assume a chronic character.

ÆTIOLOGY.—1. A weak state of the general system may induce passive congestion, owing to the feeble activity of the circulation, and the impaired nutrition and want of tone in the tissues. It especially affects parts which are dependent, or which are distant from the heart, as evinced by coldness and blueness of the extremities, nose, and ears. The so-called *hypostatic* congestions which are met with in various low fevers and debilitating diseases also come partly under this category.

2. Morbid conditions of the blood may cause passive hyperæmia. That which accompanies imperfect aëration of the blood is considered by some to be of this nature; and deficiency of fibrinogenous elements also favours its occurrence.

3. If an organ or part is debilitated from any cause, and the functions of its tissues are impaired, it is prone to become the seat of passive congestion. As illustrations of this may be mentioned the congestion which is often observed in paralyzed limbs; and that which follows excessive functional activity of an organ, whereby it has become exhausted. It may also succeed active congestion or inflammation, owing to the perverted relations thus set up between the blood and the tissues, as is frequently noticed after tonsillitis.

ANATOMICAL CHARACTERS.—Redness, varying in its tint and form according to the nature of the congestion, is the essential anatomical character of all the varieties of this morbid condition. In *active* congestion the colour is bright red, and it usually assumes the form of a minute net-work, but may appear to be uniform or in points, when certain special structures are involved. It must be borne in mind, however, that there may have been active hyperæmia during life, and yet no redness be apparent after death, owing to the arteries having contracted and expelled the blood into the veins. Points of redness are sometimes seen, which are due to minute extravasations of blood. The colour of *mechanical* or *passive* congestion is generally more or less dark red, but it may present a blue, purple, or livid tint; while the veins are often visibly distended, and form a net-work.

Organs are often said to be congested in *post-mortem* descriptions, when this has not been the case in reality during life, simply because the blood has gravitated after death into dependent parts, and has thus given rise to this appearance. Tissues also are subject to *post-mortem* staining by the colouring matter of the blood, which may simulate congestion. The results of congestion already described are frequently evident after death, such as dropsy or hæmorrhage, and if it has been long-continued, considerable changes in the physical characters and structure of organs may be observed.

TREATMENT.—All that can be done here is to point out the principles which are to be borne in mind in the prevention and treatment of congestion, the particular measures to be adopted necessarily varying much in different cases. The indications are:—1. To remove the *cause* of the hyperæmia, if possible, especially should this be of a mechanical nature. 2. To attend to *position*, so as to obviate the effects of gravitation, and assist the passage of blood through, and its return from, the congested part. 3. To modify the *general circulation* of the blood, by acting upon the heart and vessels, either reducing its force when this is excessive, or assisting it when it is languid and feeble.

4. To diminish the *quantity* of the blood, either by venesection, or by local methods, such as the application of leeches or cupping. 5. To *draw blood away* from the seat of congestion, by means of sinapisms, heat or other irritants applied to the skin, dry-cupping, and similar agencies. 6. To employ *local measures*, with the view of diminishing the quantity of blood in the affected region, such as the application of cold or pressure; or intended to promote its circulation, for instance, friction, shampooing, or galvanism. 7. To alter the *quality* of the blood, employing low diet, purgatives, diuretics, and such remedies if there is plethora; improving its quality, by the administration of good food and preparations of iron, if the blood is impoverished. 8. To improve the condition of the *general system*, when this is below par, especially in cases of passive congestion. It may be remarked that it is particularly important to endeavour to prevent and relieve congestion in febrile diseases; not to allow mechanical congestion to continue for any length of time if it can be avoided; and to warn aged persons against performing acts which lead to sudden temporary congestion, as their vessels are very liable to give way.

---

## CHAPTER II.

### DROPSY—HYDROPS.

DROPSY is only a symptom or pathological condition, though often a very important one, associated with certain general or local diseases. It consists in an accumulation of serous fluid, which has escaped from the blood-vessels, either in the subcutaneous or submucous cellular tissue, in serous cavities, or in the cellular tissue of certain organs; it may occupy all these parts at the same time. The following terms are used to express the site of the dropsy:—Dropsy of the subcutaneous cellular tissue, if at all extensive, is named *anasarca*, if localized, *œdema*; *hydrothorax* signifies dropsical accumulation in the pleuræ; *hydropericardium* in the pericardium; *ascites* in the peritoneum; *hydrocephalus* in the ventricles of the brain or the arachnoid cavity; dropsy of organs is termed *œdema*, for example, *œdema* of the lungs. When dropsy involves both the subcutaneous cellular tissue and serous cavities, it is said to be *general*.

It is necessary to mention certain morbid conditions which are known as *spurious dropsies*, but which really have no pathological relation to dropsy. They include ovarian dropsy, which is a cystic disease of the ovary; accumulations of fluid in the interior of hollow organs, as the result of obstruction at an orifice, or of inflammation, such as dropsy of the uterus (*hydrometria*), or of the gall-bladder; certain serous effusions consequent upon inflammation, for instance, *hydrocele*, and acute *œdema* of the glottis; dropsy of the kidney (*hydronephrosis*), which is either due to cystic disease, or, more frequently, to obstruction of the ureter and consequent accumulation of urine and inflammatory products within the pelvis of the kidney, which gradually destroy this organ.

**PATHOLOGY AND ÆTIOLOGY.**—A dropsical accumulation is the immediate result, either of excessive flow of fluid out of the vessels; of deficient absorption; or of both combined: in short, the balance between exhalation and absorption is in some way disturbed. This derangement may be due to the following pathological conditions:—

1. *Over-distension of the vessels*, in the different forms of congestion, but especially that dependent upon mechanical interference with the return of blood through the veins, is one of the most common causes of dropsy, which is then due both to an excessive escape of fluid from the vessels, and to their diminished power of absorption. Obstruction to the circulation on the right side of the heart thus causes more or less general dropsy, beginning in the feet and ankles, and extending upwards; the same result may follow serious impediment to the circulation through the lungs. Obstruction on the left side of the heart leads to œdema of the lungs, because the pulmonary vessels are distended. Any local obstacle may originate correspondingly limited dropsy. Thus, interference with the portal circulation is followed by ascites; a clot in a principal vein of the arm or leg, or external pressure upon it, will give rise to œdema of the affected limb. Hydrocephalus is chiefly the result of pressure upon the small veins returning the blood from the ventricles of the brain. Gravitation necessarily influences much the seat of the congestive form of dropsy, and may itself induce it under certain conditions. Active congestion does not give rise to any great amount of dropsy as a rule, but it often causes local œdema.

2. A *feeble and relaxed state of the vessels and tissues*, in consequence of which the former readily yield and allow transudation of fluid, often aids in the production of dropsy. The œdema of the feet and ankles which is met with in some cases of general debility is partly due to this cause, being assisted by feeble cardiac action, which induces mechanical congestion.

3. An *unhealthy condition of the blood* may occasion dropsy, especially if this fluid is very watery, deficient in albumen, or impregnated with certain morbid materials, such as urea. Under these circumstances its liquid portion more readily transudes through the walls of the vessels. This cause often aids materially in the production of all forms of dropsy, but it is most important in connection with the anæmic and renal varieties.

4. It has been asserted that dropsy depends chiefly on a *withdrawal of nervous influence* from the vessels, and experiments have been made to prove that so long as the nerves remain intact dropsy will not occur, even though the veins are over-distended. That the nervous system does exercise considerable control over the processes of exhalation and absorption by the vessels is beyond doubt, and it must therefore influence the occurrence of dropsy, but there is not sufficient reason to believe that it occupies the important relation to this symptom attributed to it by some pathologists. Œdema is not uncommonly observed in paralyzed limbs.

5. It is highly probable that a *deficient power of absorption* on the part of the *lymphatic vessels* assists in giving rise to dropsy in some instances. Possibly this may exercise an influence in certain cases of cardiac dropsy, the chief lymph-ducts being unable to empty themselves into the distended veins.

Such being the immediate pathological conditions which explain the



occurrence of dropsy, and which are often more or less combined, its more obvious causes may be summed up as follows :—

1. *Any cardiac disease that interferes with the circulation of the blood, and leads to overloading of the veins and capillaries.* The most important are affections of certain of the orifices and valves of the heart; dilatation of its cavities; and degeneration of its walls, with consequent weak action. The heart may also be displaced, or pressed upon by morbid conditions external to it; or it may be extensively adherent.

2. *Affections of the lungs impeding the circulation.* When acute bronchitis complicates extensive emphysema, considerable dropsy may supervene. Pulmonary affections also not uncommonly aggravate cardiac dropsy.

3. *Diseases of the kidney attended with deficient elimination of water and urea, but allowing the escape of albumen in the urine.* As a consequence the blood is impoverished and impure, and the vessels are over-distended. Scarlatina demands special mention in this connection as a cause of dropsy, which generally depends upon acute renal inflammation when associated with this disease.

4. *Diseases of the liver, or any other morbid condition causing obstruction to the portal circulation.* This is a local form of dropsy, resulting from mechanical congestion of the tributaries of the portal vein.

5. *Exposure to cold and wet, or anything occasioning a chill.* This cause is generally supposed to act by driving the blood inwards, and inducing active congestion, the resulting dropsy being named *active* or *febrile*. It chiefly acts, however, by checking elimination by the skin, and at the same time giving rise to venous congestion of the kidneys, these organs being consequently unable to perform their functions properly; hence the vessels become overloaded, and the fluid portion of the blood transudes.

6. *Any local obstacle in connection with a particular vein.* Local dropsy is not uncommonly due to this cause, resulting from the pressure of a pregnant uterus, ovarian and other tumours, enlarged glands, or aneurisms; as well as from inflammation of veins, varicose veins, and thrombosis.

7. *Gravitation of the blood into dependent parts.* Prolonged standing may of itself lead to dropsy, especially if the blood is watery, and the tissues are wanting in tone.

8. *Causes which impoverish the blood.* Dropsy may be induced or aided by a want of proper diet, especially if combined with other unfavourable hygienic conditions; by hæmorrhage or excessive discharges, either natural or morbid; and by various acute or chronic diseases, such as fevers, especially malarial, phthisis, cancer, splenic disease, affections of the absorbent glands, scurvy, or purpura.

9. *Certain conditions leading to active congestion.* Dropsy is said occasionally to follow the rapid disappearance of chronic skin-diseases, or the sudden suppression of habitual discharges, and is then believed to result from active congestion. This may also be due to the irritation of some morbid deposit, such as tubercle or cancer; and it accounts for the œdema often observed in the neighbourhood of inflamed parts.

It must not be forgotten that in many individual cases more than one, it may be several, causes have been instrumental in originating dropsy.

**ANATOMICAL CHARACTERS.**—The seat and extent of dropsy vary considerably in different cases, as already pointed out; and the same remark applies to the quantity of fluid accumulated. Dropsical fluid presents the following characters:—It is almost always thin and watery; either quite colourless or light-yellow as a rule, but sometimes tinged by the colouring matter of the blood or of bile; clear and transparent, or rarely opalescent; usually varying in its specific gravity from 1008 to 1012 or 1014. Its reaction is generally alkaline, but occasionally neutral or slightly acid. Chemically it is allied to the serum of the blood, consisting of water holding in solution albumen, alkaline and earthy salts, especially chlorides, and extractive matters, but the proportion of these ingredients varies much in different parts and in different cases, especially the amount of albumen, and the composition is never identical with that of blood-serum, the proportion of solids being much less. Fat, especially cholesterin, fibrin, or pigments are sometimes present; and urea may be found in one special form, namely, in renal dropsy.

**SYMPTOMS AND COURSE.**—As a rule dropsy comes on more or less gradually, but sometimes its progress is extremely rapid, and it may extend over the whole body in a few hours. It usually appears first, and is most abundant in dependent parts, especially such as are distant from the heart; in those which are exposed; or in regions where there is much loose cellular tissue. It is liable to vary with position, being necessarily influenced by gravitation.

The objective signs of anasarca or œdema are swelling of the affected part, and superficial pitting on pressure, the skin being generally pale, but sometimes congested. The degree of distension varies much; it may be so great as to cause the skin to assume a tense, shining aspect, or even to burst or slough. The vitality of dropsical tissues is impaired, and hence they are very liable to erysipelatous and other forms of low inflammation, either spontaneously or from slight irritation. When fluid accumulates within serous cavities, it may or may not produce evident enlargement, but its presence can be made out in most cases by certain “physical signs,” to be hereafter described. The subjective symptoms accompanying dropsy of external parts may amount to more or less discomfort or uneasiness, and a feeling of tightness or stiffness, but no actual pain or tenderness is experienced. An accumulation of dropsical fluid interferes mechanically with organs, and may thus cause most serious disturbance of their functions. In certain parts it may lead to a rapidly fatal issue, as, for instance, when œdema occurs in the neighbourhood of the glottis.

The general symptoms will necessarily vary according to the cause of the dropsy. If it is at all considerable in amount, the normal secretions are as a rule deficient in quantity.

**DIAGNOSIS.**—It is usually not difficult to determine *whether dropsy is present*, when it is looked for. There is a peculiar condition, named *myxœdema* by Dr. Ord, which might be mistaken for general dropsy, but is of a different nature. (See MYXŒDEMA.) The chief point in diagnosis is to make out the *cause* of dropsy. In order to ascertain this, of course it is necessary to enquire into the history of the patient; to observe what other symptoms are present, both local and general; and to examine carefully those organs, diseases of which are known to occasion dropsy. Much help may, however, be derived from a consideration of certain facts with regard to this particular symptom, namely:—

1. **Its place of origin, seat, and extent.**—*Cardiac* or *pulmonary* dropsy begins in both feet and ankles, and extends upwards, ultimately becoming more or less general. *Ascites* only follows, with rare exceptions, after the circulation through the liver has been for some time obstructed. *Renal* dropsy frequently starts in the face and upper part of the body, especially about the eyelids, where there is much loose cellular tissue, and in the hands, because they are exposed. It may rapidly spread all over the body, and involve all the serous cavities, though not usually to a great extent. *Hepatic* dropsy is confined to the peritoneal cavity at first, because the portal system is alone interfered with. The abdomen may become considerably distended before any dropsy is observed elsewhere, but in most cases after a while anasarca of the legs sets in, in consequence of the pressure exercised by the accumulated fluid upon the *vena cava inferior*. Anasarca of the legs and ascites may appear simultaneously, should there be any pressure upon the *inferior vena cava* just before it passes through the diaphragm; this vessel may also be obstructed lower down, giving rise to dropsy of both legs. *Anæmia* alone never causes much dropsy; it is always limited to the subcutaneous tissues; and is usually only seen about the feet and ankles, or in the loose tissue of the eyelids. *Local* dropsy, as, for instance, œdema of one leg or arm, always indicates some local obstruction of a vein. Rarely the *superior vena cava* is pressed upon, and dropsy of the upper part of the body is one of the consequences.

2. **Its rate and mode of progress.**—*Cardiac* dropsy is generally slow and gradual in its progress, liable for a time to vary more or less in amount, according to position, but ultimately this does not influence it much. It may increase rather quickly, in consequence of some acute pulmonary complication. *Renal* dropsy, if acute, may be extremely rapid in its course, in some cases producing enormous enlargement of the whole body, and obliterating the features in a few hours. This is the only form of dropsy in which such a mode of progress is observed; it may also disappear in the same rapid manner. *Hepatic* dropsy usually progresses slowly and steadily as a rule. That of *anæmia* comes and goes easily, being often present about the feet in the evenings, but disappearing with a night's rest, while the eyelids are puffy in the mornings.

3. **The effect of pressure** is said to distinguish between *cardiac* and *renal* dropsy, when subcutaneous, but this is a very unreliable sign. The latter is stated to pit much less, and to retain the impression of the finger longer, elasticity not being quite lost.

4. **The appearance** of a dropsical part may assist the diagnosis. Thus in some cases of *renal* disease the skin presents a very peculiar dull-white, pasty aspect. In *cardiac* dropsy signs of venous congestion are often present, and the skin may be shining and tense.

5. **Characters of the fluid.**—That of *renal* dropsy is of a very low specific gravity, containing only a small quantity of albumen, and urea can in some instances be detected in it.

6. **The effects of treatment.**—The dropsy of *anæmia* is easily got rid of; the *renal* form can frequently be removed for a time, or permanently, by appropriate treatment; it is often difficult to bring about absorption of *cardiac* dropsy, if it is at all considerable in amount, and it is liable to return speedily.

**PROGNOSIS.**—The chief questions with which the prognosis of dropsy is concerned, are its immediate danger to life; the probability of curing



it permanently; and its temporary removal or alleviation. Caution should be exercised in giving an opinion, especially an unfavourable one, for cases which seem quite hopeless sometimes improve in a remarkable manner. The main data upon which the prognosis is founded are:—1. The causes of the dropsy, and the possibility of removing such causes, special attention being directed to those organs which are so frequently accountable for this symptom. 2. Its seat, dropsy of some structures, for example, cedema of the larynx or lungs, being immediately dangerous to life; while in other parts it is very difficult to get rid of. 3. Its extent over the body, and the quantity of fluid accumulated. 4. Its duration and progress, acute and rapidly-spreading dropsy being highly dangerous, but at the same time often more easily dispelled than that which is chronic and steadily progressive. 5. The possibility of adopting appropriate treatment, and the effects resulting therefrom. This applies particularly to those active measures which have for their object the absorption of the fluid, much depending upon the strength of the patient, and his ability to undergo the requisite treatment, as well as upon the state of those organs which are directly acted upon. 6. The condition of dropsical parts, there being more danger if their nutrition is obviously impaired, or if they are the seat of any form of low inflammation.

**TREATMENT.**—The objects to be kept in view in the treatment of dropsy are:—1. Its removal or cure. 2. The prevention of its recurrence. 3. The prevention and management of its injurious effects. The particulars of treatment must necessarily be governed by the cause of the dropsy, and the condition of the several organs, but there are certain general principles which need attention, of which an outline will now be given, as well as of the means by which they are to be carried out.

1. **Removal of the cause.**—As illustrations may be mentioned the relief of any pressure or constriction affecting a vein; or of an attack of acute bronchitis in cases of cardiac disease, which may seriously aggravate dropsy due to this cause. Of course it is highly important to attend specially to any organ, a morbid condition of which is keeping up dropsy, and to try to cure the disease, or at all events to render the organ capable of performing its functions, so far as this is possible.

2. **Attention to rest, position, and regulated pressure.**—Far too little heed is usually paid to the influence of *rest* and *position* in the treatment of dropsy. The part affected should, if necessary, be maintained continuously and for a long time in an elevated position. Much benefit may often be obtained by keeping the legs, if they are the seat of anasarca, on a level higher than the body; or by raising an œdematous scrotum by means of a pillow of cotton-wool placed underneath. *Pressure* is also very valuable in many cases, if carefully and properly applied.

3. **Promotion of absorption of the fluid.**—This indication is carried out by the use of *diaphoretics*, saline and hydragogue *purgatives*, or *diuretics*, so as to promote free secretion by the skin, intestines, or kidneys respectively, and thus to remove more or less of the watery portion of the blood.

The only *diaphoretic* that usually proves of much practical value in the treatment of dropsy is some form of bath which promotes perspiration, such as the warm, vapour, hot-air, or Turkish bath. Either of these may be used as frequently as circumstances require; and local baths may sometimes be employed with much advantage if the patient

cannot sustain general baths. It is in the treatment of renal dropsy that they are most valuable, and especially of acute cases. An occasional bath is also useful in preventing this form of dropsy. Diaphoretic medicines are often given, such as ipecacuanha, antimony, spirits of nitre, liquor ammoniæ acetatis, or citrate of potash, but they afford little or no assistance from their diaphoretic action in the removal of dropsy. Jaborandi has proved serviceable in some cases.

*Watery purgatives* are frequently highly efficient in relieving dropsy, but care must be exercised in their administration, as they tend to weaken a patient. The most important are extract of elaterium (gr.  $\frac{1}{6}$  gradually increased to gr.  $\frac{1}{2}$ ); jalap (℥ i to ʒ i); and cream of tartar (ʒ i to ʒ ij); the last two form a very effective combination, as in the compound jalap powder. They may be given two or three times a week, or oftener if required. Dr. Matthew Hay has recommended the administration of a concentrated solution of a saline purgative, especially sulphate of magnesia, in the treatment of certain cases of dropsy. It thus acts both on the intestines and the kidneys; and rapidly reduces the fluids of the blood. He has found this treatment more useful in general dropsies than in local dropsies; and most beneficial in those depending on a stasis of the circulation, as cardiac dropsy. As requisite conditions he recommends that the patient should abstain from food for some hours previously; and that the salt should be administered along with the smallest possible quantity of water. Other strong purgatives are employed in treating dropsy, such as gamboge, veratrum, podophyllin, calomel, or croton oil, but these are much less admissible ordinarily, though some of them are occasionally serviceable. There can be no doubt that the effect of pills administered by certain quacks, which is sometimes really marvellous, is due to powerful purgatives which they contain.

*Diuretics* are most beneficial in some forms of dropsy. Those usually given are the nitrate, acetate, or citrate of potash or soda in full doses, freely diluted; cream of tartar in small doses; spirits of nitre; infusion or tincture of digitalis, or the powdered leaf made up into pills with other ingredients; squill in the form of tincture or pill; spirits or infusion of juniper; infusion of fresh broom tops; or oil of turpentine. The balsam and resin of copaiba have been found efficacious in the treatment of ascites and other forms of dropsy. Caffein is also a valuable diuretic. The following pill is sometimes very efficacious in relieving dropsy, given about every other night:—R. Ext. elaterii, gr.  $\frac{1}{6}$  to gr.  $\frac{1}{2}$ ; Pulv. scillæ, gr.  $\frac{1}{2}$  to gr. i; Pulv. digitalis, gr.  $\frac{1}{2}$  to gr. i; Ext. hyoscyami, gr. 1½. M. fiat. pil. Another pill which has been much recommended consists of digitalis, squill, and blue-pill. Digitalis is also used as an external application, poultices of the leaves being placed over the abdomen, or fomentations of its infusion being employed. Gin or whiskey freely diluted undoubtedly acts as an efficient diuretic in the treatment of some cases of dropsy.

Blood-letting has been recommended under certain circumstances, with the view of unloading the vessels, and thus assisting the action of other remedies, but such treatment can rarely, if ever, be indicated.

4. **Removal of the fluid by operation.**—If dropsy cannot be got rid of in any other way, it is necessary to have recourse to certain operations, and I believe that these are often delayed until too late a period, and ought in appropriate cases to be performed, not as last resources, but as curative measures. These operations include:—1. *Paracentesis* or *tapping* of serous cavities, especially to be adopted in certain cases of

ascites. 2. *Acupuncture* or *scarification* of the skin, or the introduction of small canulæ into the subcutaneous cellular tissue, in cases of anasarca. It is generally quite sufficient to make several superficial punctures with an angular needle in dependent parts, repeating them as often as may be required, and taking care that the punctured spots are not inflamed by urine or other sources of irritation. Dr. Southey has, however, introduced a really useful and efficient mode of treatment which is applicable to many cases of anasarca, namely, the introduction through the skin of small canulæ by means of a trochar, these being left in after the withdrawal of the trochar, so that the dropsical fluid may drain away through them, a drainage-tube being attached to each canula. By this method a considerable quantity of fluid is often rapidly removed. Occasionally it becomes requisite to make free scarification or incisions.

5. **Improvement of the condition of the general system and blood.**—Treatment directed to this object is generally of much service, and it may be the chief or only thing called for, as in cases of dropsy due to anæmia. The digestive and nutritive functions must be attended to, as well as the diet, which should be of a nutritious character, without much liquid. All hygienic conditions must be properly regulated. Tonics are often indicated, and above all some preparation of iron, especially the tincture of the perchloride, which has a marked influence upon the composition of the blood.

6. **Prevention of irritation of dropsical parts.**—It is important to keep all external dropsical parts clean and dry; to prevent them from being unduly pressed upon; and to ward off all other sources of irritation. Should erythema, erysipelas, or other complications supervene, they must be treated accordingly.

---

### CHAPTER III.

## HÆMORRHAGE.

HÆMORRHAGE signifies an escape of blood out of the current of the circulation, either from the heart itself, or from the arteries, capillaries, or veins. *Capillary* hæmorrhages are most frequent in medical practice. Generally the vessels are obviously ruptured, but it is supposed that bleeding may occur without actual destruction of their walls, as it may be impossible to discover any lesion, even on the most careful examination, and it is known that the blood-corpuscles can penetrate the coats of the vessels.

The blood may be poured out on a free cutaneous, mucous, or serous surface; or into the interstices of tissues, the substance of organs, or morbid growths. An accumulation of blood in a solid organ or mass of tissue is named an *extravasation*, *apoplexy*, or, under certain circumstances, a *hæmorrhagic infarct*; subcutaneous hæmorrhages assume the form of *ecchymoses* or more or less extensive patches, *stigmata* or minute points, *petechiæ* or rounded spots, and *ribices* or lines.

Special names are used to indicate whence the blood comes, of which the chief are *epistaxis* or bleeding from the nose; *hæmopytisis*, from the



air-passages or lungs; *hæmatemesis*, from the stomach; *melæna*, from the bowels; *hæmaturia*, from the urinary organs; and *menorrhagia*, from the female genital organs.

Certain general terms are also often applied to hæmorrhages, with the view of classifying them, the meaning of which is sufficiently obvious. Thus they are said to be *traumatic* or *spontaneous*; *idiopathic* or *symptomatic*; *active* or *passive*; *arterial*, *venous*, or *capillary*; *vicarious*, *critical*, or *periodical*.

**ÆTIOLOGY.**—The causes of hæmorrhage may be thus arranged:—

1. **Traumatic.** A vessel may be directly injured by a cut or contusion; by hard and rough substances, such as a calculus in the bladder, a foreign body, or dry hard fæces in the alimentary canal; or by the destructive effects of ulceration, gangrene, or cancer. 2. **Congestion, leading to extreme distension of the vessels.** All forms of congestion may end in hæmorrhage, but especially if the force of the circulation is at the same time much increased, the pressure becoming so great as to cause the vessels to give way. Therefore whatever can occasion considerable congestion may bring on hæmorrhage, which is then usually of the capillary variety. As illustrations may be mentioned hæmorrhage into the stomach induced by cirrhosis of the liver; critical and vicarious hæmorrhages; those resulting from over-straining or local irritation; and that which follows embolism. 3. **Morbid conditions of the walls of the heart or coats of the vessels.** Among the most important are degeneration or aneurism of the cardiac walls; atheromatous or calcareous degeneration of the arteries; arterial or miliary aneurisms; varicose veins; degeneration or mere functional debility of the minute vessels. Where the vessels are not well-supported, as in the brain, or if the tissues are functionally weak and toneless, hæmorrhage from this cause is much more likely to take place. The feeble new vessels in recent inflammatory exudations, and those in certain vascular cysts or villous growths, are very liable to give way. 4. **Abnormal states of the blood, and constitutional conditions.** These often predispose to hæmorrhage, as in *anæmia*, especially if this is due to previous loss of blood; in *scurvy* or *purpura*; in low fevers, particularly *typhus* and *small-pox*; and in the condition induced by bad diet, chronic diseases, or other lowering agencies. A plethoric state of the vascular system favours some forms of hæmorrhage, and hence this symptom may be predisposed to by over-feeding, sedentary habits, and other causes of *plethora*. There is a special condition termed *hæmophilia* or the *hæmorrhagic diathesis*, which will be separately considered.

Hæmorrhage may occur at any time of life, but it is most common about the period when growth and development are proceeding rapidly; and in advanced life, when the vessels and other tissues have undergone degeneration. It is also prone to affect different parts at different ages—thus in the young *epistaxis* is frequent; in young adults *hæmoptysis*; later on *hæmatemesis*, *melæna*, and *hæmaturia* are more common; and in old age cerebral hæmorrhage chiefly occurs. Some individuals are much more liable to hæmorrhage than others.

**ANATOMICAL CHARACTERS.**—When hæmorrhage has taken place from the capillaries of a mucous membrane, it is often impossible even on the most careful examination to detect the vessels from which the blood has escaped. It has been assumed that under these circumstances no actual rupture of their walls has taken place. Generally the source of the bleeding can be discovered, and remnants of the blood are visible. Ex-

travasations vary in amount from minute points of blood to large clots, which are either distinct and well-defined, or mixed up with the tissues of the structure involved; there may be one or more collections of blood in the same organ. The blood is generally found coagulated more or less firmly, and at first presents a dark red colour. It may more or less speedily cause irritation of the surrounding tissues, as evidenced by redness, inflammatory exudation, softening, or even the formation of an abscess. Its colouring matter may also stain neighbouring structures. If the extravasation does not soon prove fatal, it undergoes the following changes:—1. The colour gradually becomes paler, at the same time changing to brown or yellow, and ultimately it may become almost white; a granular pigment often forms simultaneously, along with crystals of hæmatoidine. 2. The clot contracts in size, becoming firmer, and being surrounded with a strong fibrous capsule; it also frequently itself undergoes organization into fibrous tissue, and becomes vascularized, in which condition it may remain permanently. 3. In some cases it is absorbed, leaving a cystic cavity containing fluid, which fluid may also ultimately be taken up, the walls of the cyst coming together, and only a hard cicatrix remaining, sometimes coloured by pigment. Occasionally nothing is left but altered blood pigment, of a yellow or black colour, especially on membranous surfaces; or there may be no trace of a previous hæmorrhage except a puckering and contraction. In some instances a clot softens and assumes a puriform appearance.

**SYMPTOMS.**—Active hæmorrhage is often preceded by premonitory symptoms. Thus there may be general excitement of the circulation, with a quick, sharp, and full pulse; or local sensations of weight or fulness and heat are complained of, with increased pulsation, the extremities being at the same time cold. Special symptoms frequently indicate the approach of bleeding in connection with particular organs.

The actual symptoms may be described as **general** and **local**.

**General.**—According to the quantity of blood lost, and the rapidity with which it escapes, hæmorrhage may either be unattended with any general symptoms at all; or it proves more or less speedily fatal; or gives rise to faintness or syncope; or merely originates an anæmic condition.

**Local.**—The local phenomena necessarily vary with the seat and amount of the hæmorrhage. Instant death may result from the mere local effects of the blood effused. The symptoms usually observed are dependent upon:—*a. Physical interference* with the functions of an organ, owing to the mere presence of the blood, as when it escapes into the pericardium, and presses upon the heart; or when a large quantity accumulates in the bronchial tubes. *b. Destruction of tissues*, which may be broken up or lacerated. This generally happens when extravasation takes place into the substance of an organ, the functions of which are thus materially disturbed. *c. Irritation* by the coagulated blood, this being liable to give rise to more or less local inflammation, with its accompanying symptoms. *d. Excitation of certain acts*, which have for their object the discharge of the blood, should it escape on a free mucous surface. Thus blood in the stomach often causes vomiting; in the air-passages it excites cough. The amount and characters of the rejected blood vary considerably, and valuable indications are usually thus afforded as to the source of a hæmorrhage. It is often more or less mixed with secretions and other materials. The presence of blood may be obvious to ordinary objective examination, as in the case of subcutaneous



hæmorrhages, or bleeding from visible mucous surfaces; or it may be indicated by special *physical signs*, as, for instance, when it accumulates in the bronchial tubes or a pleural cavity.

DIAGNOSIS.—There are three main classes of cases which are met with in medical practice, bearing upon the diagnosis of hæmorrhage. At present it will only be practicable to offer a few general remarks with reference to these several groups, more complete details on the subject being given when treating of individual hæmorrhages.

1. Blood may escape or be discharged externally, as through the mouth or anus, or with the urine. The points to be then determined are:—*a.* The actual occurrence of hæmorrhage, and the amount of blood lost. *b.* The source of the bleeding, both as regards the organ or part from which the blood comes; and the vessels from which it escapes. *c.* The immediate cause of the hæmorrhage, and the pathological condition or lesion upon which it depends. At the outset it is necessary to warn strongly against relying too implicitly on the mere statements of patients with regard to hæmorrhage, it being the duty of the practitioner to see the blood for himself, if possible, and to submit it to an adequate examination, thus ascertaining definitely and certainly, not only whether bleeding has really taken place, but also the quantity and characters of the blood discharged. It must be borne in mind that malingerers sometimes pretend to be the subjects of hæmorrhage, while other patients mislead unintentionally; that certain materials may resemble and be mistaken for blood; and that this is sometimes so altered in its characters, or mingled with other substances, especially when it comes from the alimentary canal, that it cannot be recognized as blood. The observation of the quantity and characters of discharged blood is often of material assistance in the diagnosis of the source and direct cause of hæmorrhage, which is further aided by a consideration of the history of the patient; the mode in which the blood is expelled; the accompanying symptoms, both general and local; the results of objective, and particularly of special modes of physical examination; and the further progress of the case.

2. The diagnosis of extravasation of blood into the substance of organs or tissues has next to be considered. When hæmorrhages are subcutaneous, they are generally easily recognized, but those which take place in connection with organs, as the brain or lungs, or into the substance of deep tissues, often present more or less difficulty in their diagnosis. Extravasation associated with an organ is usually indicated by obvious local symptoms, referable to such organ, which are frequently sudden in their onset. In this class of cases the diagnosis comprehends:—*a.* The fact of hæmorrhage having occurred, and its distinction from other lesions. *b.* The exact seat of the extravasation, so far as this can be determined. *c.* The amount of blood effused. *d.* The direct effects produced upon the implicated organ, and the further morbid changes subsequently set up by the extravasation. These questions can only be decided by a careful consideration of each individual case in all its details.

3. Hæmorrhages sometimes take place into cavities within the body, as into serous or mucous cavities, and these may be on a large scale. Not uncommonly such cases are very obscure, and it is impossible to make a positive diagnosis, but this might be arrived at from a previous knowledge of the presence of some morbid condition likely to be attended with such an event, for example, an aneurism; the



occurrence of general symptoms of shock and loss of blood; and the discovery of local physical signs indicative of the accumulation of blood.

**PROGNOSIS.**—In the large majority of instances hæmorrhage must be regarded as more or less serious, but its gravity differs very widely in different cases, and occasionally it is a favourable event within certain limits. The chief circumstances which influence the prognosis when blood escapes externally are:—1. Its amount. 2. Its source, and the immediate cause of the bleeding. 3. The power of checking the hæmorrhage, and its liability to recur. 4. The previous condition of the system, and the effects of the loss of blood upon it. Extravasations into organs are always grave, but their degree of danger depends on:—1. Their size and number. 2. The organ involved, and the precise seat of the lesion. 3. The cause of the hæmorrhage. 4. The immediate and remote effects upon the organ implicated. Hæmorrhage into internal cavities is generally highly dangerous, on account of its ordinary causes; the serious interference with the functions of important organs which it involves; and the loss of blood, which cannot be prevented by any direct means.

**TREATMENT.**—The general principles and indications which are applicable to the treatment of hæmorrhages in medical practice, and the means by which they are to be carried out, are as follows:—

1. The first indication is to **stop the bleeding**, should this be desirable; and to **prevent its recurrence**. It is not always advisable to check hæmorrhage, provided the amount of blood lost is not serious, for it may be the means of warding off some worse evil, as in many cases of bleeding from piles, or of epistaxis. Usually it needs to be stopped, and the following are the measures to be borne in mind for this purpose:—

*a.* Absolute *rest of the body* in the horizontal posture is frequently demanded; and, so far as this is practicable, the part from which the blood comes should be kept quiet, every disturbing action being avoided, such as cough in cases of hæmoptysis, or vomiting in those attended with hæmatemesis. Any exertion that is liable to excite the heart, or to cause undue pressure upon the interior of the vessels, should be guarded against and avoided.

*b.* Attention to *position* may prove most serviceable, one object being to assist the return of blood by the veins; and every impediment to the venous circulation must be removed or avoided. Sir Joseph Lister explains the influence of certain positions in stopping bleeding, by the theory that the arteries become contracted, through the influence of the nervous system, and he believes that the effect may travel some distance by extension along the vessels, or may influence remote vessels by reflex or sympathetic action.

*c.* The *circulation* must be maintained in as calm a state as possible, and should it be excited, while the heart is acting unduly, *vascular sedatives* are of the utmost value. Venesection is sometimes resorted to with the view of lowering the cardiac action, but this measure is only rarely indicated.

*d.* The administration of *astringents* in full doses is generally called for in medical practice, the most important being acetate of lead, gallic and tannic acids, sulphuric acid, alum, iron-alum, oil of turpentine, ergot of rye, tincture of steel, and tincture of hamamelis (℥ 2 to 5 or more). Some of these may be advantageously combined, and either

tincture of opium or digitalis frequently forms a most useful adjunct. Subcutaneous injection of ergotine or ergotinine may often be employed with great advantage. All food and drink should be taken cold, and stimulants must not be given unless absolutely required. In some forms of hæmorrhage the constant sucking of ice is exceedingly serviceable.

*e. Local remedies* are often indicated, such as pressure; astringent applications; cold, especially in the form of ice, which may be usefully applied to neighbouring parts, as to the chest or epigastrium in cases of hæmoptysis or hæmatemesis, or even to distant parts, as in the treatment of epistaxis, which may sometimes be checked by the application of cold to the back of the neck. In cases of hæmorrhage from certain mucous surfaces, it may be desirable to employ astringent injections, in order to bring the remedy into direct contact with the source of the bleeding. It may also be necessary to have recourse to surgical measures, such as the use of the actual cautery, or torsion or ligature of vessels.

*f.* It is highly important to *improve the general condition* of the patient, and the *state of the blood*, should these be at fault and give rise to a tendency to bleeding, by the exhibition of good diet, tonics, tincture of steel, and similar remedies. In this way hæmorrhage may often be prevented when it tends to recur. On the other hand, when it is associated with a full plethoric habit, the administration of *saline aperients* from time to time is very serviceable, while the diet must be restricted.

*g.* Sometimes it is advisable to *draw off blood* to parts distant from the seat of hæmorrhage, by means of heat or sinapisms to the extremities, Junod's boot, leeches, or dry or wet cupping; or to *prevent it from entering a part*, by the aid of pressure upon the main arteries.

2. The second indication is to attend to the **general effects** of the loss of blood. Syncope must be treated by position, stimulants, and other appropriate measures, as will be more fully described hereafter. In some cases transfusion of blood is demanded, in order to save life, and to replace the blood which has been lost. Anæmia calls for the administration of some preparation of iron, with proper dietetic and hygienic management.

3. The **local effects** of effused blood must also receive attention. In rare cases it may be desirable to have recourse to some surgical operation, in order to remove an accumulation. Usually all that can be done is to keep the part affected entirely at rest, and to employ measures which tend to promote the absorption or removal of extravasated blood. The administration of iodide of potassium, and the application of blisters are often decidedly useful measures for the purpose of aiding its absorption. If inflammation is set up, this must be treated by appropriate remedies.

#### HÆMOPHILIA.—HÆMORRHAGIC DIATHESIS.

**PATHOLOGY AND ÆTIOLOGY.**—Under these terms are included certain cases in which there is a liability to excessive hæmorrhages without any adequate obvious cause. They are supposed to be due to a peculiar constitutional condition or diathesis, which has received the special appellation given above. The condition is often hereditary, and may be observed in several members of the same family, the



individuals being known as "bleeders." Mr. Hutchinson thinks it may be associated with hereditary gout. The tendency to hæmorrhage is congenital. Hæmophilia is far more frequent in males than females, and the latter do not usually present the complaint in its typical form. At the same time it seems to be through the mothers rather than the fathers that it is transmitted to the sons. The subjects of hæmophilia do not present any obvious peculiarities. It has been stated that the blood presents certain changes to account for the hæmorrhage, namely, deficiency of fibrinogenous elements and of red corpuscles, but the best authorities affirm that the blood is not altered; nor have any definite changes been discovered in the blood-vessels, although it is presumed that these vessels are really in an abnormal state.

**SYMPTOMS.**—Hæmophilia very rarely manifests itself at birth, but usually in early infancy; sometimes at the time of the second dentition, or even later in life. The symptoms include a marked tendency to hæmorrhages, either both spontaneous and traumatic, or in the less marked cases merely spontaneous; and swelling of the joints. The bleeding is almost always of the capillary variety. When spontaneous, it may be preceded by local premonitory symptoms similar to those which are met with in other forms of hæmorrhage. It usually takes place from mucous surfaces, epistaxis being most frequent in children. The frequency of the hæmorrhages, their duration, and the amount of blood lost, vary in different cases and at different times. Interstitial extravasations, either diffused or circumscribed, and superficial ecchymoses may also occur. Traumatic hæmorrhages also vary in their site and extent, and may arise from contusions; wounds or cuts, however insignificant in themselves; or slight operations, such as vaccination, leeching, tooth-extraction, cutting the *frænum linguæ*, or opening an abscess.

When the joints are affected, those of large size are mainly involved, especially the knee-joint. Usually there has been some injury, but not necessarily. An affected articulation is swollen, more or less fluctuating, and painful. The enlargement seems to depend upon extravasation of blood into the interior of the joint.

The hæmorrhages may prove fatal, either rapidly or slowly; or they leave a condition of extreme anæmia, which lasts for a long time, or may never be recovered from. In connection with the joint-affection febrile symptoms may supervene, and this condition sometimes continues for a long period, while relapses are very liable to occur. Those who are the subjects of the hæmorrhagic diathesis rarely recover completely.

**TREATMENT.**—Persons who are affected with hæmophilia should, if practicable, live in a warm climate. They require good food, including plenty of meat. Ferruginous preparations and cod-liver oil seem to be the best medicinal remedies. Marriage ought to be prevented, and this particularly applies to women belonging to families of "bleeders." On no account should any operation be performed likely to cause hæmorrhage, especially tooth-extraction. It is very difficult to check bleeding by the ordinary measures, but early compression appears to answer best in traumatic forms. Tincture of steel may at the same time be given freely internally. The effects of the loss of blood must be treated on ordinary principles, and transfusion may be called for. In the treatment of the joints, rest and support are chiefly indicated.



## CHAPTER IV.

## INFLAMMATION.

INFLAMMATION is one of the most common and most important pathological processes with which we have to deal. It is impossible at the present time to give any definition of inflammation which shall be generally acceptable. Moreover, it will only be practicable in this work to present a summary of the chief facts relating to the subject which have been established, and of the more important views which are entertained concerning the inflammatory process and its results.

**ÆTIOLOGY.**—Under this head it is only intended to point out the obvious causes by which inflammation is originated. Questions relating to pathology will be subsequently discussed. These causes are numerous, and they vary much according as the morbid process affects different structures, but the following summary will serve to indicate their general nature.

**A. Exciting causes.**—Inflammation immediately results from some injury to the affected tissue, either direct or indirect, which is not sufficiently powerful actually to destroy, but merely to impair its vitality, and to disturb the conditions which are essential for healthy nutrition. Such injury may be inflicted in the following ways:—

1. *Mechanical injury or irritation* of some kind is a common cause of inflammation. Thus it may result from a wound, bruise, or strain, foreign bodies, calculi, extravasated or coagulated blood, floating particles in the air, retained excretions, worms and parasites, tumours, diseased bone or cartilage, gouty concretions, various morbid deposits, or other organic changes capable of setting up irritation.

2. *Chemical irritants* are also powerful agents in exciting inflammation. A specific kind of inflammation may be set up in this way, as, for example, when croton oil, tartar emetic, or a blister is applied to the surface of the skin; or when arsenic or cantharides is introduced into the system, the former always affecting the stomach, the latter the kidneys. The inflammation resulting from the contact of air with certain surfaces, or of pus or gangrenous fluids, is supposed by many to come under this category; and so does that set up in the alimentary canal by many kinds of improper food, and that due to the irritation produced by some animals and stinging plants.

3. The direct action of undue *heat* or *cold* upon animal tissues is a prominent cause of inflammation, its degree depending upon the intensity of the irritant and other circumstances.

4. Certain *specific organic poisons*, either by their local action, or as the result of their presence in the blood, are most important causes of inflammation, each giving rise to its own specific lesions, and affecting particular structures. These poisons either enter the body from without, as in the case of the several contagious diseases; or they are

generated within the system, of which the morbid agents originating acute rheumatism and gout afford illustrations.

The ætiological relation of living organisms to certain forms of inflammation is now very generally believed in, but this point will be again alluded to under PATHOLOGY.

5. Internal inflammations are often caused by conditions which give rise to a *general chill*, such as exposure to cold and wet, or to a draught when the body is heated and perspiring. These probably act chiefly by contracting the small cutaneous vessels, and driving the blood inwards, thus causing internal congestion; partly by interfering with cutaneous excretion, in consequence of which noxious materials accumulate in the blood. Some think that the effect is produced through the nerves.

6. *Deleterious products* may collect in the system from other causes, and induce inflammation. Thus the materials formed as the result of excessive destruction of tissues in high fever may lead to this result; and the inflammation which sometimes follows the sudden disappearance of a chronic skin disease, or the suppression of habitual discharges, is believed to be induced in this way.

7. *Undue activity* of an organ, whether physical or physiological, may give rise to inflammation of its structure.

8. Inflammation may originate in *irritation of a nerve*, and it may then appear at a distance from the seat of irritation.

9. When an organ or structure is inflamed, it may set up *secondary* inflammation in other parts, either by direct extension; it is said by metastasis; or by the convection of emboli or septic matters, through the agency of the blood-vessels or lymphatics.

**B. Predisposing causes.**—These influence not only the occurrence of inflammation, but also the part it affects, and the variety it assumes. They may be classed as *general* and *local*.

1. *General*.—The condition of the entire system, but especially that of the blood, may materially assist in predisposing to inflammation. The most important general predisposing causes are debility with impoverishment of the blood, in whatever way originated; plethora, especially from over-feeding, combined with excessive indulgence in stimulants and general luxurious habits; and, above all, the presence of some morbid material or poison in the blood, as in the case of the eruptive fevers, gout, rheumatism, syphilis, or diabetes; or when the products of tissue-change accumulate in the system, either from too rapid disintegration, as in fevers, or from deficient action on the part of the excretory organs, especially the kidneys and skin. Children and old people are on the whole most liable to inflammation, but this statement does not apply to all structures. Persons of sanguine temperament are also believed to be more predisposed than others.

2. *Local*.—The chief local predisposing causes of inflammation are mechanical or passive congestion; defective nutrition of the tissues, their power of resistance being diminished, as after a previous attack of inflammation, or when the vessels are in a state of degeneration; and impaired innervation. The last cause acts partly by lowering the vitality of the tissues; but chiefly by diminishing the power of sensation and motion, so that the patient is not aware of the presence of irritants, and is at the same time unable to remove them.

**ANATOMICAL CHARACTERS.**—In considering this part of the subject, it will be expedient to describe at the outset the minute changes which are characteristic of the inflammatory process.

**A. Histological changes.**—The minute changes which occur in inflammation have been studied by many careful observers, with the aid of the microscope. The process itself has been watched in the transparent vascular tissues of animals, in which inflammation has been excited by different kinds of irritation, such as the web of a frog's foot, its mesentery or tongue, the wing of a bat, or the mesentery or omentum of certain rodent carnivora. The effects of inflammation have also been studied in a similar manner in the cornea, the ear of the rabbit, and other structures. The results of these observations may be described under the following heads:—

1. *Changes in connection with the Blood-vessels, Circulation, and Blood.* The phenomena associated with the vascular system constitute a most important element of the inflammatory process, and they occasion some of the more obvious anatomical signs of the change.

*a. Blood-vessels.*—In almost all cases inflammation is attended with an immediate *dilatation* of the small arteries; rarely there is a primary contraction of short duration, according to some observers. The eminent pathologist Cohnheim, whose untimely death we have recently had to deplore, states that this primary dilatation may subside, the vessels returning to their normal size, or even in some instances becoming contracted. If the irritation is sufficient, however, to cause inflammation, permanent dilatation ensues, which increases gradually for some ten or twelve hours, and then remains stationary, the vessels also becoming elongated and tortuous. In other cases the dilatation is continuous from the outset. The veins enlarge after a while, and they assume a varicose or aneurismal aspect, presenting little irregular bulgings and contractions. The capillaries also dilate, while their walls undergo structural changes in course of time, fat-granules accumulating in their substance, especially around the nuclei, and they send out processes by budding, which finally join together. In an inflamed vascular structure vessels which were previously visible become obviously enlarged; while new vessels come into view where none were seen before.

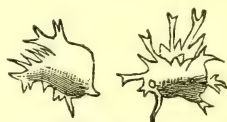
*b. Circulation.*—Inflammation has been usually described as being attended at first with *increased rapidity* or *acceleration* of the flow of blood, constituting what has been long termed *determination of blood*; but the latest experiments seem to prove that this phenomenon is not essential, and some pathologists consider that it forms no actual part of the process. The acceleration is soon followed, when it occurs, by a rather sudden return to the normal rate of movement, and afterwards the circulation lags, and gradual *retardation* takes place, this change commencing in the veins. In some experiments the blood-stream has appeared under the microscope to flag almost suddenly. A to-and-fro *oscillation* is then often seen; and finally complete capillary *stasis* or stagnation occurs, the vessels appearing to be crowded with red corpuscles. The stasis may be at first transitory, this being but a “local exaggeration of the general retardation of the progressive motion of the blood,” but it afterwards becomes permanent. Finally *thrombosis* or coagulation of the blood may occur, but not until the capillary walls are dead. In an inflamed area all the different conditions affecting the movement of the blood may frequently be observed in neighbouring vessels. The quantity of blood which actually flows through an inflamed part is greatly increased in the early part of the process; and Cohnheim has proved experimentally that about four times the normal amount of blood may flow through an inflamed limb.



c. *Blood*.—Important phenomena are observed in connection with the *blood-corpuscles*:—The *white corpuscles* accumulate in the vessels, especially in the veins, falling out from the mid-stream, loitering along and finally adhering to the wall, thus forming a continuous motionless layer, the diminished central current still persisting, though at a much diminished rate, until stasis is completed. Some observers believe that there is an actual production of these corpuscles in the inflamed part, and that their number is in this way increased. After a time they exhibit the phenomena of what has been termed *migration*, which was described imperfectly by Dr. W. Addison in 1842, and more completely by Dr. Augustus Waller in 1846, but which has in recent years come into special prominence in the pathology of inflammation, through the investigations of Cohnheim and others. The corpuscles penetrate the walls of the vessels, through which they may be observed in various stages of their transit, forming button-shaped elevations, then hemispherical prominences, then pear-shaped bodies attached by a pedicle, and finally separating altogether. No actual openings are left in the vessels to indicate the points at which they have escaped. The liberated white corpuscles are named *leucocytes*, and after they leave the vessels they send out processes, assume peculiar shapes, and migrate far and wide into the surrounding tissues, at the same time often undergoing a process of division, and thus becoming increased in number. The *red corpuscles* exhibit the same tendency to aggregation and stasis, so that they frequently look like rods of red coral, or they may adhere to each other so closely that their outlines are quite obscured. Often a sudden extravasation of a number of coloured blood-discs takes place from a capillary, and this has been termed *diapedesis*. They also migrate through the walls of the vessels, chiefly the capillaries, but not nearly to the same extent as the white blood-cells as a rule; in the most severe inflammations, however, where blood stagnates in a large number of capillaries, the reverse may be the case. In an inflamed area no migration occurs in the centre, where there is stasis; around this both white and red corpuscles escape; but in the outer circle only white corpuscles migrate. The corpuscles at first remain near the vessels from which they have escaped; afterwards they are pushed away by others, or washed by exuding fluid, or the white corpuscles migrate by their own movements. Dr. Lionel Beale affirms that in inflammation minute particles of *bioplasm* or germinal matter of the blood pass through small rents or fissures in the capillary walls, and afterwards grow and multiply by division. Some of these particles, he says, are detached from white corpuscles. He considers that most of the particles seen outside the vessels originate in this way, and not from the direct transit of white corpuscles.

Another phenomenon which almost invariably occurs in inflammation is the *exudation* of the liquid portion of the blood out of the vessels into the surrounding tissues. Though usually called *liquor sanguinis*, the exuded liquid is rarely identical with this fluid in its composition. It may be mere serum, but as a rule contains fibrinogenous materials, as well as albumen, and also a considerable proportion of phosphates, chlorides, and carbonates. Its nature and quantity will vary much according to the seat and intensity of the inflammation, as will be more fully pointed out further on.

FIG. 1.

Amoeboid Leucocytes. (v.  
Recklinghausen.)

The alterations thus far described cannot of course be observed in structures which have no vessels, such as the cornea and cartilage; but they may then be noticed in the vessels of neighbouring tissues, from which the nutriment which supplies the non-vascular structures is derived.

2. *Changes in the affected tissues.*—Until a comparatively recent period, a primary disturbance of the process of nutrition in the tissues themselves was regarded as the most important element in inflammation. This was the special doctrine of the “cellular pathology,” which was at one time generally accepted. Although it is still held by some pathologists, the latest experiments and observations of Conheim, Senftleben, Dowdeswell, and others have led many to discard it altogether; while others regard the nutritive changes as secondary and subordinate to the vascular phenomena already described. There can be no doubt that nutrition does become affected in inflammation, and this is observed in different degrees in different tissues; but probably the changes are the result, and not a part of the inflammatory process. Some inflammations have been named *parenchymatous*, such as that involving cartilages, this term being intended to imply that nutritive changes in the tissues themselves constitute the prominent feature of the process.

In the earlier period of inflammation the change which the affected tissue is believed to undergo, by those who hold the doctrine of “cellular pathology,” is an increase in the nutritive activity of certain cellular elements. This increased activity is said to be observed chiefly in connection with those cells which are active in health, and which contribute by their growth and proliferation to the maintenance of the structure of which they form a part. Hence it is presented most strikingly by the epithelial elements of the skin, mucous membranes, and glandular organs; and to a less degree by the endothelium lining serous membranes and allied structures. It is very questionable whether the fixed cells of connective tissue or of the cornea, or cartilage-cells undergo any active changes in inflammation, but at any rate they are far less in degree; while in the higher tissues, such as nerve-cells, they do not occur at all. The inflammatory process affects young cells much more than those of older growth; and the changes are more marked in proportion to its intensity. These changes are thus described. The cells exhibit active amœboid movements, and undergo many alterations in form, throwing out processes in various directions. They generally enlarge, their protoplasm increasing in amount, at the same time becoming cloudy and granular, so that it conceals any enclosed nuclei. This is exemplified by the so-called “cloudy swelling” of the renal epithelium in acute inflammation of the kidney. *Cell-proliferation or germination*, as it is termed, then takes place, leading to the active production of new cells by endogenous development. This results from division of the nucleus and protoplasm, or by a process of vacuolation.

The later effects of inflammation upon tissues is in many cases to impair their nutrition more or less, or even to destroy them, as will be more fully pointed out presently. New cells are prone to decay, especially if they are very quickly produced, and if the inflammation has been severe; but they may develop into a permanent tissue, which tends to be of lower organization than the original one. Where intercellular substance exists, as in cellular tissue or cartilage, this often softens and breaks down, and the entire structure may become at last



completely destroyed, the histological elements being involved in the destruction.

Dr. Beale describes the bioplasm of inflamed tissues as increasing greatly in amount.

**B. Pathological Terminations and Products.**—Having considered the minute changes regarded as characteristic of inflammation, it will now be convenient to point out the more obvious effects which it produces, and its modes of termination.

1. The changes already described having taken place to a greater or less extent, what is termed *resolution* may follow, that is, a subsidence of the vascular disturbances, and the absorption of any exudation, the tissue affected being restored to its normal condition. Any leucocytes which are present in it either undergo fatty degeneration before absorption, or possibly may re-enter the blood-vessels or lymphatics. Resolution may take place very quickly, this being termed *delitescence*; or it is supposed that *metastasis* may happen, which implies the disappearance of inflammation from one part, with its simultaneous appearance in some other structure.

2. *Exudation and Effusion.*—As already stated, inflammation is usually attended with an escape of fluid from the vessels, varying much in quantity and composition. The material exuded may be either *serum*; *fibrinous exudation* or *lymph*; *blood*; or *mucin*.

*a. Serum.*—The best examples of this effusion are seen in connection with inflammation of serous membranes, and in the submucous tissue of certain parts, such as the larynx. The fluid is not uniform in its composition and characters, but contains a variable amount of albumen, and frequently also a small proportion of fibrinogenous elements, with a considerable quantity of phosphates and chlorides. It may remain for a long time unaltered; or is absorbed if the inflammation subsides; or becomes more or less purulent. The less severe the inflammatory process is, the more likely is the effusion to be merely serous.

*b. Fibrinous Exudation—Lymph—Coagulable Lymph—Inflammatory Exudation.*—These terms are applied to an exudation which is produced in certain forms of inflammation, the material exuded containing more or less fibrinogenous elements, and being spontaneously coagulable. It is especially observed in inflammation of serous membranes; and is more likely to occur when the inflammation is severe. There is in reality, however, no distinct line of demarcation between this exudation and inflammatory serous effusion. It contains a number of cells, which are probably all leucocytes. Dr. Beale describes particles of bioplasm as being present in it.

Two kinds of lymph have long been recognized, namely, the *plastic* or *fibrinous*, which contains abundant fibrin-forming ingredients, tends to coagulate, and promotes tissue-development; and the *aplastic*, *corpuscular*, or *croupous*, in which there are a large number of cells, exhibiting but little tendency to organization, but being on the other hand prone to degeneration, and to the formation of pus or other low products. The state of the patient, the seat and intensity of the inflammation, and other conditions materially influence the nature of the exudation.

After inflammation has subsided the lymph frequently undergoes organization into a new tissue; some pathologists affirm that only the leucocytes and the cells produced by proliferation, or the particles of bioplasm (Beale), become developed, the liquid portion merely nourishing these; others maintain that the fibrin coagulates and fibrilates, and



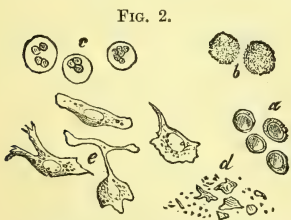
itself contributes to the formation of tissue. Probably most of the new material is usually derived from cell-development. Some kind of connective or fibrous tissue is generally produced, but bone, elastic tissue, epithelium, or fat may be ultimately formed. Certain of the higher tissues, such as muscle and nerve, are never developed under these circumstances. This organization is well seen in the changes which occur in the granulation-tissue by which wounds cicatrize, and in the adhesions and thickenings formed in connection with inflamed serous membranes. The consequences of these changes are often very serious, various structures becoming thickened, hardened, contracted, or bound together, and transparent tissues being rendered opaque.

After organization a process of degeneration may set in, evidenced by wasting or withering, the substance becoming dry, yellow, horny, and stiff; by fatty or liquefactive change, which may lead to its absorption; or by the formation of black pigment. Similar changes may occur in the products of corpuscular lymph.

*c. Blood* is sometimes present in variable quantities in inflammatory exudations. It is partly the result of migration of the red corpuscles, but some may have escaped owing to the actual rupture of vessels, especially of those recently formed.

*d. Mucin.*—In inflammation of mucous membranes this substance is sometimes met with, and gives a tenacious, stringy character to the fluid discharged from the surface.

3. *Suppuration or Formation of Pus.*—The tendency to suppuration varies according to the tissue affected, and the constitutional condition of the patient, but it is generally more liable to take place if the inflammation is very severe and concentrated. Pus may form on a free surface and be discharged, being then often mixed with other materials; it may accumulate in serous or other cavities: or it may involve the substance of tissues and organs, either as a circumscribed abscess, or as diffuse purulent infiltration. As regards its physical characters healthy pus is a thick, viscid, pale yellow liquid, odourless, alkaline in reaction, with a specific gravity of about 1030. It consists of a fluid—*liquor puris*—in which float pus-corpuscles and other microscopic particles. *Liquor puris* is an albuminous fluid, but also contains salts, pyin, chondrin, and fat. The corpuscles, as usually seen under the microscope, closely resemble white blood-corpuscles in size and appearance, being more or less round or sometimes irregular, and granular,



Pus-corpuscles. *a.* From a healthily-granulating wound; *b.* From an abscess in the areolar tissue; *c.* The same treated with dilute acetic acid; *d.* From a sinus in bone (necrosis); *e.* Migratory pus-corpuscles. (Rindfleisch.)

and having one or more nuclei, which are rendered more evident by acetic acid, and often break up when acted upon by this reagent. They have the power of spontaneous movement and migration, and can alter in form, as well as increase in number by fission. Dr. Beale describes pus-corpuscles in the *living* state as being masses of bioplasm without any cell-wall, which assume a variety of forms, but are never spherical, send out protrusions in all directions, these becoming detached and forming new corpuscles, and which are capable of spontaneous movement. He

further states that when *dead* they assume the spherical shape, their movements cease, a sort of cell-wall forms, they become more granular,

and bacteria are developed in them. It is in this condition they are usually seen under the microscope. It is now generally considered that the great majority of the pus-cells, especially in the earlier stages of inflammation, are merely leucocytes; and some maintain that they are solely derived from this source. Beale believes they are derived from the particles of bioplasm which escape. It is supposed that others are subsequently formed by proliferation of the cells and germinal matter of the affected tissue, and that they increase in number by cleavage and endogenous formation. Leucocytes possess the power of destroying the tissues with which they come into contact, and it is partly in this manner that an abscess makes its way to the surface.

Various kinds of pus are described, such as *healthy* or *laudable*; *ichorous* or *watery*; *serous*; *sanious* or *bloody*. It may decompose and form highly noxious gases, and sometimes undergoes physical and vital changes if not discharged, its fluid portion being absorbed, while its cells become withered and undergo fatty degeneration, so that it is converted into a cheesy mass, containing fat-granules, cholesterin, shrivelled cells, and nuclei. Ultimately the remains may become the seat of calcareous deposit.

4. *Softening* of tissues is not an uncommon result of inflammation, and it may terminate in the complete breaking-down and destruction of the structures involved. This may be illustrated by the softening which usually accompanies inflammation of the brain and cord.

5. *Induration* is another consequence of inflammation, especially when it is chronic, being due to the substitution of an imperfect fibrous tissue for the normal structures.

6. *Interstitial absorption* is sometimes observed, as in the case of inflammation of bone.

7. *Ulceration*.—When inflammation destroys the tissues on a surface, an *ulcer* is formed. If this is quite superficial, only epithelium being removed, it is termed an *excoriation* or *abrasion*. Ulcers of different kinds frequently come under the notice of the physician in connection with mucous surfaces. Usually there is a discharge of pus after the inflammatory process subsides. An ulcer cicatrizes by the development of granulation-tissue into fibrous tissue, and the cicatrix afterwards tends to contract, and may thus lead to serious consequences. Ultimately the original structures may be developed anew, but this is not accomplished for a long time, and some tissues are never reproduced.

8. *Gangrene* or *Mortification*.—If inflammation is very intense, rapid death of the involved tissue in mass may result under certain circumstances, and a slough is formed, which becomes isolated from the living textures, and undergoes a process of separation, leaving an ulcerated surface. Almost any tissue may thus mortify, but gangrene is particularly observed in the subcutaneous areolar tissue, and in the mucous membrane of the alimentary canal, being but rarely seen in the substance of organs. It is in these cases of the moist kind, and therefore the slough is liable to undergo rapid decomposition. Bone may become necrosed as the result of inflammation.

GENERAL MORBID ANATOMY.—The exact appearances and other objective characters which accompany inflammation must of necessity vary widely, according to the part involved; the intensity, rate of progress, and type of the inflammatory process; the nature and amount of its products, which differ materially in different tissues; and numerous other circumstances. Hence it is impracticable to give a definite



and precise general description of its more obvious anatomical characters. It may, however, be useful to present a brief summary of, and to contrast the more evident effects which inflammation produces in connection with some of the principal classes of structures.

**The Skin.**—The morbid changes which accompany cutaneous inflammation are so obvious and well-known, that they have come to be regarded and familiarly described as the typical and characteristic signs of the inflammatory process. One of the most constant anatomical characters is redness, varying in its extent, degree, and hue, the latter usually tending towards brightness. If the inflammation is limited, the colour is more intense at the centre of the affected area, and fades off towards the circumference, while it diminishes or disappears under pressure. The small veins may be visibly enlarged. There is increased local heat, either evident to the touch or detected by the thermometer. Swelling or thickening of the cutaneous structures is often evident, and they feel soft or firm according to circumstances. The epithelium undergoes proliferation, while the superficial layers frequently soon lose their vitality and are shed freely. Serum may be effused upon the surface of the skin, giving rise to superficial moisture; under the epidermis, originating vesicles, or larger bullæ, or blisters; or into the tissues beneath, causing subcutaneous œdema. A fibrinous exudation containing leucocytes may also collect in the substance of, or beneath the skin, the tissues hence feeling thickened, firm, and brawny. The glands are liable to become enlarged, their cells increasing rapidly. Papules, wheals, or pustules form in certain varieties of inflammation; or it may lead to superficial cracks or abrasions, ulceration, diffuse suppuration, or gangrene.

The skin is the seat of peculiar kinds of inflammation in connection with some of the acute specific fevers. In chronic cases the cutaneous structures undergo serious and permanent organic changes.

**Serous and allied membranes.**—In serous inflammations there is at first marked redness, with loss of polish, more or less opacity, and thickening of the membrane. Then a fibrinous exudation collects on the surface, varying much in its amount, characters, and arrangement, which contains abundant cells, consisting chiefly, and, according to some pathologists, entirely of leucocytes, but according to others partly derived from proliferation of epithelium cells. At the same time an effusion of serum takes place into the cavity, which is more or less turbid, and may contain coagula, as well as numerous cells similar to those in the fibrinous layer. The further tendency is usually towards the absorption of this fluid, and the formation of thickenings, adhesions, or agglutinations in connection with the membrane. These are generally supposed to result from the organization of the layer of lymph with its enclosed cells, fibrous tissue being thus developed. It has been stated, however, that, at least in many cases, this fibrinous layer does not become organized, but undergoes fatty degeneration and subsequent absorption; and that the adhesions result from the development of small vascular papillæ or granulations which form on the surface of the membrane, under the epithelium. If the inflammation is intense or prolonged, the fluid may become purulent, and the same event is apt to occur in certain constitutional conditions.

Though serous inflammations vary much in their extent and products, it may be stated that their general tendency is to originate materials which are prone to become organized.



**Mucous Membranes.**—In connection with mucous membranes three varieties of inflammation are described, namely, *catarrhal*; *croupous*, *membranous*, *plastic*, or *fibrinous*; and *diphtheritic*.

*a. Catarrhal.*—This is the ordinary form of mucous inflammation. It commences with hyperæmia and swelling of the membrane, which is at first abnormally dry. Soon, however, there is an increased secretion of a watery or viscid mucus, the latter containing abundant cells, and if the inflammation continues the discharge assumes a more or less purulent appearance, owing to the large number of cells present, many of which have the precise characters of pus-cells. The mucous glands and follicles enlarge, and become also filled with cells. In some cases the submucous tissue becomes infiltrated, and if it is of lax texture a considerable amount of serum may collect in its meshes. Abrasions or ulcers of the mucous surface are not unfrequently originated, and gangrene may ensue. If the inflammation becomes chronic, considerable changes are produced in the structure of the membrane and its glands.

*b. Croupous.*—This variety differs from the former in that a layer of so-called *false membrane* is deposited on the surface, varying in thickness and consistence. It consists of coagulated fibrin, either amorphous or fibrillated, enclosing epithelium and other cells; or it may be entirely made up of altered epithelium-cells, without any fibrin. Even when distinctly fibrillated it shows no tendency towards permanent organization.

*c. Diphtheritic.*—Some pathologists regard diphtheritic as differing from croupous inflammation, in that a fibrinous exudation forms not only upon but in the substance of and beneath the mucous membrane, which as a consequence is destroyed and converted into a slough, and an ulcerated surface is left on its separation. The reality of this distinction is very questionable.

It will thus be seen that inflammation of mucous membranes differs from that of serous membranes in that the products have no tendency to become organized.

**Organs.**—In addition to other causes which influence the inflammatory process, the pathological results of inflammation of an organ depend very materially upon the structures entering into its formation, and they further differ even in the same organ according to the particular tissue involved. In acute inflammation there is almost always a change in colour, which usually, though not invariably, tends towards redness at first, but subsequently may pass through various hues. The vessels are overloaded, and an unusual amount of blood escapes on section. Some organs become œdematous in the early stage of inflammation, so that a quantity of serous fluid can be expressed from their tissues. The principal more immediate effects are proliferation and detachment of epithelium-cells, where these exist, the products either accumulating or being carried away; changes in special structures, such as those of the liver or nerve-centres, usually of a destructive character; escape of fibrinous exudation containing leucocytes; and either increase, or softening and breaking down of interstitial tissue, where this is present. On account of these effects many very obvious alterations in physical characters are brought about. Inflamed organs are often enlarged and heavier than normal, or the specific gravity of the affected portion is increased. Occasionally inflammation leads to wasting and diminution in bulk, or this may remain unaltered. The consistence may be increased or diminished, some structures becoming apparently

firmer and more solid, others undergoing rapid softening; it is often found, however, that even when organs seem to be unusually firm, their consistence is really lessened, their tissue readily tearing or breaking down under pressure. A section frequently reveals marked alterations in the general appearance and structure; and inflammatory products may be visible to the naked eye, or microscopic examination reveals their presence. The subsequent progress varies considerably. Resolution or absorption often takes place, the structures being restored to their normal condition; or the inflammation may terminate in speedy destruction of the tissues, the formation of an abscess, diffuse suppuration, or gangrene. Ultimately an inflamed part may remain permanently more or less altered in its characters and structure; or remnants of the pathological products may be evident, in the form of fibroid tissue, caseous or calcareous deposits, &c. Chronic inflammation of organs usually tends to cause contraction and hardening, with increase of cellular or fibrous tissue.

**PATHOLOGY.**—There are many points relating to the pathology of inflammation which are still matters of dispute, and about which conflicting views are entertained. It will only be practicable in the ensuing remarks to attempt a brief summary of the most authoritative opinions upon those questions which are of chief importance, and to give such explanations of the phenomena observed in connection with the process, as seem to be most in accordance with the present state of knowledge, and with the results of the latest experimental investigations of Cohnheim and others.

**1. Origin and Nature of Inflammation.**—*a.* The most simple view regarding the origin or immediate causation, and the essential nature of inflammation is, that the process is the *direct result of injury to a tissue*, in whatever way such injury may be produced, provided it is not of such intensity as to destroy its structure or vitality. This is the theory maintained by Prof. Burdon Sanderson, whose latest definition affirms that “Inflammation is the physiological effect of the damage done to a tissue when it is injured.” According to this view, inflammation is not a new process which is set up; nor is it the result or indication of disorder of function, but of arrest; “not the diversion of the resources of nutrition into new channels of activity, but merely the accumulation of such resources where they are useless.” In short, inflammation is regarded as the consequence of impairment or depression of the vitality of a tissue. Dr. Sanderson quotes in his *Lumleian Lectures*, 1882, striking experiments in favour of this theory. He further is of opinion that the *vascular system* is alone concerned in the process, and that all its phenomena are due to changes affecting the blood-vessels and the circulation. Lister long ago maintained that the essential lesion of inflammation was a *change in the vessel-wall* resulting from an injury, and that it was a step towards death.

*b.* According to a second theory, which is strongly supported by Prof. Lister, the influence of the *nervous system* is held to be a necessary factor in the production of inflammation. There is by no means an agreement of opinion, however, as to the modes in which this system is supposed to act. The chief views are, that its influence is exerted in different cases, either through a direct disturbance leading to abnormal action of the nerves of the part affected; through certain cerebro-spinal centres; by nervous sympathy of related parts, or diminished action of the nerves of one part leading to excessive action



of those of another part; or by vaso-motor reflex action. With regard to the effects which the nervous system produces, it is generally supposed to act upon the vessels, affecting their calibre, and thus influencing the circulation of the blood; but some hold that there are special trophic nerves, and that these are directly concerned in the inflammatory process, through their influence upon the nutrition of the tissues.

c. Only a few years have elapsed since the view almost universally accepted as regards the nature of inflammation made it a part of the general doctrine known as the "cellular pathology" of Virchow and his followers, and this is still upheld by many, at least in a partial degree. According to this theory, inflammation is an *active process*, and consists in an *alteration or perversion of the nutrition of the tissues themselves*. It was advanced by the late Prof. Goodsir, and by Sir William Bowman, who described inflammation resulting from an injury of the cornea as "a change wrought in the natural acts of nutrition then existing in the wounded part;" and was founded on the "proliferation of cells" supposed to be observed taking place abundantly in inflamed structures, and on the textural changes which inflammation leaves behind it. The latest experimental investigations in connection with the cornea, and those performed by Cohnheim on the ear of the rabbit, appear to have entirely disproved this theory, and show that the cells which were believed to originate in proliferation have really migrated from the blood-vessels. Beale holds a distinct theory coming under this category, in that he believes the inflammatory process to be attended with a rapid growth of bioplasm.

d. The latest theory as to the origin of inflammation is a development of the "germ-theory" of the causation of disease, which is at present in such high favour. It will be convenient to discuss briefly in this connection the different opinions held as to the relation of germs or organisms to the process of inflammation. Hueter has advanced the view that this process is associated with the *action of germs or organisms* universally present in the air or in water, which he terms "viable inflammation-exciters." He maintains that acute inflammation invariably depends on the morbid influence of septic organisms—air-germ, water-germ, or germ which sticks to the missile or weapon which inflicts an injury. He assumes that these organisms exist everywhere, with the exception of mountainous regions, near and above the line of perpetual snow, ready, whenever access is offered to them, to enter the body and fulfil their morbid functions; and when a tissue is injured in any way, its vitality being impaired and depressed, they are enabled to penetrate it. Dr. Ogston found the pus in acute abscesses loaded with micrococci, and also the pyogenic membrane infiltrated with similar organisms; he therefore attributes the suppuration to the action of these germs, and infers that acute inflammation in general depends on a similar cause. In short, the outcome of the theory now under consideration is that acute inflammation cannot possibly arise without the presence of these septic organisms. Mr. Watson Cheyne maintains that there are a number of varieties of micrococci, some hurtful and some harmless, and that many abscesses are directly caused by hurtful forms; or that under certain circumstances, as in a case which he instances, where the patient was in a very weak state of health, even harmless forms may cause an abscess.

Dr. Burdon Sanderson strenuously combats Hueter's and Ogston's view as to the origin of inflammation, and quotes numerous experi-



ments which are opposed to it, although he thinks that Ogston has pretty conclusively shown that micrococci invariably take part in the formation of acute abscesses. This eminent authority maintains that organisms have nothing to do with setting up inflammation primarily, although they may do so secondarily; that the only inflammations to which they stand in relation are those which, from their proved dependence on previously existing inflammation, may be properly termed secondary or infective; and that organisms are not so much mischief-makers as mischief-spreaders. Although an inflammation may come into existence without their aid, their presence communicates to it, after it has come into existence, the power of reproducing itself in previously healthy tissues, whether by extension or dissemination. "Inflammation is essentially a terminable process, having no tendency to spread or last beyond the limits of the proximate cause." "An uncomplicated inflammation is neither reproductive nor infective, neither benignant nor malignant. If it have any tendency, it is the tendency to leave off as soon as the occasion for it ceases." Such are the views entertained by Dr. Sanderson on this subject, and he has employed the term "mycotic inflammation" to define that form, the spread of which from tissue to tissue is accompanied by the growth and multiplication of organisms. It will be convenient, and will make the matter more clear, to state here the propositions which he laid down and illustrated in his *Lumleian Lectures*, and the conclusions at which he has arrived, as to the relation of germs to the inflammatory process:—1. The exudation of a normal inflammation is not infective. 2. No organisms endowed with inflammation-producing phlogogenic particles exist in the atmosphere, or in the ordinary aqueous liquids with which our bodies come into contact. 3. Whenever an inflammation becomes infective, it owes that property to chemical changes in the exudation-liquid, of which the presence of microzymes is a necessary condition. Conversely, septic organisms which are infective owe their infectiveness to the exudative soil in which they have grown. 4. The introduction into the lymphatics, and eventually into the blood-stream, of microzymes which have been grown in septic exudation is one of the mechanisms by which the seeds from which secondary or infective inflammations spring are disseminated.

The experiments of Lassar prove that the mere admixture of a large proportion of inflammatory exudation with the blood is not in itself a matter of serious moment; and that even pus-corpuscles have no toxic properties, so long as they retain their integrity, and are not unfit again to enter the circulation. Moreover, septic organisms may be brought into contact with living tissues without producing any disturbance; and experiments show that when septic products enter the circulation by the lymphatics, they possess the power, not of producing inflammation in undamaged tissues, but of transforming the normal effect of injuries into infective processes.

## 2. Explanation of phenomena and products of inflammation.—

It is not difficult to explain the more obvious signs of inflammation, and the evident changes which it produces. Redness is due chiefly to overloading of the blood-vessels, and to blood-stasis; partly to migration of red corpuscles, or to small extravasations. The alterations in the blood-pigment, and the accumulation of inflammatory products will explain subsequent changes in colour. Swelling, increase in bulk, and alterations in consistence and specific gravity, depend upon the

augmented quantity of blood in the affected part, the presence of inflammatory exudation or effusion, and, according to those who believe in the process, upon cell-proliferation. Acute softening and wasting of an organ is usually due to rapid degeneration and destruction of its tissues. With regard to the increased heat of external parts which are the seat of inflammation, the experiments of Cohnheim seem to have proved conclusively that it depends on the activity of the circulation. Until recently it was believed that it was due to the chemical processes associated with excessive tissue-change in the affected structures, while Beale attributed the phenomenon to the rapid growth of bioplasm. The most recent experiments, however, indicate that a focus of inflammation is not a focus of "calorification," for they have shown that in all sorts of inflammation, and at every stage of the process, the temperature of the inflamed part never exceeds that of the rectum; and they have corroborated John Hunter's statement, "that a local inflammation cannot raise the temperature of an inflamed part above the source of the circulation."

Coming now to the consideration of the minute changes observed in inflammation, the dilatation of the blood-vessels is accounted for by Sanderson simply on the ground that they have lost their power of resistance, or their "vital power," and Cohnheim's experiments support this view. Others believe that it is due to a paralysis of the muscular coat, through nervous influence, and the notion has even been advanced that there are vaso-dilator fibres which cause active dilatation of the vessels. It appears from the most reliable observations that the increased rapidity of the flow of blood along the dilated vessels, or so-called "determination of blood," which is noticed at the outset of the inflammatory process in some instances, is not essential, and is often absent, and it is regarded by Sanderson and others as forming no actual part of this process. The experiments of Glax and Klemensiewicz show that injury immediately diminishes the quantity of blood flowing through the blood-vessels of the injured part in a given time, under absolutely constant pressure. Experiments made by Dr. Thomas upon the mesentery are also in favour of the opinion that the initial determination of blood is not a true inflammatory phenomenon. The retardation of the circulation, and the diminished velocity of the blood-current which thus ensues, is at first due to the widening of the blood-stream, and then to the *changes in the walls of the vessels* themselves, which render it more difficult for the blood to pass through them than in the normal condition, the natural friction being increased. There is no detectable structural change in the vessel-wall. Cohnheim calls it "molecular," and regards it as possibly chemical in its nature. It was believed that changes in the blood itself accounted for the retardation of its flow, and especially the aggregation of red corpuscles, but Lister long since concluded from his experiments that this accumulation of corpuscles in inflamed tissues is due to their natural tendency to cohere under abnormal conditions, just as they do when blood is removed from the body. Stasis of the blood is also accounted for by the changes in the vascular walls, and not by any alteration in the blood itself. This stasis Sanderson does not regard as a necessary part of the inflammatory state, but rather as a sequela or consequence, occurring only here and there in the inflamed area. He states that when the action of the cause can be limited, stasis, if it occur, is limited to the parts most injured; and he looks upon it as an expression of a



higher degree of injury than that required to produce the inflammatory state.

The retardation of the blood-stream is the proximate cause of the loitering of the corpuscles, and this retardation, especially in the veins, appears to be essential before migration can take place. They penetrate the vascular walls by virtue of "the power which leucocytes, and other amœboid bodies resembling them, possess of intruding their own substance into that of dead tissues, and of any material capable of imbibition with which they are brought into contact in an active state." They do not make their way through the vessels by any increased activity on their own part.

The increased exudation of coagulable fluid is also due to the impaired vitality of the vessels, there being consequently excessive leakage, which has been proved by the experiments of Glax and Klemensiewicz to be an immediate result of injury. The accumulation of this fluid is subsequently partly the effect of deficient absorption or drainage, due to increased percentage of solids, and especially of the number of corpuscles in the exudation, and partial obstruction of the lymph-ducts; while less escapes from the vessels. Its coagulation is said to depend on the fact that some leucocytes which have escaped undergo disintegration, become dissolved in the liquid in which they are suspended, and impart to it the property of producing fibrin.

The origin of pus-corpuscles is a point about which there is much difference of opinion. Sanderson believes that they are entirely leucocytes; others maintain that they are partly products of cell-proliferation, and that they can themselves multiply. Beale is of opinion that they are derived from particles of bioplasm.

**3. Terminations.**—By many pathologists inflammation is regarded as taking an active part in the processes by which the lesions it sets up are repaired, but Sanderson is strongly opposed to this notion. His views are thus expressed:—"Resolution of an inflammation means either that the temporarily arrested processes of normal life simply go on again; or, if the process has caused destruction, this has to be repaired, not by a continuation of the active process, but simply by the restitution of the normal condition." "Stasis is the mechanism by which inflammation kills, and thereby gives occasion to further pathological changes, of which necrosis is the starting-point." "The only direct after-effect of inflammation is necrosis, that being dependent on the arrest of the circulation; and the processes of restitution of structures which have thus ceased to live, cannot with any regard to accuracy, be regarded as parts of inflammation, or as in any way different from those by which the same structures are restored when destroyed by other agencies. The determining cause in the one case, as in the other, is the power of renewal inherent in the adjacent undestroyed tissue." Observations on the reparation of the cornea after injury, showing that this takes place by an exogenous growth from the margin; and also the phenomenon of healing by "grafting," are corroborative of this view.

**SYMPTOMS.**—The precise clinical phenomena associated with inflammation necessarily present much diversity, but the following outline will indicate their general nature.

**1. Local.**—*a.* If an inflamed part is visible it usually presents the characteristic *objective* phenomena already described under the anatomical characters, especially redness, swelling, and increased local heat.



*b.* Inflammation is commonly attended with *local subjective symptoms*. Of these pain is one of the most frequent, its intensity and characters differing much according to the tissue affected; it may be entirely absent, however, even when structures are involved inflammation of which is usually attended with much pain. Anything that disturbs or irritates the affected part will generally aggravate the painful sensations; while there is almost always tenderness on pressure, if the inflamed part can be thus reached, which may exist even without any spontaneous pain. Other morbid sensations, not amounting to actual pain, are often complained of, such as uneasiness, itching, burning, fulness or tension. These symptoms are necessarily due to the nerves being affected in some way or other, either being involved in the inflammatory process, or pressed upon by exudation. Sympathetic pains are sometimes referred to parts distant from the seat of inflammation; or pain may only be felt in some structure which is supplied by the same nerve as that which is affected.

*c.* The *functions* of inflamed organs and tissues are always disturbed more or less, on account of the vascular disorder; of actual changes in the involved textures; of the mechanical effects of the pathological products; and of the acts which these frequently excite with a view to their removal, such as cough in bronchitis. Secretions are often modified as to quantity, general characters, and composition, when the organs forming them are inflamed.

*d.* The accumulation of effusion or exudation may lead to more or less serious symptoms, by *interfering with neighbouring structures and organs*. This is well exemplified in cases of inflammation of serous membranes leading to much effusion.

*e.* When internal parts are the seat of inflammation, this condition can often be made out by *physical examination*, which reveals physical signs of a more or less significant character.

**2. General or constitutional**—At present it must suffice to state that the symptoms of *acute* inflammation are those of *fever* or *pyrexia*, which will be hereafter described. The fever is usually of the so-called *inflammatory* type at the outset, but differs much in its intensity, especially according to the tissue affected. The occurrence of suppuration is often indicated by one or more severe rigors, and the fever is then apt to assume the *adynamic* or *hectic* type. *Typhoid* or *adynamic* symptoms are also liable to be developed under other circumstances, but particularly if the inflammation assumes a low form, or if it terminates in gangrene.

In many instances the blood is hyperinotic, containing excess of fibrinogenous elements, and coagulating firmly, often presenting the "buffy" coat. Water is in excess, but albumen and salts are deficient. The red corpuscles often show a marked tendency to run together and, under the microscope are seen to form "rouleaux."

The fever attending inflammation is *symptomatic* or *sympathetic*, but its cause is not satisfactorily explained. It has been attributed to centric nervous disorder; to general vaso-motor disturbance; and to increased temperature of the entire mass of blood, consequent upon local excessive production of heat, but experimental observations are opposed to the last theory. In some cases the pyrexia probably depends upon septic products circulating in the blood.

**VARIETIES.**—Many named varieties of inflammation are recognized, founded on different characters. Thus it is said to be:—*a. Acute, sub-*

*acute, or chronic*, according to its intensity and rate of progress. *b. Sthenic or asthenic*, according to the general symptoms present. *c. Plastic, adhesive, suppurative, ulcerative, or gangrenous*, according to its products and mode of termination. *d. Circumscribed or diffuse*. *e. Healthy or phlegmonous, or unhealthy*. *f. Primary or idiopathic, or secondary*. *g. Non-specific or specific*, the latter including rheumatic, gouty, syphilitic, gonorrheal, strumous, tubercular, and other special forms.

TREATMENT.—It is not easy to give even a general outline of the treatment of inflammation, as this has to be so materially modified under different circumstances. At present only the chief principles of its management can be briefly pointed out.

A. Measures must be taken to *prevent* inflammation, should there be any condition present in which this process is likely to be set up. For instance, after an injury the part affected should be kept at rest, and appropriate remedies applied. In conditions of the blood which tend to originate secondary inflammations, every care should be exercised in warding off such influences as are likely to favour this event. If there is paralysis of any part, all local sources of irritation must be avoided. These illustrations will suffice for this point.

B. Supposing inflammation to have become established, the main indications for treatment, and the means for carrying them out are as follows:—

1. The first indication is to *subdue the morbid process* as soon as possible, and to prevent or limit the accumulation of the various exudations and effusions. In order to carry out this principle, it is essential to remove the cause of the inflammation, if practicable; to keep the part affected in as complete a state of rest as possible, physiological as well as physical; to avoid every source of irritation; and in many instances to attend to position, so as to obviate accumulation of blood. By such measures further disturbance will be avoided, while the involved structures are left in conditions most favourable for recovery.

The active measures employed are those usually termed *antiphlogistic*, which have for their more immediate object the lowering of the increased vascular action in the inflamed tissue. It is necessary to allude briefly to the most important of these agencies.

*a. Removal of blood.* This was in times past the great remedy for inflammation, but at the present day the tendency is to go to the opposite extreme, and to ignore blood-letting altogether. Blood may either be removed by venesection—*general blood-letting*—by which the heart's action is at the same time moderated; or it may be taken immediately from the vessels in the neighbourhood of the affected structure, by means of leeches, cupping, punctures, scarification, or incisions—*local blood-letting*. With regard to the former method, without entering into any discussion on the subject, I venture to express the opinion that it is not often required in medical practice, and great care should be exercised in determining that any individual case demands its adoption. As will hereafter be pointed out, inflammation of certain tissues and organs may necessitate venesection, but it should never be practised if the patient is debilitated, or if the inflammation is dependent upon some morbid poison in the blood. If performed at all, it should be had recourse to at an early period, before inflammatory products have accumulated to any extent. Local blood-letting is frequently most serviceable, and there can be no



doubt that it is not made use of to the extent which it deserves. By this means the vessels of a part can be considerably relieved, and thus a most beneficial local effect produced, while the general bulk of the blood is not materially diminished, or the patient injured in any appreciable degree.

*b.* Some powerful medicinal agents have of late years come into vogue in the treatment of inflammation, namely, certain *vascular depressants*, which affect the circulation through a direct influence upon the heart and vessels. Of these the most important are aconite, veratrum viride, and digitalis in full doses. Tincture of aconite has been found especially useful in the milder and limited forms of inflammation. Tartar emetic has long occupied a prominent position in the treatment of certain inflammatory affections, and justly so; it exerts a powerful influence over the heart, while at the same time it increases some of the secretions.

*c.* A class of remedies often of much value, if properly employed, are those which increase the principal secretions and excretions, and thus relieve the blood-vessels, namely, *purgatives*, *diaphoretics*, and *diuretics*. Purgatives must be used cautiously, but it is generally advisable to keep the bowels freely open, and particularly when the blood is loaded with products of tissue-waste. The best diaphoretic is some form of bath, especially the vapour, hot-air, or Turkish bath. Jaborandi has been found useful in some forms of inflammation. Salines are also frequently of much service. Of course these classes of remedies are severally contra-indicated should there be any local inflammation of the bowels, skin, or kidneys.

*d. Local treatment.*—There are certain important local means of subduing the increased vascular action in inflammation. Among these the most valuable is the application of cold. Cold may be applied by means of rags dipped in water or evaporating spirit-lotions; irrigation; ice, or a mixture of ice and salt contained in a bladder or india-rubber bag. It is in the early stage that this remedy proves most useful, and when the inflammation is quite superficial or affects structures near the surface. Heat and moisture act very beneficially in some cases, applied in the form of hot poultices or hot fomentations. In others, turpentine fomentations, dry-cupping, mustard poultices, or blisters are valuable. All these applications act mainly by inducing determination of blood to the surface. The local use of belladonna has been found valuable in inflammation of superficial parts, especially a mixture of equal parts of extract of belladonna and glycerine; lead-lotion and allied applications are also useful in some cases.

2. The second indication is to endeavour to promote the speedy absorption, or the removal in some other way, of *exudation or other morbid products*, and thus to restore the implicated organ or tissue to as normal a condition as possible. This will be favoured by attention to many of the points already mentioned, such as rest and position. In order to aid absorption certain *alteratives* are extensively used. Of these mercury in some form is very commonly employed. In syphilitic inflammations this drug acts most efficiently, but in other cases it ought as a rule to be avoided, or at least used with particular caution; a great deal of harm has been and still is done by its indiscriminate use. Iodine, especially in the form of iodide of potassium, is often of real value. Liquor potassæ and the alkaline bicarbonates are also serviceable sometimes.



Local measures are frequently followed by excellent results, particularly the employment of various forms of counter-irritation, such as blistering, painting with preparations of iodine, the use of irritating liniments, issues, setons, or the actual cautery. Friction and regulated pressure are in some cases most serviceable, and in practising the former it may be advisable to use absorbent liniments or ointments. Mercurial ointment is much employed in this way.

Absorption may undoubtedly be assisted in some instances by acting freely on the various excretory organs, especially by promoting the functions of the skin by means of suitable baths.

In some cases the products of inflammation cannot be absorbed, and then it may be necessary to have recourse to operations for their removal. In others, what has to be aimed at is to encourage certain acts by which these products are discharged, such as the act of coughing in cases of bronchitis.

3. In the next place the *general condition of the patient* must be attended to:—The various forms of *fever* met with must be treated according to the principles which will be laid down when considering this subject. If the inflammation is of a *specific* character, special remedies are called for, such as mercury in syphilis, or colchicum in gout. In certain other forms of inflammation also particular medicines have been found most useful, such as tincture of iron in erysipelas, and chlorate of potash in inflammations about the mouth and throat. Diet must be regulated according to circumstances, and it is impossible to lay down any definite rules, so much depending on the part affected, the state of the patient, and other circumstances. Should there be any tendency to depression, nourishing diet and stimulants are called for, often in considerable quantities, and this is especially the case if suppuration, ulceration, or gangrene sets in. Tonics, such as quinine, bark, mineral acids, or steel, as well as cod-liver oil, are then also indicated. Of course it is necessary to pay careful attention to all hygienic conditions.

4. Every precaution must be taken against the occurrence of *un-toward terminations*, such as suppuration, ulceration, or gangrene; should either of these occur, however, it must be treated by appropriate measures. The escape of pus must be encouraged, or its formation checked; ulceration healed; and the separation of dead parts promoted.

5. It is often necessary to attend to *local symptoms*, these necessarily depending upon the part affected. Among them a prominent one is pain. For its relief many of the remedies already considered are very valuable, but the most important drug for this purpose is opium. It is exceedingly serviceable in many inflammations in various other ways, such as by inducing sleep, stopping the peristaltic action of muscular tissues, allaying irritability, and probably directly influencing the inflammatory process. Opium is contra-indicated or must be given with great caution under certain circumstances, namely, when the respiratory organs, kidneys, or brain are involved. Morphia, hydrate of chloral, bromide of potassium, tincture of henbane, and other *sedatives* are also very useful in many cases for the relief of pain and sleeplessness.

---

## CHAPTER V.

## ALTERATIONS IN GROWTH.

## I. HYPERTROPHY.

HYPERTROPHY implies an over-growth, and ought to be strictly limited in its meaning to the "increase in an organ or structure of its normal tissue-elements." The tissue may be augmented either from an enlargement of its previously-existing constituents—**simple hypertrophy**; or from the formation of new and additional elements—**numerical hypertrophy** or **hyperplasia**. It must be remembered that an organ is usually made up of several structures, either of which may become hypertrophied, and thus its active functions may be improved or the reverse. For instance, as has been pointed out by Dr. Quain, in the heart either the muscular tissue, the fibrous tissue, or the fat may be increased, and each form is a true hypertrophy of a certain kind. In most cases, however, it is the active tissue of an organ which is increased, and its functions are thereby promoted. Muscle is peculiarly prone to become hypertrophied, both voluntary and involuntary.

ÆTIOLOGY.—1. In the great majority of cases hypertrophy is the result of *an organ or part being called upon to do extra work*, and it is truly a conservative or compensatory lesion, to ward off evil consequences which might otherwise ensue. Thus, in the case of hollow muscular organs, such as the stomach, heart, or bladder, whenever an obstruction exists at an orifice or elsewhere, interfering with the passage or exit of their contents, the tissues, especially the muscular, almost always hypertrophy. Involuntary muscular tissue is very frequently hypertrophied, and this may be a natural event for a certain definite purpose, of which the pregnant uterus affords an illustration. When the elements of a secretion or excretion accumulate in excess in the blood, hypertrophy of the organ or organs whose function it is to remove them often ensues. For example, if one kidney is unable to perform its functions, the other becomes enlarged and thus does double duty. If either lung is incapacitated from any cause, the opposite one generally becomes hypertrophied.

2. Probably *continued excessive action of an organ due to mere nervous irritation* may lead to hypertrophy. Nervous palpitation of the heart, for example, does in exceptional cases cause permanent enlargement of this organ. It is a familiar fact that voluntary muscles enlarge in proportion to the degree in which they are exercised.

3. *Excess of blood flowing to a part* may unquestionably originate hypertrophy of the structures thus unduly supplied with nutriment.

4. Increase in a particular tissue may result from *the presence of an excess of its formative elements in the blood*. This is illustrated by the large formation of adipose tissue throughout the body which occurs in some individuals whose blood contains an excess of fat.

5. Occasionally hypertrophy of certain structures takes place without any obvious cause.

**ANATOMICAL CHARACTERS.**—Increase in weight is the only necessary result of hypertrophy. Usually there is enlargement of an organ thus affected, and sometimes change in shape, but not always. The walls of hollow organs are generally thickened. The tissue involved may be quite natural as regards colour, consistence, and other physical characters; or these may be more or less altered. The newly-formed structure is often prone to undergo degeneration, as happens not uncommonly in the case of the heart, and this leads to further changes in appearance and other characters.

**SYMPTOMS.**—It frequently happens that hypertrophy of an organ is not attended with any symptoms whatever, especially when it is compensatory. There may be signs of its increased functional activity, of which the patient is also sometimes conscious, as in the case of the heart, and this excessive action occasionally leads to more or less serious consequences. An enlarged organ may also mechanically interfere with neighbouring structures. Physical examination often reveals hypertrophy when it cannot be detected in any other way.

**TREATMENT.**—Should anything be required in the way of treatment, the indications are to get rid of the cause of the hypertrophy, if practicable; to reduce the functional activity of any organ affected; to diminish the supply of blood, or alter its composition; and to use such remedies as are known to influence the growth of any special organ or structure.

## II. ATROPHY.

**ATROPHY** is the reverse of hypertrophy, and implies a diminution in the size or number of normal tissue-elements, the former being termed **simple**, the latter **numerical** atrophy, but both forms are frequently met with in combination.

Degeneration often accompanies atrophy, and when structures are actually destroyed and disappear, they must of necessity pass through a process of decay during their removal—*necrobiosis*. Atrophy may be general, involving all the tissues and fluids of the body, some, however, more than others; it may be limited to a particular class of structures, such as the muscular or glandular; or it may only affect a special organ, or even one of its constituent tissues, for example, the heart, liver, or kidney.

**ÆTIOLOGY.**—1. Whatever interferes with the *proper nutritive qualities of the blood* will give rise to general wasting to a greater or less degree. Hence this may arise from direct loss of blood; from deficiency in the quantity or quality of the food; or from diseases which interfere with digestion and assimilation, as well as those which lead to the excessive consumption or waste of the nutritive elements of the blood, such as Bright's disease, diabetes, prolonged suppuration, or phthisis. In cancer there is frequently an extraordinary degree of wasting, involving also the internal organs.

2. Combined with the foregoing cause, or acting alone, there is often *increased waste of tissue*, which cannot be repaired, such as that which occurs in fevers and many other diseases. In some instances atrophy from this cause is limited to one organ, of which acute atrophy of the liver affords an illustration.



3. The *vitality* and *nutritive activity* of the tissues generally may be impaired, or only those of some particular part or organ, and thus general or local atrophy may result. This is in many instances the normal course of events, associated with a natural impairment or cessation of functions, as, for example, in *senile* atrophy, of which it is an important element, as well as in the wasting of organs or structures, such as the thymus gland, spleen, and lymphatic glands, which at a certain period of life become atrophied because their functional activity is at an end. The same thing is seen in the rapid diminution in the size of the uterus after delivery. In other instances the impairment of vitality is due to some previous disease, such as inflammation. Excessive use on the one hand, or deficient exercise on the other, may produce the same effect. There can be no doubt that if certain organs are exercised unduly they may waste, for instance, the brain or testicle; while examples of the opposite condition are found in the wasting of the muscles of paralysed limbs, of bone after amputation, or of nerves after their connection with the cerebro-spinal axis has been severed.

4. An important cause of atrophy is a *deficient supply of arterial blood*, in whatever way this may be brought about, whether by something directly interfering with its entrance into a part, or by over-loading of the veins in long-continued mechanical congestion. To some extent this will explain senile atrophy, the heart and arteries having undergone degeneration, and the circulation being consequently impeded. It is in the production of local atrophy, however, that this cause mainly acts, and any structure may be affected if the supply of blood is not adequate to the demand, provided this is not so deficient as to lead to gangrene.

5. *Direct pressure* upon an organ or tissue may occasion atrophy, partly, but not entirely, on account of the interference with the vascular supply thus brought about. The pressure of pericardial adhesions and thickenings upon the heart occasionally causes atrophy of this organ, but the best illustration of this form of atrophy is that which follows the continued pressure of aneurisms and other tumours, by which bones and other structures are often extensively wasted.

6. It has long been known that the *nerves* exercise an important influence over nutrition, and hence when any nerve is paralyzed, atrophy is liable to follow in the structures which it supplies. This is partly to be attributed to the resulting cessation of functions; partly to the influence exercised on the supply of the blood through the vessels; but probably to some extent to the direct control which nerves exercise over the process of nutrition.

7. Certain *medicines*, such as mercury, iodide or bromide of potassium, and alkalis, when administered for some time, have the power of causing the absorption and wasting of particular organs or tissues. This power is made use of for the purpose of promoting the removal of morbid products.

8. Some forms of atrophy are met with the cause of which has not been satisfactorily determined, and is still a matter of dispute.

ANATOMICAL CHARACTERS.—In *general atrophy* or *marasmus* the entire body is more or less wasted, though this may be more evident in some parts than in others. The wasting may terminate in the most extreme emaciation. The fat is first removed, then follows muscular tissue,

and subsequently other structures become atrophied, as well as the elements of the blood and the internal organs. There is corresponding loss of weight, both as regards the whole body and particular organs. In a case of cancer attended with much emaciation which came under my notice, the heart only weighed  $3\frac{1}{4}$  ounces. The tissues generally feel flabby and wanting in healthy tone.

*Local atrophy* is necessarily attended with diminution in weight of the organ or part involved, and there is usually a lessening in dimensions as well, though this is not invariably the case, and there may even be apparent enlargement, as is sometimes observed in atrophy of bone. The physical characters of a wasted organ are generally altered. It appears paler than normal and less vascular, while its consistence is changed. Frequently it feels unusually dry and firm, and may be remarkably tough, because its fibrous element remains longest unaffected; wasted structures may, however, lose in consistence and become softened, while bone is in some instances much rarified and rendered very brittle, so that it crackles and breaks down readily under pressure. Atrophy occasionally leads to the entire removal of a particular structure, or even of an entire organ, so that not a trace of it remains.

It is needful to warn against mistaking other organic changes which lead to diminution in the size of organs for mere atrophy; and to call attention to the fact that congestion or other conditions may obscure the loss of weight which characterizes this process.

**SYMPTOMS.**—The appearance of the patient constitutes the most prominent clinical sign of general marasmus, but it must be remembered that the face may present a tolerably healthy aspect, while the body is much emaciated. The degree of wasting can only be made out accurately by weighing the patient from time to time, and this is especially required in the less-marked cases. It may be rapid or gradual in its progress. As a rule the patient experiences a sense of weakness, corresponding and in proportion to the emaciation, but this is not always the case. The muscles usually feel flabby and deficient in firmness. There may be signs of impaired activity on the part of certain organs, particularly the heart.

When an organ becomes atrophied, there may be no signs whatever of this condition, or more or less serious symptoms may result from interference with its functions. Local atrophy can also frequently be discovered by physical examination, as in the case of the heart and lungs; and when an external part, such as a limb, is wasted, this is easily recognized by proper objective investigation.

**TREATMENT.**—In the treatment of *general atrophy* the indications are:—1. To endeavour to remove the cause, and to cure the disease with which it may be associated. 2. To repair the waste, by introducing into the system a due supply of nutritious food, in such form as it can be best assimilated. Milk and cream are of great service in many cases, as well as prepared amylaceous and other kinds of food; alcoholic stimulants in moderation are also often very valuable. If from any cause food cannot be swallowed, it is frequently of great consequence that it should be administered by means of the stomach-pump or enemata. 3. To attend to the digestive organs, if required, giving remedies to improve the appetite, or to promote or assist digestion. 4. To regulate the hygienic conditions, change of air being in many cases an important part of the treatment. 5. To administer medicines, either with the

view of curing a disease, or of checking any pathological process which may be the cause of wasting, such as fever; or of giving tone to the system, and assisting in its nutrition. Cod-liver oil is a most valuable remedy in many forms of marasmus.

In *local atrophy* attention must be mainly directed to improving the nutrition of the affected part; at the same time guarding against making any undue call upon it; or, on the other hand, as in cases of atrophy from paralysis, endeavouring to rouse the involved tissues into activity, and thus to promote their growth and vitality.

## CHAPTER VI.

### DEGENERATIONS.

STRICTLY speaking a *degeneration* is a *retrograde metamorphosis*, and implies the conversion of some tissue into one less organized than itself, which is incapable of performing efficiently its normal functions. This may result either from an immediate change in its albuminoid components; or from a molecular absorption of the structural elements, and their replacement by others lower in the scale. The term *degeneration* is, however, also applied to certain pathological processes, in which a new material derived from the blood is deposited in the midst of the original elements of a tissue, which frequently leads to their absorption, and may ultimately replace them entirely. To these two kinds of so-called degeneration the terms *metamorphosis* and *infiltration* are respectively applied. They comprehend pathological changes of the utmost importance, which need to be severally considered in some detail.

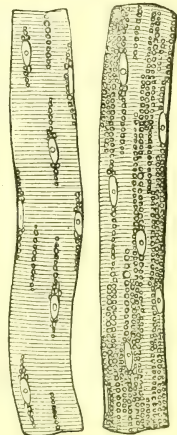
#### I. FATTY DEGENERATION.

This includes **Fatty metamorphosis** and **Fatty infiltration**, each of which requires separate notice.

1. **Fatty metamorphosis.**—The fact of the direct conversion of the albuminoid or protein elements of tissues into fat has been established by observation and experiment. It takes place in certain physiological processes, and is a frequent pathological occurrence, being also one of the natural events of decay in old age, when it often leads to serious consequences. The formation of adipocere after death is due to fatty degeneration, and this change can be produced artificially in dead tissues. The process may take place in connection with cells or fibres. The fat is usually deposited in a granular form, but ultimately it may accumulate into masses, or run into drops of oil. The granules are recognized by their dark and distinct outline; their peculiar refractive power upon light; and their solubility in ether. Fatty metamorphosis can be best studied by a description of the process as observed in certain individual tissues.

**Muscular tissue.**—Voluntary muscles may become the seat of fatty change, but it is in the fibres of the heart that this degeneration is most commonly observed. Under the microscope these fibres are seen

FIG. 3.



Fatty degeneration of fibres of striped muscle, (Rindfleisch.)  
300.



in the early stage to be somewhat dim as regards their transverse striæ, owing to the presence of a few minute fat-granules, arranged either in transverse or longitudinal lines, or in an irregular manner. Ether dissolves the fat and brings the striæ again into view. The increase in the number and size of the granules causes them to become more and more obscure, until eventually every trace of muscular fibre disappears, and its place is occupied by fat molecules and oil drops. In voluntary muscular tissue the sarcolemma may ultimately rupture, and the fat then becomes scattered about. The cells of involuntary muscular tissue are also liable to undergo fatty change, granules gradually filling them up and concealing their nucleus, complete destruction finally ensuing. The conversion of muscle into adipocere after death is, as already mentioned, a form of fatty degeneration.

*Blood-vessels.*—The arteries are very prone to fatty degeneration as age advances, the process either starting as an immediate change in the cells of the inner coat or in the muscular coat, or being associated with the condition named *atheroma*. At last the tissues may be more or less destroyed and carried away by the blood-current, leaving uneven erosions on the inner surface of the vessels. The capillaries are also apt to undergo a fatty change.

*Nerve-tissues.*—Both nerve-cells and nerve-fibres, are subject to fatty degeneration, becoming the seat of much molecular fat, and finally breaking down. This is well seen in softening of the brain or spinal cord, and in the changes which occur in the peripheral portion of divided nerves.

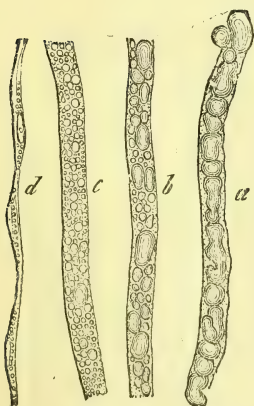
The following may be mentioned in further illustration of fatty degeneration in cells. The so-called “compound inflammatory globules” or “exudation-corpuscles,” as well as pus-corpuscles, are merely the

result of the conversion of the contents of leucocytes or other cells into granular fat. In the epithelium of the renal tubes, as well as in the cells of the liver, supra-renal capsules, and lymphatic glands, fatty degeneration also occurs; while the “*arcus senilis*” is due to the same process in the cells of the cornea. Most organs in their natural decay undergo this change; and it is further exemplified in the formation of many secretions; in the production of the corpus luteum in the ovary; and in the degenerative changes which the placenta undergoes on the approach of the full period of pregnancy. Morbid products, such as cancer, tubercle, infarctions, inflammatory exudations, and various tumours are likewise subject to fatty metamorphosis.

When cells undergo fatty degeneration, they often enlarge and become more spherical and distended. The granules first appear at a distance from the nucleus, scattered irregularly; they then increase in number and size, obscure the nucleus, and finally render it quite invisible. The cell-wall is often ruptured or absorbed, leaving merely an accumulation of

granular fat, which frequently separates into its constituent particles, owing to intermediate liquefaction.

FIG. 4.



Fatty degeneration of double-bordered nerve fibres in the peripheral part of a divided cerebro-spinal nerve. *a.* After three days have elapsed; *b.* After a fortnight; *c.* After three weeks; *d.* After two months,  $\frac{1}{300}$ . (Rindfleisch.)

**Caseation; Caseous or Cheesy Degeneration.**—These terms have come into considerable prominence of late years, and are used to signify the conversion of various structures into a kind of soft, dryish, cheesy-looking substance of yellowish colour. It is really a process of partial fatty degeneration with drying, and the material formed is found to consist of withered cells, fat granules, partially saponified fat, and crystals of cholesterin. Caseation generally occurs in connection with some morbid product, or where there is a great accumulation of cells pressing closely upon each other, and it is especially met with where vessels are few, so that the tissue is dry. Pulmonary phthisis affords some of the most frequent examples of caseous degeneration, but by no means necessarily associated with tubercle. It is also often seen in scrofulous lymphatic glands, cancer, and chronic abscesses. Ultimately a more or less creamy or puriform-looking fluid substance may be produced, or a kind of fatty emulsion, which may be completely removed by absorption or in other ways; or it may become encapsuled by dense tissue, and finally calcify. Many pathologists regard caseous material as a morbid poison, which, after absorption into the blood, is capable of originating tubercle by an infective process.

**ÆTIOLOGY.**—Some interference with nutrition, either affecting the general system or some local part, is, in the great majority of cases, the immediate cause of fatty degeneration. Possibly the presence of an excess of fat in the blood may occasion a fatty change. The obvious causes to which it may be due are:—1. Senile decay, during which most of the tissues undergo degeneration. 2. Deficient supply of arterial blood to a part, owing to obstruction or compression of, or morbid changes affecting the coats of the vessels. 3. Some general disease or condition lowering vitality, such as phthisis or cancer; or considerable loss of blood. In this connection may also be mentioned the acute fatty degeneration which occurs in bad cases of certain acute specific fevers and other acute diseases. 4. Poisoning by phosphorus, arsenic, or antimony. 5. Congestion, inflammation, very rapid development of a tissue, or undue exercise of its functions, whereby its vitality is impaired.

**ANATOMICAL CHARACTERS.**—The changes induced by fatty degeneration are usually quite obvious if the process is at all advanced, but in less marked cases they can only be detected by the aid of the microscope. There is an alteration in colour, the affected tissue becoming usually paler than normal, and assuming a yellowish or brownish tint; this is well seen in muscular tissue, but in some instances, as in softening of the brain, the colour ranges from white to red according to circumstances. A tendency towards opacity is also evident. One of the most marked changes is a diminution in the consistence of the affected structure, which varies from a slight degree of softening, the tissue breaking down under pressure or tearing more easily than in health, to its conversion into almost a fluid pulp. All vital properties, such as elasticity or contractility, are at the same time impaired or lost, as well as the power of resistance. When the process is advanced, the affected tissues may have an oily feel, and ether will dissolve out a considerable quantity of fat.

The remote pathological consequences of fatty degeneration are often very important. For instance, the affected structures are liable to give way and rupture, as happens sometimes in the case of the heart or vessels. Arteries may also become the seat of aneurism; or by diminishing the supply of blood to structures which depend on them for their

nutrition, the vascular changes ultimately lead further to the degeneration of these structures.

**SYMPTOMS.**—There may be no clinical indications whatever of fatty degeneration. This may be due to the fact that the structure involved is of but little consequence; but even when an important organ is implicated, such as the heart, the process may go on insidiously until it becomes very marked, and is only revealed by some sudden grave symptoms. The phenomena to be looked for are those significant of impaired functional activity on the part of the affected structure, or of the secondary lesions to which fatty degeneration may give rise. Physical examination may also reveal the change in some instances, as in the case of the heart and vessels.

**2. Fatty Infiltration or Growth.**—This is a process essentially distinct from fatty degeneration, there being no necessary change in the elementary structures themselves, but merely a deposit of fat from the blood within the cells of the part affected, and this infiltrates the tissues to a variable extent. It might, in fact, be described as a *fatty hypertrophy*. The deposit occurs in the form of oil-drops, which ultimately run together, completely obscuring the other contents of the cells, without of necessity destroying them. In course of time the tissues may degenerate, from the mere pressure of the infiltrated fat, and may even finally become absorbed. The best examples of fatty growth are found in the increase of ordinary adipose tissue observed in some individuals, subcutaneous as well as around internal organs, constituting *general obesity*; in connection with the heart; in the cells of the liver; in voluntary muscles; and in the form of fatty tumours. In the first two instances the connective-tissue cells become filled with fat; while in the liver the hepatic cells are more or less loaded with drops of oil, which cause them to become larger and more spherical, and hide their contents.

FIG. 5.



Liver-cells, infiltrated with oil,  $\frac{300}{\times}$  (Rindfleisch.)

**ÆTIOLOGY.**—1. *Excess of fat in the blood* is a common cause of general obesity, and of fatty infiltration of organs. This may result from consumption of too much fat, or of aliments which contribute to its formation; from a deficiency in the amount of exercise taken, and general luxurious habits; or from both these causes combined. There is also a diminution in the ordinary waste of fat under these circumstances.

2. In certain *wasting affections* some of the organs are prone to become the seat of fatty infiltration, especially the liver. This is best seen in phthisis, and is supposed to be due to the absorption of the general fat, and its consequent accumulation in the blood, from which it is afterwards deposited in the liver.

3. Undoubtedly *interference with the respiratory process* may lead to fatty infiltration, because the fat is then not properly consumed, and thus its not infrequent occurrence in connection with certain pulmonary and cardiac affections will be partly accounted for.

4. *Local inactivity* may be the cause of fatty infiltration. Thus it may be observed in voluntary muscles which are paralyzed or otherwise rendered inactive.

**ANATOMICAL CHARACTERS.**—Fatty infiltration may occasion enlargement of organs, accompanied with a certain degree of alteration in form,



there being a tendency towards roundness of margins and of the general outline. The colour also becomes paler, and may be similar to that of adipose tissue. The principal changes observed are that the affected structure is softened, and has the doughy feel characteristic of ordinary fat; while evidence of the presence of more or less oil may be obtained, either by the finger, the knife, blotting-paper, or ether. Microscopic examination reveals the appearances already described.

**SYMPTOMS.**—General obesity is usually evident enough in the appearance of the individual. The subjects of this condition feel languid, are deficient in bodily activity and vitality, disinclined for exertion, and easily tired. The muscles are wanting in healthy tone and firmness. Digestive disturbances are common; the cardiac action is easily disturbed; and there is shortness of breath on exertion. As a rule the mental faculties are also dull and inactive. In extreme cases the patient experiences great distress and discomfort, and is incapable of any exertion.

Fatty infiltration of an organ may be indicated by impairment of its functions; as well as by physical signs. The condition is, however, often difficult of diagnosis; but it may be of little moment.

**TREATMENT.**—In the treatment of fatty degeneration all that can be done is to improve the general nutrition, as well as that of the affected structure, by the aid of good food, tonics, and cod-liver oil. Care must be taken to avoid any undue strain upon important structures which are the seat of this change, such as the heart or vessels.

General obesity must be treated by restricting and regulating the diet, especially limiting those aliments which contribute to the formation of fat; by making the patient avoid sedentary habits, and take sufficient exercise; by keeping the bowels acting freely, especially by means of mineral waters; by employing baths to promote the action of the skin; and in every respect regulating the habits of the patient. Medicines are of but little service, but liquor potassæ seems to be of use in some cases, probably by injuring the digestion. Iodide of potassium in large doses has also been employed. Preparations of *fucus vesiculosus*, or sea-weed, have been administered with supposed advantage. Iron is of service when obesity is associated with anæmia. Local fatty infiltration requires no special treatment.

## II. MINERAL OR CALCAREOUS DEGENERATION.—CALCIFICATION.— PETRIFICATION.

It is important to distinguish *calcification* from true *ossification*. In calcification there is no formation of bone, but merely an infiltration of the tissue involved with particles of calcareous matter. These assume the form of very minute molecules, chiefly deposited irregularly between the histological elements, but partly in their interior as well. Under the microscope they look like dark, opaque, irregular particles under transmitted light, and when aggregated have a glistening aspect. They often resemble fat in appearance, but may be distinguished by their solubility in dilute mineral acids, this being frequently attended with effervescence and the formation of small bubbles of gas, owing to the decomposition of earthy carbonates. The deposit occurs first immediately around small vessels, where these exist, but ultimately it may increase so as to form irregular patches or concretions of considerable extent and size. Chemically it is made up chiefly of calcic and magnesian

phosphates and carbonates, but other salts are also present, and the composition is not uniform in all structures.

Calcification is particularly prone to occur in tissues which have lost their vitality, and which have previously undergone other forms of degeneration, especially the fatty change. It is in fact very commonly the final stage of the degenerative process, after which no further alteration can take place. Among its most frequent seats are the arteries, and the valves and orifices of the heart, and it is in connection with these structures that its injurious effects are most obvious. Calcareous deposit may, however, be met with in many other tissues, namely, in fibrous or fibro-serous membranes, such as the pericardium, dura mater, or tunica albuginea; in the walls of hollow organs, as the gall-bladder or stomach; in the pia mater and choroid plexuses of the brain, constituting "brain-sand;" in cartilage, muscle, and nerve-tissues; in various organs and glands, for example, the kidneys, lungs, absorbent glands, thyroid, prostate, and pineal gland; and in connection with different morbid products, as tubercle, cancer, inflammatory exudations, blood-clots, chronic abscesses, and tumours of all kinds.

ÆTIOLOGY.—1. Calcification is usually associated with *deficient vitality and nutritive activity*, either general, such as attends advanced age; or local, being then dependent upon an insufficient supply of blood, with slowness of the circulation. As already stated, it is frequently the termination of other atrophic and degenerative processes. With regard to the immediate cause of the accumulation of calcareous matter, it is supposed to be due partly to inability on the part of the tissues to take up the nutritive fluid in which the salts are dissolved; partly to the precipitation of these salts, because the carbonic acid which holds them in solution escapes, owing to the stagnation of the fluid.

2. Occasionally calcification is dependent upon *the presence of an excess of calcareous salts in the blood*. This may arise in connection with diseases of bone, such as mollities ossium or extensive caries or necrosis, in which its salts are rapidly absorbed, and "metastatic deposits" are afterwards formed in other parts, often involving many structures and organs. An interference with the urinary secretion may also lead to this condition, the salts not being properly eliminated, and when this happens the kidneys are particularly prone to become the seat of calcareous deposit.

ANATOMICAL CHARACTERS.—The deposit of calcareous matter occasions more or less roughness, hardness, stiffness, or rigidity, often combined with brittleness. A gritty sensation is felt on making a section, and when membranes are involved they can frequently be broken up with a crackling noise. Stony masses of some size may be formed sometimes. Now and then a kind of chalky fluid is produced, or a substance like cement. The most injurious consequences often result from the change in size and shape of structures; the roughness of surfaces; the interference with free movement, and with elasticity and contractility; and the brittleness which attend calcification. Thus, in the case of arteries, it narrows their calibre, makes them rough and rigid, destroys their elastic and muscular tissues, and renders them liable to be easily ruptured. Hence it leads to insufficient supply of blood to parts, with consequent atrophy, degeneration, or gangrene; or to the formation of clots obstructing the vessels; or to hæmorrhage. In connection with the valves and orifices of the heart it may cause serious obstruction and interference with their normal functions. In some cases, however,



calcification is distinctly a favourable termination, and indicates the cessation of injurious morbid processes. It is, in fact, in such instances a practical cure, and the calcified substance may remain for many years inert, without causing any further disturbance. This is well seen in connection with phthisical consolidations and serofulous lymphatic glands. A case fell under my notice some years ago, where, in a highly serofulous young man, aged 21, the whole of the absorbent glands within the abdomen were converted into calcareous masses, and had evidently been in this condition for many years without leading to any inconvenience, the patient dying from an entirely independent acute illness.

SYMPTOMS.—There may be objective signs of calcification, as in the case of the arteries. Various symptoms may arise from the injurious effects of the process mentioned above. Particles of calcareous matter from internal parts, for instance, the lungs, may be discharged externally. Frequently petrification is obviously a favourable event, and is indicated by the cessation of symptoms previously associated with some active disease.

### III. FIBROID DEGENERATION.

Tissues are sometimes gradually changed into a tough, inelastic material, made up of imperfect fibres resembling those of fibrous tissue. There is no apparent exudation to any extent, but a hyperplasia of the cellular-tissue elements occurs. The affected part becomes more or less opaque, whitish, thickened, and stiff, sometimes being hard and rough. The fibro-serous and serous membranes often present this change, in the form of thickened patches, which are well seen in connection with the pericardium. It also affects other structures, such as the coverings of organs, as that of the spleen or liver; the sheaths of vessels; or the valves, tendinous cords, and muscular tissue of the heart. The functions of the involved parts may be much impaired. Ultimately the structures may become calcified. This fibroid degeneration results from pressure and friction; from repeated traction; or sometimes from long-continued congestion. It is not always practicable to draw a line between the effects of this process and those of chronic inflammation, and some pathologists consider that the latter always precedes and is the cause of the fibroid change, which merges into the conditions termed *cirrhosis* and *sclerosis*.

### IV. PIGMENTARY DEGENERATION.—PIGMENTATION.

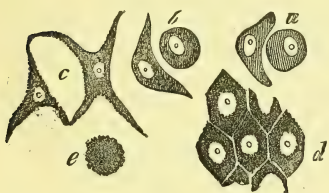
General or local changes of colour in tissues may result from various causes. As illustrations may be mentioned the colour due to jaundice or local staining by bile; that produced in adjacent structures by the action of the intestinal gases, or of those set free in mortifying parts; the bronzed hue of the skin observed in Addison's disease; and the discoloration following the prolonged administration of nitrate of silver. What is at present under consideration, however, is the deposit of actual pigment in connection with various textures, and it is necessary to discuss the origin, nature, and characters of the different kinds which may be met with.

1. In the great majority of cases in which pigment is found, it is derived from the colouring matter of the blood, which undergoes certain changes. This colouring matter may be present in some limited part, either from its mere transudation through the coats of the vessels; from the migration of red corpuscles; from actual hæmorrhage having taken



place; or from capillary stagnation. At first it is diffused, and stains the tissues which it involves, especially the cells, colouring their contents, but leaving the nucleus and envelope unaffected. After a while the colour changes, assuming a variety of tints, such as yellow, yellowish-brown, brown, reddish-brown, dark brown, grey or black; the exact hue depends much upon the length of time the pigment has been formed, and the tissue which it occupies. At the same time it separates into minute molecules, or crystals, or both, which are found within or outside the cells. The molecules may aggregate into larger granules. They are round or irregular in shape, well-defined, opaque or sometimes glistening in appearance. The crystals are in the form of minute oblique prisms, needles, or plates, which present various tints of yellow, red, brown, or black, and have a shining aspect. These particles are very persistent, and are not easily destroyed. They resist the action of ordinary acids; water, alcohol, and ether do not affect them; but they are dissolved by strong alkalis, forming a red solution, while concentrated mineral acids bring out a series of changes of colour. Chemically the pigment is supposed to consist of *hæmatoidine*, but when it becomes black it is named *melanin*. Examples of these changes of blood-pigments are frequently met with in connection with cerebral apoplexy; in pulmonary congestion or hæmorrhage; in subcutaneous extravasations; and in the formation of the corpus luteum in the ovary. The alterations in the colour of the "rusty expectoration" of pneumonia during the progress of this disease are due to the same cause. In certain affections the blood itself contains corpuscles enclosing a quantity of black pigment. This occurs in the rare disease called *melanæmia*; as well as after prolonged attacks of ague, when the spleen becomes enlarged, and contains an abundance of the same pigment, which is supposed to

Fig. 6.



Cells in various stages of pigmentary infiltration; (a, b, c, e.) from a melanotic cancer; d. Pigmented epithelium from the vessels (same specimen)  $\frac{300\times$  (Rindfleisch.)

be absorbed into the blood. It is, however, originally derived from the blood-pigment. The same condition is observed in melanotic tumours. Occasionally the minute portal vessels of the liver become filled with black pigment.

In some instances pigment is supposed to be directly formed and secreted in certain cells, but here again the blood must be looked upon as its ultimate source. This is presumed to occur especially in growths connected with tissues which normally contain much pigment, such as the choroid coat of the eye.

Allusion may here be made to the so-called *brown atrophy of the heart*, in which condition granules of brownish-yellow or blackish pigment form, the muscular fibres being at the same time atrophied, as well as often the seat of fatty change.

2. An important source of pigmentation in connection with the lungs and bronchial glands is the inhalation of certain substances along with the air breathed. All persons are liable to inhale small particles of carbon, the product of imperfect combustion, and this occurs especially in large towns and manufacturing districts. Hence it is found that the lungs become darker as age advances. It is, however, in the lungs of those who, owing to their occupation, are exposed to the constant breath-

ing of air containing various minute particles suspended in it, that the most marked alterations are observed. Colliers inhale fragments of coal, and their lungs become ultimately perfectly black. Miners, stonemasons, and others following similar occupations are also subject to changes in the colour of these organs, due to the inhalation of solid particles.

The carbon or coal exists in the form of minute granules, which resist all chemical change, and these, after entering the small bronchi and air-cells, somehow penetrate the tissues, passing into the epithelium cells, as well as into the tissue between the lobules and around the bronchi, where they lie either free or enclosed in the connective-tissue corpuscles. They are taken up by the lymphatics and conveyed to the bronchial glands, which also soon become quite black. Abundant pigment is seen within the cells which the expectoration contains, and the sputa may be perfectly black. The change of colour in these cases is, however, not entirely due to the direct deposit of material from without, but partly to the irritation caused by this material setting up inflammation, with stagnation of blood, the pigment of which undergoes the usual alterations.

#### V. MUCOID DEGENERATION.

Some tissues occasionally undergo a process of softening or liquefaction, to which the term *mucoid degeneration* is applied. They become changed into a mucilaginous substance, colourless and homogeneous, which yields *mucin*. In fact they seem to return to their original foetal condition. The change may take place extensively or in limited spots, in which, being surrounded by healthy tissue, it may give rise to an appearance of cysts. The intercellular tissue is most affected, but sometimes the cellular elements become involved in the degeneration. This degeneration is met with in cartilage, bone, serous membranes, and in the choroid plexuses of the brain. Some tumours are of a *mucoid* structure when first formed, and many others may undergo this change to a greater or less degree.

#### VI. COLLOID DEGENERATION.

The substance formed in colloid degeneration is of a jelly or glue-like consistence, glistening, transparent, and devoid of colour. It is derived from the albuminoid tissues, and differs from mucin in that it has sulphur entering into its composition, and is not precipitated by acetic acid. This degeneration also involves the *contents of the cells* themselves, and not the intercellular substance.

Ultimately separate portions of the colloid substance may coalesce so as to form considerable masses, which often appear to be contained in cystic cavities. New growths sometimes start as *colloid tumours*, and other tumours are liable to the change. Formerly all colloid tumours were looked upon as being of a cancerous nature, but it is now recognized that non-malignant tumours may also become the seat of this degeneration. Enlarged thyroid and lymphatic glands occasionally contain colloid material.

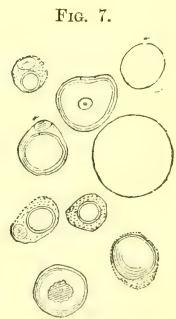


Fig. 7.  
Cells undergoing colloid degeneration. From a colloid cancer. (Kindscheisch.)

VII. LARDACEOUS DISEASE,—ALBUMINOID INFILTRATION OR DEGENERATION.—AMYLOID DEGENERATION.—WAXY DISEASE.

By these names, amongst others, a very important morbid condition is recognized. The following account conveys tolerably complete information as to the present state of knowledge concerning this subject, although there are some points which are still not settled.

**ÆTIOLOGY.**—Albuminoid infiltration is invariably a consequence of some previous disease, which in the great majority of cases is attended with *long-continued and excessive suppuration*, but this is certainly not absolutely necessary. The chief individual diseases with which this morbid condition is associated are :—*a.* Caries or necrosis of bones, and their consequences. *b.* Syphilis, especially if it has caused disease of bones with considerable suppuration, or if much mercury has been given. Children who are congenitally syphilitic may be the subjects of albuminoid disease. *c.* Chronic pulmonary phthisis, or other lung-affections attended with much purulent expectoration. *d.* Chronic empyæma, especially fistulous. *e.* Extensive ulceration of the intestines. *f.* Pyelitis and some other kidney-affections. *g.* Chronic glandular abscesses.

Albuminoid disease has also been attributed to prolonged ague or exposure to malarial influence; and to rickets, but the changes in organs in this disease, which were supposed to be due to albuminoid degeneration, are believed by some to be of a totally different nature.

**ANATOMICAL CHARACTERS.**—1. **Characters of the albuminoid material, and objective changes in the affected tissues.**—The material, the presence of which in certain tissues is characteristic of albuminoid disease, is colourless, refractive, structureless and homogeneous, somewhat tough and consistent, and at first nearly transparent. It is not prone to decomposition, and is unaffected by most chemical agents, but yields certain characteristic reactions. When a watery solution of iodine is applied to the cut surface of an affected organ, a deep reddish-brown or mahogany colour is brought out, but this is not invariable, even when the disease is advanced, and it may be of more service in indicating a slight degree of the change, especially when the solution is applied to microscopic sections. The subsequent addition of a drop of strong sulphuric acid may develop a violet or dark blue colour, owing to the precipitation of the iodine in a molecular form. Another test is the readiness with which albuminoid material is stained blue by solution of sulphate of indigo. This material may undergo granular or fatty degeneration; and it is also stated that it sometimes tends to contract, and to become changed into fibrous tissue.

When an organ or tissue is the seat of marked albuminoid disease, it presents certain striking characters. The organ is enlarged, sometimes to a great degree, but without any irregularity in form or outline, the surface being quite smooth, and the margins inclined to be rounded. The weight is proportionately increased; the specific gravity is high; and the organ feels heavy, solid, and firm. It may be cut into regular fragments, quite smooth and presenting sharp margins; or very thin slices may easily be removed. It can also be torn into pieces, while the consistence is peculiar, presenting a combination of toughness and



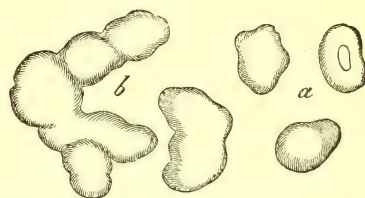
resistance with elasticity, resembling somewhat that of wax, or wax and lard combined—hence the names *waxy* and *lardaceous*. A section is dryish, paler than normal, anæmic, and presents a glistening or semi-translucent aspect, being also quite smooth, uniform, homogeneous, and compact. In many instances, however, the mischief has not extended to such a degree as to alter much the general physical characters of organs; and it may be limited to the vessels or to certain spots, as is well seen in the so-called “sago-spleen,” in which the material is confined to the Malpighian corpuscles. In still less advanced cases the change may only be detected by examining sections of the affected tissue under the microscope, and to these, well-washed, the iodine test may be applied. The minute arteries and capillaries are almost always first involved, especially their muscular coat, and the cells of their inner coat. The walls become thickened, the channel is narrowed, and on section the vessels remain patent, while they assume a compact, homogeneous, translucent, shining appearance, so that they come to resemble silvery cords or threads. After a time the material extends to the cells and intercellular tissues, enlarging the former and making them more spherical, at the same time displacing their normal contents, the nucleus being ultimately destroyed, so that the cells become converted into amorphous masses, with a tendency to irregular fracture.

They then coalesce; and the whole structure presents finally the peculiar glistening appearance mentioned above. It also involves the walls of ducts. It is supposed by some pathologists that the substance makes its way directly through the walls of the vessels, and afterwards extends into the tissues around.

**2. Organs and tissues involved.**—Albuminoid disease is particularly liable to affect small arteries and capillaries, cells, and involuntary muscular fibres. Any organ or tissue in the body may be implicated, and usually several organs are involved at the same time. The liver, spleen, kidneys, and absorbent glands are most frequently affected, but other structures are also attacked sometimes, namely, the stomach and intestines, supra-renal capsules, bones, voluntary muscles, brain and spinal cord and their membranes, tonsils, serous membranes, heart, lungs, pancreas, uterus, and bladder; morbid deposits, such as inflammatory exudations, tubercle, or cancer, may also present the albuminoid change. In some cases, when it follows disease of bones, it begins in the neighbouring lymphatic glands. In many of the organs above mentioned the disease seems to be limited to the minute vessels.

**PATHOLOGY.**—Various theories have been held as to the nature and origin of the morbid material characteristic of albuminoid degeneration. Virchow, on account of its chemical reactions, formerly considered the substance to be allied to starch or cellulose—hence the term *amyloid*; others believed it to be a form of cholesterin. These views have, however, been entirely disproved, and that commonly adopted at present is, that the material is of an *albuminoid* nature, being allied to albumen and other protein elements. The results of chemical analysis show that it is a nitrogenous compound. Dr. Dickinson affirms that organs in

FIG. 8.



Liver-cells infiltrated with amyloid matter. *a.* Isolated cells; *b.* A fragment of the secreting network in which the boundaries of the individual cells have ceased to be visible. (*Kindigsch.*)

which it exists are deficient in alkaline salts, and this observer has advanced the view that the substance consists of *de-alkalized fibrin*. Marcet found that the affected structures were deficient in potash and phosphoric acid, but contained excess of soda and chlorine. Cholesterin is also present. With regard to the origin of the albuminoid material, two distinct theories are held, namely, (1.) that it is the product of some local *degeneration* or *metamorphosis of albuminous tissues*; or (2.) that there is a *direct deposit from the blood*, in consequence of some alteration in this fluid, which deposit infiltrates the tissues. Nothing of the nature of this albuminoid substance has, however, been detected in the blood. Dr. Dickinson thinks that the blood is deprived of its alkali, as the result of prolonged suppuration, and that the material is then deposited. Dr. Grainger Stewart strongly advocates the degeneration-theory, and calls attention to the distinction between the waxy degeneration proper, and the secondary deposit of fibrinous material which results from it. Most pathologists are in favour of this theory.

**SYMPTOMS.**—It will only be needful to offer here a few general remarks with regard to the symptomatology of albuminoid disease. There are no definite clinical signs of the change until it has become somewhat advanced. It is, moreover, not always easy to make out, in a particular case, what symptoms are due to the original affection, and what to the albuminoid disease. Nutrition is usually impaired, the patient being thin or emaciated, it may be extremely so, at the same time becoming pale and anæmic, and presenting a peculiar transparency of tissues, or a waxy look. There is great debility in many cases, with a tendency to syncope. Oedema of the legs is often observed, due to weakness of the tissues and anæmia. With regard to the various organs, should either of these be the seat of albuminoid disease, its functions are liable to be interfered with more or less when the change becomes considerable, local symptoms being thus developed; while certain organs are frequently obviously enlarged, and may give rise to pressure-symptoms, at the same time presenting well-defined characters on physical examination.

**TREATMENT.**—This must be directed in the first place to the primary disease, suppuration being checked, or any other condition which is causing the albuminoid degeneration being attended to. At the same time the general health must be improved by good diet; attention to hygienic measures; and the administration of tonics, iron, and other remedies which may be indicated in particular cases. The use of the syrup of iodide of iron has, in my experience, been attended with considerable benefit in some cases, if persevered in for some time.

## SECTION III.

THE remainder of this work is chiefly devoted to the consideration of individual diseases. There has been much controversy with regard to the NOMENCLATURE and CLASSIFICATION of diseases, and many systems have been proposed. The arrangement adopted in the following chapters is mainly in accordance with that recognized by the College of Physicians, most of the complaints being described as they come under one or other of the groups mentioned below, though it will be expedient to deviate from this arrangement in some instances.

I.—GENERAL DISEASES.—These affect more or less the entire system, and although local morbid conditions are often present, they arise secondarily, as the necessary or accidental consequence of the general disorder. Under this class are included :—

(A.) The various **Idiopathic Fevers**, and certain other affections which are due to the action of a **specific poison** on the system, introduced from without. *Ex.* Scarlatina, small-pox, ague, hooping-cough, diphtheria, hydrophobia.

(B.) **Constitutional diseases**.—These maladies are dependent upon some unhealthy condition of the blood or general system, or a so-called *cachexia* or *diathesis*, which, however, is usually revealed by local lesions, often developed in several parts of the body at the same time or in succession. Many of them originate from the action of a definite morbid poison, either entering from without, or more commonly generated within the system, or handed down by hereditary transmission. In some of these diseases no such morbid agent can be detected. *Ex.* Rheumatism, gout, cancer, syphilis, tuberculosis, scurvy.

II. LOCAL DISEASES.—This group comprises the various affections to which the several organs and tissues of the body are liable. It will be convenient to describe under this heading not only primary local disorders, but also certain complaints which are more strictly of a general character, but which present special local manifestations.



## I. GENERAL DISEASES.

---

### A. IDIOPATHIC FEVERS AND ALLIED DISEASES.

#### ACUTE SPECIFIC FEVERS.—ACUTE EXANTHEMATA.

BEFORE proceeding to discuss the several diseases included under the above group, it will be expedient in the present connection to consider the following subjects:—Fever; Contagion; and Epidemics.

---

#### CHAPTER I.

### FEVER OR PYREXIA.

THE phenomena characteristic of fever may be observed in connection with two distinct classes of cases:—First, they may follow and be the result of some local lesion in a tissue or organ, especially inflammation, when the fever is termed **secondary, symptomatic**, or merely **pyrexia, fever**, or the **pyrexial state**. The fever attending pneumonia will serve to illustrate this group. Secondly, they may constitute the chief and primary deviation from the normal state, not being due to any evident local cause, and if any special organ or tissue becomes affected in such a way as to increase the pyrexia, this occurs as a secondary event. **Idiopathic, essential, primary**, or **specific** are the terms applied to fever under these circumstances, or it is simply denominated **a fever**. It then originates from the presence of some morbid poison in the blood, either introduced from without, or developed within the body. The acute specifics and rheumatic fever afford examples of this class.

**ESSENTIAL PHENOMENA AND SYMPTOMS.**—The precise symptoms which may be observed in different febrile diseases are necessarily very variable, both in themselves and in their combinations, but there are certain phenomena which are characteristic of the pyrexial condition, to which attention will now be directed. They may be arranged thus:—

1. **Rise in Temperature.**—This has been generally looked upon as the only absolutely essential sign of fever, provided the increase of temperature continues for a certain time. The excessive heat of the body may be revealed in the aspect or sensations of the patient, or the skin may feel hot or burning to the touch; but no reliance ought to be placed on these sources of information, and the existence of pyrexia can only be satisfactorily determined by the use of the thermometer. The temperature may range from only just above the normal to 108°, 110°, 112° Fahr., or even higher than this, but it does not often exceed 105° or 106°. When the temperature is below 101° fever is said to be “slight”; up to 103° “moderate”; to 105° “high”; and above 106°

the condition is termed *hyperpyrexia*. It may continue to rise for some time after death.

**2. Alterations in the Secretions.**—Pyrexia is attended with *deficient elimination of water* from the system, there being at the same time *excessive destruction of tissues*. Hence the secretions and excretions are as a rule diminished in quantity, as well as altered in quality, from which result some prominent symptoms, namely:—*a. Dryness and roughness of the skin*, though this is not always observed, in some cases there being more or less perspiration, which may be profuse. *b. Derangements of the alimentary canal*. The salivary, gastric, and intestinal secretions are deficient; hence the tongue is furred, and the mouth dry or clammy; the taste is unpleasant; there is much thirst, but no inclination for food; the breath is offensive; and the bowels are constipated. Nausea and vomiting are also not unfrequent symptoms. *c. Changes in the urine*. This is much diminished in quantity, high-coloured, very acid, with a strong odour, and of high specific gravity. It also generally contains an excess of its organic nitrogenized constituents, especially uric acid and urea, a much larger quantity of these substances being excreted during the twenty-four hours than in health, and deposits of urates or sometimes of uric acid taking place when the urine cools on standing. There is likewise an increase in hippuric acid, sulphates, phosphates, potash-salts, and colouring matters, though not always. Chlorides are commonly deficient, and may be absent altogether; while soda-salts are also below the normal. Abnormal ingredients may be present; while slight albuminuria is common in febrile cases.

**3. Derangements of the Circulatory System.**—The pulse is increased in frequency, and may rise to 120, 140, 160, or more, being as a rule in proportion to the temperature. A rise of 1° Fahr. has been stated to be attended with an increase in the frequency of the pulse of eight beats per minute, but to this statement there are many exceptions. In its other characters the pulse varies greatly under different circumstances, but a typical febrile pulse is full, bounding, and hard. In long-continued or severe cases it tends to become very weak, irregular, or even intermittent, these characters being associated with feeble action of the heart.

The blood is altered in composition, there being a diminution in the amount of alkalies present, and in the alkalinity of the serum; after a time the albumen and red corpuscles also become deficient, while the white corpuscles are often increased in number. In some forms of fever the proportion of fibrinogenous elements is much above the normal; in others it is greatly reduced. In certain cases the blood is dark and fluid.

**4. Disorders of Respiration.**—The respirations tend to be increased in frequency in febrile diseases, but their number bears no definite ratio to the temperature. Some observers affirm that they are in proportion to the pulse; others that the ratio is always disturbed, the respirations being in excess. Neither of these statements will hold good for all cases. With regard to the elimination of carbonic acid, there can be no doubt that this is above the normal on the whole, sometimes greatly, owing to the increased frequency of breathing, although there is a smaller relative proportion in the expired air.

**5. Disturbance of the Nervous System.**—Symptoms referable to the nervous system usually attend the pyrexial state. In the early stage chills or rigors are often experienced: with general muscular pains,

aching or soreness, especially in the back and limbs, and a sense of weariness, exhaustion, languor, and inaptitude for any occupation or effort. Some fevers present special localized pains; while headache and giddiness are very frequently experienced. Restlessness, insomnia, and slight nocturnal delirium are also common symptoms. Alternations of chilliness or even rigors and flushes of heat may occur during the course of a pyrexial attack, and rigors are particularly induced by exposing parts of the surface of the body. Under certain circumstances very serious nervous disturbance may arise, indicated by great general prostration; delirium, either violent or muttering; somnolence, or actual stupor tending to coma; muscular disorders, such as tremors, subsultus tendinum, picking at the bed-clothes, or convulsions; or peculiar symptoms.

6. **General symptoms.**—Owing to the undue waste of tissues, while at the same time but little food is taken to make up for the loss, and even this is assimilated with difficulty, patients suffering from fever lose flesh and weight more or less rapidly, and usually feel debilitated and depressed; they may become exceedingly emaciated and prostrated in a short time, the tissues being very rapidly consumed. Anæmia is also frequently induced.

**COURSE AND TERMINATIONS.**—A typical febrile attack is characterized by three stages, namely, those of *invasion*, *acme*, and *decline* or *defervescence*. These are seen in their fullest development when the paroxysm runs through its course rapidly, as during a fit of ague, or in many cases of “hectic” fever. They are also present in a less marked and more prolonged form in those cases of fever which are continuous for a longer or shorter period, it may be for weeks, and in such cases there are also daily or more frequent paroxysms; but the several stages are not then distinct and well-marked as a rule, and it is often impossible to recognize them. In the stage of *invasion*, also called the *cold* stage, the patient feels cold and chilly, and generally has more or less severe shivering or rigors. The internal temperature rises from the very outset, as proved by clinical and experimental observations, even before the occurrence of the phenomena just mentioned. At the same time the skin may continue to be cold and pale, owing to the contraction of the small vessels, and may even appear shrunken and bluish or dusky. These conditions are especially noticed in the hands and feet, and about the ears, nose, and lips. At this time the pulse is small, hard, and prolonged, from contraction of the arteries. Headache is frequently more marked in this stage than later on, and the patient feels usually much depressed. In the *second* or *hot* stage, the surface becomes hot, and in many cases red; there is a subjective sensation of heat, and the pulse becomes full and bounding. The actual temperature presents much variety. The *third* stage varies considerably in its characters. As applied to a particular paroxysm it is usually called the *sweating* stage, as it is commonly accompanied with more or less free perspiration, while the temperature falls, and the pulse becomes softer and less rapid. Taking the course of a “febrile” disease as a whole, *defervescence*, in cases of recovery, may be brought about by the following modes:—1. **Crisis.**—This mode is especially likely to occur when the invasion has been rapid, and is characterized by a sudden or speedy abatement of the fever, indicated by a rapid fall in temperature, which may become normal in a few hours; at the same time there is generally a considerable increase in the various excretions, evidenced by profuse perspiration,



a free flow of urine containing a large amount of solid ingredients, or watery diarrhœa, which are regarded as *critical discharges*. Occasionally some form of hæmorrhage occurs, such as epistaxis. Crisis is often followed by more or less collapse. 2. **Lysis**.—Here there is a gradual defervescence, the temperature subsiding slowly and regularly for some days, and there being no critical discharges. 3. **Combination of crisis and lysis**.—At first a rapid fall of temperature occurs to a certain point, this being followed by a gradual lowering; or for some days a regular alternation of high and low temperatures is observed. 4. **Irregular**.—Occasionally defervescence is quite irregular in its progress. During convalescence the temperature and amount of excretion often fall below the normal standard. Death frequently terminates cases of febrile diseases, but this is only occasionally traceable to the fever itself, which may cause death by excessively high temperature, blood-poisoning, or asthenia and wasting.

**TYPES OR VARIETIES**.—The symptoms described as belonging to fever are very variously combined, and present a wide range as regards their severity and course. Hence certain types are recognized, which will now be pointed out.

1. **Types depending upon the course and mode of progress of the symptoms**.—*a. Continued*.—This group comprehends all febrile diseases which run a tolerably regular course, the variations in temperature at different periods of the day being within a limited range. It includes the acute specific fevers (typhus, typhoid, small-pox, scarlatina, &c.), and most cases of inflammatory fever. In these complaints the temperature rises more or less rapidly up to a certain point, then remains tolerably stationary for a time, and finally defervescence occurs in one of the ways already mentioned. *b. Remittent*.—Here the fever presents marked remissions alternating with exacerbations, as indicated by the temperature and other symptoms. During the remission the temperature may fall below the normal. This variety is met with chiefly in tropical climates, but remission is also an important characteristic of *hectic* fever. *c. Intermittent*.—This type is characterized by temporary complete cessation of all febrile symptoms, which only come on at certain regular intervals, and run through a definite course, the temperature in the meanwhile being quite normal. The different forms of *ague* afford examples. *d. Relapsing*.—In some forms of fever, after an attack of the *continued* type, defervescence and apparent recovery take place, but this is followed after some days by a *relapse*, which course of events may be repeated more than once.

2. **Types depending upon the severity and combination of symptoms**.—*a. Simple*.—This form is the simplest expression of fever, presenting the characters already described, but in a mild degree. It is well exemplified in ordinary *febricula*.

*b. Inflammatory*.—As the term suggests, this type is usually associated with local acute inflammations, at all events at the outset. It does not however, necessarily accompany every inflammatory disease, nor does the degree of pyrexia always bear a proportion to the intensity and extent of the inflammation. It is more likely to be present, and to be greater in intensity, when some tissues are affected than others; also in young and plethoric persons, and in those of a sanguine temperament. The symptoms are well-marked, though varying much in severity, and they are of a *sthenic* character. Shivering or distinct rigors mark the onset, followed by considerable reaction. The temperature is high, the

skin feeling hot and dry. There are marked pains in the limbs, with much headache. Vascular excitement runs high, as evidenced by a frequent, strong, and full pulse. The blood contains an excess of fibrin-forming materials, and exhibits the "buffy-coat." The digestive organs are much disturbed, there being a thickly-furred but moist tongue, disagreeable breath, marked thirst, total loss of appetite, and constipation. The urine is distinctly febrile. There is much restlessness, with sleeplessness or nocturnal delirium; and occasionally severe nervous symptoms, such as convulsions or delirium, usher in the attack, especially in the case of young children.

*c. Hyperpyrexial.*—Here the temperature is very high, varying from  $106^{\circ}$  or  $107^{\circ}$  to  $115^{\circ}$  or more. It shows a tendency to ascend very rapidly, this being associated with grave symptoms referable to the respiratory and nervous systems. The breathing becomes very hurried and shallow as a rule, the mouth being open, and the nostrils distended during inspiration, which act may be attended with a sucking or sipping sound. The nervous phenomena include marked restlessness, mental confusion and disorder, passing into violent or muttering delirium, and coma. Hyperpyrexia has been most frequently met with in cases of acute rheumatism, sun-stroke, and septicæmia, but it may be observed in other febrile conditions, such as that due to pneumonia or typhoid fever.

*d. Low types.*—Under this group may be included the following:—*i. Asthenic or Adynamic.*—The patient is very weak, and feels much prostrated. The temperature is only slightly raised, and the pulse is feeble and small, though accelerated. In short, febrile reaction is not prominent. At the same time there is not much thirst, and the tongue continues moist. Usually cerebral symptoms are not present, but there may be nocturnal delirium. *ii. Typhoid or Ataxic.* "*The Typhoid State.*"—This presents some important distinctions from the former. There is great prostration. The tongue tends to become small, dry, and covered with a brown or black crust; the lips are dry and cracked; the teeth and gums at the same time being encrusted with sordes; deglutition and speech are consequently more or less interfered with. The heart's action is much impaired, as evidenced by the characters of its impulse and sounds; by the pulse, which becomes more and more rapid, is very weak and compressible, often irregular or intermittent, dicrotic or undulating, and may be at last imperceptible at the wrist; and by the tendency to capillary congestion and stagnation of the circulation in dependent parts, leading to low inflammation, or to bed-sores over parts which are pressed upon. Before death the superficial capillaries generally may dilate, and profuse sweating takes place, which may be offensive, while parts of the surface become dusky and the skin may be cold, especially that of the extremities and over exposed parts. Breathing is usually shallow, and more or less frequent. Diarrhœa may set in, with offensive stools. Nervous symptoms are prominent, especially low muttering delirium, muscular tremblings and twitchings, subsultus tendinum, and stupor ending in coma. The special senses are blunted, and there may be marked deafness. Ultimately the condition culminates in profound exhaustion, the patient lying on his back and sinking towards the bottom of the bed, being indifferent to all around. The typhoid condition is not solely met with in febrile cases, being also observed in uræmia, acute atrophy of the liver, and other conditions. *iii. Malignant.*—In some cases the symptoms are of such an exceedingly



low type, being frequently attended with hæmorrhages and petechiæ, that they may be truly termed malignant. The terms *putrid* or *septic* are sometimes applied to fever under these circumstances. Another form of *malignant* fever is that in which some poison seems to act upon the system so violently as to cause the patient to succumb at once, there being no evident reaction, or any local lesions set up. This is sometimes observed in connection with the exanthemata.

*e. Hæctic.*—Hæctic fever is usually associated with profuse suppuration, but may attend any great drain upon the system. Phthisis frequently presents this variety in its most typical form. It is of a distinctly intermittent or remittent type, there being exacerbations, as a rule once in the twenty-four hours, occasionally twice. The fever sets in very gradually, at first only a slight evening rise in temperature being noticed, with quickening of the pulse; after a while pyrexia becomes more or less constant, but a marked increase takes place towards evening, beginning with chilliness or rigors, followed by much heat of skin, which increases up to or beyond midnight, and is succeeded by profuse sweats, so that the patient's clothes and the bed-clothes may become saturated; in some cases the exacerbation takes place in the afternoon. The subjective feeling of heat is usually very considerable, and the palms of the hands and soles of the feet have often a burning sensation. The appearance of the patient is frequently very characteristic and striking, there being a circumscribed bright red or pink spot on each cheek, well known as the *hæctic flush*. The pulse is very easily excited and rendered quicker; during the paroxysms it may rise to 120 or more. It varies in its characters, but is generally jerky, moderately soft, and compressible. The respirations are also hurried. This fever is attended with considerable and often rapid wasting, while the patient feels much exhausted after each attack, becoming ultimately exceedingly feeble and emaciated. The mental faculties are unaffected until near the close of life, being often, in fact, peculiarly lively and brilliant. The duration of this variety of fever is very variable, but it tends to be prolonged.

*PATHOLOGY.*—The pathology of the febrile state is still unsettled. With regard to its *origin*, it seems so far clear that the condition may be set up by some morbid poison which has gained an entrance into, or been generated within the system; or in connection with some local lesion, especially inflammation. Dr. Burdon Sanderson found in his experiments that by injecting certain fluids—which he terms “pyrogenic”—in very small quantity into the circulation, fever could be excited, and these fluids either contained bacteria or the material out of which bacteria are developed. Some pathologists attribute the setting up of the febrile process solely to the action of morbid organisms, which are supposed to multiply, to increase the metabolic changes, and to give rise to products of decomposition, which exert a paralyzing action on the nervous system. However, it seems to be certain that septic fluids may originate pyrexia, without the actual presence of any visible organisms. There is a view very commonly entertained that the nervous system is immediately concerned in originating fever, especially through the pneumogastric and sympathetic nerves, or through certain nerve-centres. In favour of this view there are many important facts, and it is probable that the febrile process may be initiated directly by certain nerve-centres in some instances; while in others it is primarily due to some morbid poison, which either acts on these centres, or by vaso-motor reflex influence; and in others, again, to reflex irritation from some



local lesion, such as inflammation. Some pathologists do not agree with the notion of any direct implication of the nervous system in fever, but maintain that the cause which originates this condition acts immediately upon the blood and living tissues, giving rise to increased oxidation—in short, that it is throughout a disorder of protoplasm.

The explanation of the *phenomena* attending the febrile process must be guided by a consideration of the chief facts relating to the bodily temperature in health, but even these are not yet satisfactorily determined. They have to deal with—first, the *production*; and, secondly the *regulation* of the animal heat. There can be no doubt that the main source of the animal heat consists in the chemical changes which take place in the food and tissues, during which they undergo combustion or oxidation, and give out their latent heat, which is equal to that of the substances taken into the system in the form of nutriment. These changes are most active in muscle, nervous tissues, and the abdominal viscera. During the performance of the various functions of the body, some of the heat thus evolved becomes reabsorbed and again latent, to be subsequently set free, and these transmutations are constantly going on in the system. A slight amount of heat is generated by the friction of the blood against the vessels, and of the muscular fibres against each other; and it has even been supposed that this occurs during the passage of nerve-currents; but these are merely instances of the giving out of heat which has been latent, for the body has no power of creating heat within itself. Beale believes that the conversion of non-living into living material is the cause of the production of animal heat.

With regard to the regulation of the bodily heat, this depends mainly upon the skin, its distribution being effected by means of the circulation of the blood, which renders the temperature tolerably uniform throughout the system. The surface of the body is constantly losing heat by conduction when the temperature of the air is low, but especially by the evaporation which is perpetually taking place from the skin. The condition of the superficial circulation has thus an important influence in regulating the temperature, for when the vessels of the skin are contracted, less heat will be lost, at the same time more being produced in the deeper parts; while if the cutaneous vessels are dilated, more heat will be given off.

The influence of the *nervous system* upon the temperature demands special notice. It may be regarded as an established fact, founded upon physiological and experimental investigations, as well as upon clinical observations, that the nervous system does materially affect the bodily heat. Hyperpyrexia may be induced experimentally by section of the spinal cord in the cervical region, and in other ways; and the same condition is sometimes observed in connection with injuries to, and diseases of, the brain or cord. Indeed, remarkably high temperatures have been recorded in some cases, but there can be no doubt that these were not real, and that the patients practised deception. The nervous system influences heat-production, as well as heat-regulation. With regard to the modes in which this system modifies the temperature, in the first place it does so through the vaso-motor apparatus; but it is also believed that it directly affects tissue-change, and consequently heat-production. Indeed, some physiologists go so far as to maintain that there are special thermal nerve-centres and nerves, but at present there is no adequate proof of this. It appears that the nervous system exerts a check or restraining force upon nutritive and oxidation changes,

but in what way is not known. Dr. Broadbent has advanced the theory that it does so through tension maintained in the nerve-centres, the tension there generated in the cells being sustained to their peripheral terminations, where they are merged into, and blended with the other structures.

Coming now to the explanation of the phenomena of pyrexia, there can be no doubt, in accordance with the views of the most reliable authorities, that they are mainly accounted for by *increased heat-production*, and that the condition is attended with excessive tissue-metamorphosis and destruction. The temperature of a given quantity of water will be raised, when a febrile patient is immersed in it, more rapidly and to a higher degree than if he were in a healthy state. In febrile conditions the tissues become quickly destroyed, being changed into various substances of lower chemical composition. This destruction involves the albuminous or nitrogenized tissues, as well as the fat, and the muscles, both voluntary and involuntary, are specially affected, these structures wasting rapidly, their fibres presenting an appearance of granular degeneration under the microscope. The nerve-centres, ganglia, and nerves undergo a similar change, though to a much less degree; the bones also become lighter; and the red blood-corpuscles are diminished in number. The destruction of the tissues and blood is evidenced by the wasting which occurs, and by the presence of excess of potash-salts and colouring matter in the urine; while soda-salts, which are derived from the food, become deficient. It is found that the glandular organs do not become smaller, being, indeed, often enlarged from congestion, especially in young and healthy persons. This is particularly observed in the spleen, lymphatic glands, and liver, the cells of these organs also becoming enlarged and granular.

The substances into which the tissues are transformed are, as was proved by the late Dr. Parkes, chiefly those produced in health, only formed in excess, such as urea, uric acid, and carbonic acid. Intermediate products of decomposition may, however, be generated, some of which are probably quite foreign to the body in a state of health. As to the place where the change occurs there is no certainty. Some pathologists consider that it takes place in the tissues themselves; others believe that the albuminous elements break down into a circulating albumen, which in the blood becomes converted into materials of lower grade, such as urea.

It has been found that as a rule the rise of temperature in febrile cases is in proportion to the rapidity and extent of the changes just alluded to, especially as evidenced by the amount of the products of tissue-metamorphosis eliminated in the various excretions. In most cases excessive excretory elimination is observed, particularly by the urine, the quantity of urea discharged often bearing a close relation to the increase in temperature; but this is not always the case, so that the one cannot be made a measure of the other. In some instances there is little or no increased elimination during the progress of fever; but in those cases where the quantity of urinary solids discharged daily is not greater than in health, it is really much more than would have been passed by a healthy person on the same diet. Moreover, there may be retention in the system, or deficient elimination of the products of metamorphosis, owing partly to their great abundance, or to their transformation being incomplete, substances being thus generated which the kidneys cannot get rid of; partly to some condition of the excretory

organs which interferes with the due performance of their functions. Sometimes the urine temporarily ceases to be febrile in its characters, and this is followed by abundant discharge of waste products in this excretion. It is in cases where free elimination has not taken place that critical discharges are most likely to occur at the termination of the febrile process; whilst, as the result of the accumulation of the substances generated within the system, serious consequences are liable to ensue.

The views of Dr. Beale on this subject deserve notice. He believes that the bioplasm of the blood, blood-vessels, and tissues is greatly increased in pyrexia, and that this is the cause of the excessive heat. He further states that there is insufficient oxidation of tissues, as a result of which the blood becomes loaded with noxious materials which the excretory organs cannot remove, and this condition of the blood is favourable to the growth of bioplasm.

Some pathologists maintain that the rise of temperature in fever is the result of *retention* or *diminished loss of heat* from the surface of the body, owing to contraction of the vessels, or to a relatively diminished heat-discharge in proportion to the heat-production. This partly accounts for the internal heat in the early stage of fever, but later on, even when perspiration is profuse, as in rheumatic fever, or when sweating has been induced by jaborandi before a fit of ague, the temperature nevertheless goes up. Still, when the perspiration is diminished or checked in febrile conditions, this does act as a subsidiary cause in increasing the body-heat, and elevating the temperature.

The influence of the condition of the vessels, and of the nervous system, upon the bodily temperature must be borne in mind, as no doubt these affect the febrile condition in no small degree, as in health, in the ways already indicated.

There is no adequate explanation of the *retention of water in the system*, which occurs in pyrexia out of proportion to the amount which is consumed. It seems, however, probable that more really passes off by the skin and respiratory organs than is supposed, and than escapes in health; and that much of the loss of weight in fever is due to this cause. It was suggested by Dr. Parkes that some material forms in the blood, such as gelatine, which is powerfully hygrometric, having a strong affinity for water.

The causes of the disorders affecting the different functions of the body ordinarily observed in fever are sufficiently obvious. The increased activity of the respiratory and circulatory functions are in themselves signs of augmented tissue-change. The amount of carbonic acid expired is increased immediately, and more so at first than when the fever has reached its height, because not only is more of the gas produced, but that which is dissolved in the blood is gradually expelled by the rising temperature, while the circulation being active, the blood is more rapidly exposed to the air in the lungs. With respect to the circulatory system, in the early stage the heart is excited, but as the febrile process advances this organ becomes more or less impaired in its action, because its muscular tissue undergoes degeneration, it is supplied with impure blood, and its nervous stimulus is impaired. As a consequence, the alterations in the cardiac impulse and sounds, and in the characters of the pulse are observed; as well as the tendency to hypostatic congestions, these being also directly contributed to probably by the abnormal condition of the vessels, tissues, and blood, and,



according to Beale, by the excessive growth of bioplasm, which tends to block up the capillaries.

It remains to consider the meaning of some of the graver events which are liable to occur in connection with the febrile state. The "typhoid condition," and the low nervous symptoms attending it, are almost certainly due to the accumulation within the system of the products of tissue-destruction, owing to elimination being checked entirely, or not being equal to the requirements of the case. In short there is a kind of uræmia set up, and the patient is poisoned by these effete materials, which circulate through the nerve-centres and other organs and tissues. Low nervous symptoms have also been attributed to the direct action of some special fever-poison upon the nerve-centres; to plugging of the minute vessels of the grey matter with white corpuscles or septic emboli; or to excessive heat of the blood. It is not yet determined what degree of temperature is compatible with life in the human system, but above  $107^{\circ}$  the excessive heat itself becomes very dangerous, producing grave symptoms, and tending to cause a fatal result. It affects the heart, which after death has been found with the ventricles contracted and empty, and the auricles full of blood; but it also influences all the tissues, and the protoplasm of the body. The immediate cause of hyperpyrexia must probably be usually referred either to the nervous system, or to the injurious action of septic materials present in the body.

In some low forms of fever acute granular or fatty degeneration of the chief organs is liable to supervene, and this is attended with grave symptoms. Moreover, secondary inflammations not uncommonly occur in febrile cases, such as pneumonia, and these must be attributed either to stagnation of blood following congestion from impaired circulation; to the action of retained deleterious waste-products; or to emboli, which may be of a septic nature.

PROGNOSIS.—The prognosis in febrile cases must necessarily depend greatly on the cause of the fever, and will therefore vary with each particular disease. So far as the pyrexia itself is concerned, however, there are certain conditions which always influence the gravity of the prognosis, namely:—1. *Its intensity*.—The higher the temperature the more dangerous is the case, and the prognosis becomes very serious when it reaches above  $106^{\circ}$  or  $107^{\circ}$ . Under appropriate treatment, however, a considerable proportion of patients have recovered, even after the temperature has risen to a much higher point than this. 2. *Its type*.—All low forms of fever are grave, and any tendency towards typhoid or adynamic symptoms, especially if the nervous system is much affected, should be looked upon with anxiety. 3. *Defective elimination*.—This is an unfavourable sign, particularly if associated with a very high temperature. 4. *The previous condition and health of the patient*.—Young, robust, and plethoric persons are often more severely affected than those in opposite conditions. Some diseases, such as gout, increase the danger of febrile disorders considerably; while the presence of organic disease, especially of the kidneys or heart, renders them exceedingly grave.

TREATMENT.—The management of cases attended with fever presents much variety. Frequently it is a very simple matter, needing little or no active treatment, but merely rest and attention to diet; in many cases, however, it is a question of much difficulty, and requires the most careful and constant attention. There are two rather prevalent errors

which need to be guarded against. First, it must not be imagined that treatment is of no avail in febrile diseases, and that the physician has nothing whatever to do. By *judicious* interference it is possible to avert death, to relieve symptoms, and even to hasten recovery in some cases. On the other hand, *over-active* and *meddlesome* treatment is most injurious, and decidedly to be deprecated, especially in the case of those specific fevers which must of necessity pass through a certain definite course. The practice of endeavouring to *cut short* fevers has unquestionably often done much harm.

There are certain general indications for the treatment of the *pyrexial condition* and its accompanying symptoms, which will now be briefly considered, as well as the means by which they are to be carried out.

1. The first indication is to **diminish the temperature**, if this tends to be excessive. One of the most powerful means available for this purpose is the *external application of cold*. This acts partly by increasing the elimination of heat by the skin, but also probably by producing some marked effect upon the nervous system, and checking destruction of tissues, or, as Dr. Beale thinks, by diminishing the growth of bioplasm. The modes of applying cold are various, namely, by sponging the surface of the body with water, either tepid or cold; by employing a water-bed containing cold water; by cold affusion or douching, which may be practised whilst the patient is in a warm bath; by wet-packing in a sheet; by applying cold compresses over the chest and abdomen; by the use of cold or tepid baths; by the application of ice-bags to different parts of the body; by an ice-cap applied to the head, or a cap made of tubing, through which cold water runs in a continuous stream; by injecting iced water into the rectum; or by placing the patient in a warm or tepid bath, the temperature of which is then gradually reduced by the removal of the warm, and the addition of cold water or even ice, the latter being also in some cases applied to the head, spine, chest, or abdomen at the same time. After having been kept in the bath for a varying time, according to the circumstances of the case, the patient is dried and removed to bed, and it may then be necessary to apply hot bottles to the feet. It may be requisite to repeat the bath, even several times, and to apply ice in the intervals.

Some of the methods mentioned are not only useful in reducing temperature, and making the patient more comfortable, but they likewise diminish the frequency of the pulse; give marked relief to low nervous symptoms; and may have an influence upon the development of certain exanthematous eruptions, either in the way of encouraging them to come out, or of limiting their amount and improving their quality. A regular hydropathic treatment of all fevers is now often practised, especially on the continent. But in my opinion this plan of treatment ought not to be followed as a routine method, and there are grave objections against the more severe methods by which it is carried out. Sponging of the skin, the use of a cold water-bed, or the application of compresses over the abdomen and chest, are modes applicable to many ordinary cases, and might be practised with advantage much more frequently than is the rule at present, for they are often decidedly beneficial, and afford much relief, without being at all dangerous if proper care be exercised. When baths are used to control temperature which is not serious in itself, but from its duration, as in typhoid fever, it is unnecessary to employ them below 70° or 65°. The cases, however, in which the use of external cold is so eminently serviceable, are those in which the temperature shows



a tendency to ascend rapidly, or in which it has become and remains very high. My own experience corroborates that of Dr. Wilson Fox and others as to the remarkable benefit which may be derived from the employment of cold in the manner last described, when there is hyperpyrexia. Undoubtedly in similar cases not only is it advisable to adopt such a plan of treatment, but this is the only method which seems to offer any chance of recovery. Of course it must always be conducted under competent and strict supervision, and its effects carefully watched.

Venesection has been employed with the view of subduing fever, but there is positive evidence that it acts most injuriously, and therefore should never be practised merely for this object, although in the inflammatory variety it may be required.

Among *medicinal agents*, aconite, digitalis, veratrum viride, and tartar emetic are, in appropriate cases, most useful in fevers. They reduce the temperature in some degree, but have also a striking effect on the pulse, diminishing its frequency. Some of these drugs have a further action on the excretory organs. A favourite mode of treatment in certain cases is to give tincture of aconite in one or two minim doses every half-hour; while some recommend it every five minutes, until twenty or thirty minims has been taken. Quinine is regarded as one of the most powerful *antipyretics*, and is much used for the purpose of lowering temperature, or checking its ascent. It is well known that this drug has a powerful influence upon ague, and when given in considerable doses—v-xx grains or more, and repeated at variable intervals according to circumstances, it has some power of diminishing excessive heat. Its administration may be combined with the application of cold. Salicylic acid and salicine are valuable, especially in certain forms of fever; and various salicylates are also used. Kairin and chinoline are agents recently introduced. Sulphurous acid, in drachm doses every two, three, or four hours, has also been recommended. The employment of alcohol will be referred to presently.

2. Another important indication is to **watch the excretions** and observe whether proper elimination is taking place. Some authorities advocate energetic eliminatory treatment in fever, by which they propose to get rid of any specific morbid poison, as well as of the products resulting from destruction of tissues. Such treatment, however, is not advisable as a general rule, except in so far as it may be necessary to keep the bowels freely open, and to give some mild *diaphoretics* and *diuretics*; but it is requisite in severe cases to examine the excretions, especially the urine, at frequent intervals, and thus to ascertain whether the materials formed are being properly got rid of, and, if such is not the case, to adopt measures calculated to aid their removal. Should symptoms arise indicating that the system is being poisoned by the accumulation of the products of tissue-change in the blood, energetic eliminatory treatment is decidedly called for. This consists in measures which promote the free action of the skin, bowels, and kidneys. *Diaphoretics* and *diuretics* are very useful, such as saline mixtures, containing citrate of potash or liquor ammoniæ acetatis; along with the free use of diluent drinks. Jaborandi may be serviceable in some cases, on account of its diaphoretic action. The employment of baths also increases the skin-action. In severe cases, should the urine be deficient, it is desirable to endeavour to excite the kidneys into activity, by applying hot fomentations, linseed-meal poultices, or sinapisms over the loins, or by means of dry-cupping. *Purgatives* must be employed with caution, as



they are likely to weaken the patient, but they are often required, and the *saline aperients* are the most efficient in these cases. When diarrhœa is present, some advocate that this symptom should be permitted to continue, or even be encouraged by the aid of medicines, as it is a natural mode of elimination of a poison. It certainly is not always desirable to check diarrhœa, but should it be excessive, or should the patient be evidently becoming weakened on account of it, it is, in my opinion, decidedly advisable to restrain the discharge by appropriate remedies.

3. One of the most necessary and difficult parts of the treatment in many cases of fever consists in the **proper administration of diet**, including *food* and *alcoholic stimulants*. When acute pyrexia is present, it may be stated as a general rule that solid food should not be given, and that the diet should be of a liquid nature. In many cases low diet is indicated, at first at any rate, but often abundant nutriment is subsequently needed, or sometimes even from the outset. Then the food must be nutritious, and at the same time capable of easy assimilation. Milk is a most valuable article of diet, as well as good beef-tea, mutton-broth, chicken-broth, and eggs. Meat-extracts and juices are often of much service. A most important matter which often needs special attention is to give the food at *frequent and regular intervals, in definite and moderate quantities*, and the patient should not be allowed to sleep for too long a time, and thus be deprived of the requisite nutriment. It is quite impossible to lay down any definite and rigid rules as to diet, but each case must be treated on its own merits. In the low forms of fever large quantities of nutritious aliments are called for.

With regard to alcoholic stimulants, these are by no means always required, and their indiscriminate use may do a great deal of harm, but in a large number of cases they are of the utmost value, though much experience is necessary in order to determine the particular kind and quantity of stimulant to be administered under different circumstances. Therefore young practitioners should be extremely cautious as to how they employ these remedies, and should watch their effects very closely. Wine or brandy generally answers best, of which it may be necessary to give very large quantities, and it is astonishing how much may be taken in certain cases without producing the ordinary intoxicating effects of alcohol. It is most important that stimulants should be administered at *regular intervals* and in *definite doses*. The essential value of alcohol consists, not in its making up for food, which must be given at the same time, but in that it maintains the action of the heart while the system is struggling against the effects of the fever. Hence the chief indication for its use is to be found in the condition of this organ, as evidenced by its impulse and sounds; by the state of the pulse, as regards its frequency, force, and amount of tone; and by the condition of the capillary circulation. Other organs, however, must not be overlooked, and in judging of the effects of the administration of stimulants, attention must likewise be paid to the tongue, skin, respiratory organs, and nervous system. Their good effects are seen in the tongue becoming moist and less furred, the skin perspiring, the temperature reduced, the number of respirations diminished, and the nervous system calmed. If the tongue becomes dry and baked, the skin burning and non-perspiring, the respirations hurried, and the nervous system excited, alcohol is doing harm. It is useful to smell the breath from time to time, and to stop the administration of stimulants should this give evidence that the system is becoming saturated with alcohol. With regard to its influence

on temperature, alcohol has been proved only to lower this directly when given in large quantities, and then only to a comparatively slight degree. My own experience has convinced me that in hyperpyrexial cases it is requisite to be exceedingly careful in the administration of alcoholic stimulants, and that they ought not to be poured into the system in a reckless manner, with the view of lowering the temperature.

It is in the later stages of fever that alcohol is most useful, and especially when it tends towards an asthenic or adynamic type. No case, however, should be allowed to sink into a low condition for want of stimulants, as it may then be very difficult or even impossible to revive the patient. If there is any probability of this event taking place, they should be employed from the first. At the same time it is very important in these cases to watch constantly and thoroughly, and to observe at frequent intervals the effects of the administration of stimulants, lest they should be pushed too far. They must be given with particular caution if the urine is very deficient or contains albumen. As to the quantity required, this will vary much in different cases. Usually from a teaspoonful to a table-spoonful of brandy will be needed, given at intervals of from three hours to half an hour according to circumstances. Old people require a considerable quantity as a rule, and young children bear stimulants well. Wine or brandy may be conveniently given beaten up with eggs, the brandy-and-egg mixture (B.P.) being an excellent preparation; or along with beef-tea.

Dr. Beale has arrived at the following conclusions respecting the good effects of alcohol in cases of fever and inflammation:—1. By its *direct* action on the nerves of the stomach it immediately stimulates the heart's action, and thus promotes the capillary circulation. 2. After absorption into the blood it alters the consistence and chemical properties of fluids and solids, and cuts short the life of rapidly growing bioplasm, or causes it to live more slowly. It reduces the permeating tendency of blood-serum; renders the walls of the vessels less permeable to fluids; checks the disintegration of blood-corpuscles; interferes with or modifies chemical changes; and has a direct action upon the particles of naked and living bioplasm.

4. Attention to **general management** and to **hygienic conditions** is another point of much importance in the treatment of febrile diseases. This matter will be again specially considered, but in the meantime it may be stated that two of the most essential requisites are *free ventilation*, so as to ensure that the patient obtains plenty of fresh air, and that the vitiated air is removed; and *cleanliness*. Rest of the body and mental quietude are also most important elements in the treatment of many febrile cases, and whenever there is pyrexia, it is a good plan to keep the patient in bed. In serious cases he should not be disturbed by the presence of friends or others who are not required in the sick-room. Of course *competent nursing* is often indispensable, and the nurse should wear dresses which do not rustle, and to which, in the case of contagious fevers, the contagious poison will not readily adhere.

5. Many **symptoms** arise in the course of febrile diseases which require special treatment, and in order to avoid repetition it will be advantageous to consider here the more common of these in some detail.

a. Symptoms referable to the *digestive organs* are often troublesome. Thirst is almost always complained of more or less. For its relief the following drinks will be found agreeable in different cases, namely, simple iced water; barley-water; toast-and-water; iced milk with soda-water;

solution of chlorate of potash (3 j to Oj), which may be flavoured according to taste; some acid drink, such as lemonade made with the juice of lemons, or a drink composed of 3 j of dilute hydrochloric acid with Oj of water or barley-water, and a little honey or sugar; tamarind-water; or iced champagne with seltzer-water or soda-water in small quantities, if stimulants are indicated. The frequent sucking of small fragments of ice will generally be found of much service. Patients may also in most cases be allowed to suck juicy fruits in moderation, such as grapes or oranges. It is often necessary to give instructions to the nurse to see that the patient's mouth is cleansed from time to time; and a mouth-wash of weak solution of Condyl's fluid, or some other simple antiseptic, may be used.

Vomiting is not uncommonly a symptom needing attention. Some practitioners adopt the plan of giving an emetic at the commencement of any febrile attack, but the advantages of this routine method of treatment are by no means obvious. If, however, vomiting or an inclination to vomit seems to be due to some source of irritation in the stomach, an emetic is useful, such as a full dose of ipecacuanha wine or sulphate of zinc, followed by plenty of lukewarm water. In order to check vomiting special attention must be paid to the diet, and it will be well to give no food at all, if this symptom is troublesome, except very small quantities of iced milk with lime-water or soda-water at frequent intervals; or a tea-spoonful of brandy with the same quantity of strong beef-tea or beef-juice. Iced champagne with seltzer-water in small doses is also very serviceable, as well as the sucking of ice. With regard to medicinal remedies, the most efficacious in febrile conditions are effervescent draughts with hydrocyanic acid— $\mathfrak{m}$  ij-iv; or the latter with a little mucilage or bismuth. If opium or morphia is admissible, it is useful in some cases to add tincture of opium or solution of morphia— $\mathfrak{m}$  iij-v—to each effervescent draught. When sickness resists the ordinary remedies, minute doses of strychnia sometimes have a remarkable effect in checking it. Local applications over the epigastrium are also serviceable in obstinate cases, namely, linseed-meal poultices, sinapisms, flying blisters, or cold by means of the ice-bag. Care must be taken that the tendency to vomiting is not kept up by anything wrong in the sick-room, such as bad smells or deficient ventilation.

The bowels very often require to be regulated. In most cases constipation is the symptom calling for treatment. The best aperients in febrile conditions are usually the ordinary black-draught; sulphate and carbonate of magnesia with peppermint-water; Seidlitz powders; castor-oil; or, for children, rhubarb and magnesia. In some cases more powerful purgatives are required. Diarrhoea sometimes needs to be checked, but it must be borne in mind that this may be a mode of elimination, and therefore it should not be heedlessly interfered with. It may be generally stopped, if necessary, by the ordinary remedies in different combinations, such as opium in the form of pill, tincture, or enema; carbonate or nitrate of bismuth; chalk-mixture; tincture of catechu; mineral acids; or Dover's or compound kino powder.

*b. Head-symptoms* are among the most common needing attention in acute febrile diseases. If headache is severe or persistent, it is desirable to apply some cold lotion or an ice-bag to the head or nape of the neck, or to employ cold or warm affusion, the latter answering best for old and feeble patients. It is often advisable to cut the hair very short, or it may be needful even to shave the scalp. Dry-cupping over the nape of



the neck is also serviceable in some cases; or, if the patient is young and robust, the application of two or three leeches over the temples might be advantageous. Similar treatment is indicated should there be active or violent delirium, this symptom being often much relieved by freely douching the head with water, either cold or warm. Small blisters to the temples or nape of the neck are also beneficial in some cases. Low delirium generally calls for the administration of stimulants.

Sleeplessness is a very important symptom to treat, and demands great attention. Patients often suffer seriously from want of sleep, and I believe very injurious consequences sometimes result in fevers from a needless dread of giving narcotics. Opium and morphia are the chief remedies of this class, and it is best to give either of them in the liquid form in a tolerably full dose. If there is much throbbing headache or active delirium, it is recommended to combine the opium with a small dose of tartar emetic or with ipecacuanha, Dover's powder forming an excellent compound in which the latter is contained, and one which I have frequently found to act very beneficially. Should there be a tendency to low delirium opium may be given along with stimulants. This drug is contra-indicated if the lungs are involved to any considerable extent, and the respiratory functions much interfered with; if the kidneys are affected; if there is any tendency to stupor; or if the pupils are much contracted. Other useful medicines for procuring sleep, which some practitioners employ in preference, separately or some of them in combination, are hydrate of chloral in doses of 15 to 30 grains or more; bromide of potassium or ammonium; tincture of hyoscyamus, from 50 to 80 minims; tincture of belladonna; chloroform; and nepenthe. General restlessness and irritability is frequently much relieved by sponging the skin, or in some cases it may be desirable to put the patient into a warm bath. If the sense of hearing is unpleasantly acute, it is useful to put a little cotton-wool into the ears.

Any tendency to stupor or coma must be combated by freely douching the head; applying sinapisms or a blister to the nape of the neck, as well as sinapisms or turpentine fomentations to the legs and chest; and administering diffusible stimulants internally, with strong coffee. In extreme cases much benefit has followed the application of a blister to the shaven scalp. It must be remembered that any of the nervous symptoms above alluded to may be dependent upon retention of deleterious matters within the system, and therefore care must be taken that the excretory organs are acting properly, while it may be requisite to promote the removal of retained morbid products, by exciting a free action of these organs.

c. Measures directed against *adynamic* or *typhoid* symptoms are very often called for in the course of febrile diseases. As already stated, alcoholic stimulants and abundance of nourishing food are demanded under such circumstances, and it is extremely important that these should be administered at frequent intervals, strict directions being given that the patient must not be allowed to sleep for too long a time, and thus be deprived of the necessary support. In addition to these remedies, certain *tonic* and *stimulant* medicines are very useful in adynamic cases, namely, ammonia with decoction of bark, quinine in full doses, mineral acids, sulphuric or chloric ether, chloroform, camphor, and musk. At the same time sinapisms may be applied over different parts of the body. When there is great depression, strong coffee and phos-

phorus have been found serviceable; or subcutaneous injection of ether may be resorted to. If patients are in such a condition that they cannot swallow, recourse must be had to enemata, by means of which food and stimulants, as well as medicines, may be administered. It is important in these cases to look to the bladder, and to take care that it is properly emptied, the catheter being employed if required.

6. In acute fevers it is requisite to watch for and take every precaution against **local complications**, and to treat them as they arise. It is especially needful to look to the state of the lungs, as these organs are very liable to become the seat of hypostatic congestion or inflammation. Position will have some influence in preventing this untoward event, the patient not being allowed to lie with the head too low, while change of posture from time to time is encouraged. It is further advisable to promote cough and expectoration occasionally, so as to avoid any accumulation of mucus in the bronchial tubes; and also to instruct the patient to breathe deeply from time to time, so that the bases of the lungs may be duly expanded. Inflammatory affections arising in the course of fevers do not contra-indicate the use of stimulants, provided these remedies are otherwise called for. Indeed, not uncommonly they indicate a necessity for increasing their amount. Particular attention should be directed to the prevention of bed-sores, as they are very prone to occur in low febrile cases. If a patient is likely to be confined to bed for any length of time a firm mattress should be provided, a feather or flock bed being decidedly objectionable. The parts upon which the patient lies must be frequently examined, and kept dry and clean; continued pressure must be obviated by frequent changes of position, and the use of proper pads or air-cushions; and should there be the least sign of irritation, a water-pillow or bed ought to be provided. Various applications are used to prevent bed-sores, or to treat slight abrasions, such as spirits of wine, eau de Cologne, or some ordinary spirit, either strong or diluted with water; oxide of zinc powder; simple ointment or zinc ointment; and soap plaster. The treatment of these lesions, when they do occur, is described in surgical works.

7. Great care is usually required during **convalescence** from fevers, as regards diet, hygiene, and medicinal treatment. Tonics and remedies for promoting digestion are often very beneficial. Undue muscular exertion and fatigue must be avoided for some time. Change of air is often attended with marked benefit, and hastens convalescence considerably. Sequelæ must be watched for, and treated if they should occur.

---

## CHAPTER II.

### ON CONTAGION AND EPIDEMICS.

#### 1. CONTAGION.

THIS is the most convenient place for entering upon a brief discussion of the main facts and theories associated with the important subject of contagion, because, though it has to do with some other maladies, its chief interest is connected with the acute specific fevers. Using the

word in its general sense, a *contagious disease* may be defined as "a disease which is capable of being transmitted from one animal to another, either of the same or some other species." The agent by which it is so transmitted is named *the contagium* or *contagion*.

**1. Origin and source of contagion; conditions under which it exists; and modes of propagation.**—It is needless to enter into any discussion as to the primary origin of the various contagious poisons, and as to whether they are capable of being developed *de novo* at the present time. Perhaps a limited number of the infectious diseases may be thus generated, but the majority of these affections which have to be dealt with are always communicated from one human being to another; while a small number are transmitted from some other animal to man, such as vaccinia, hydrophobia, glanders, and malignant pustule. In some cases the latter class can be re-transmitted to the same or another animal, usually in a modified form. It has been suggested that possibly the contagious poison may occasionally be derived from plants.

The contagium exists under different forms, and is given off in different ways. There is one distinct class of affections in which it is associated with obvious parasitic animals or plants, or their germs, for instance, scabies and the various forms of tinea. In other cases the contagium is believed to be connected with organized cells, such as those of cancer. Not unfrequently the poison is conveyed by means of pus or other morbid products, derived from an inflamed or ulcerated surface, or from pustules, of which gonorrhœa, syphilis, small-pox, glanders, and puerperal peritonitis afford illustrations. It may exist also in the contents of papules or vesicles; or in the substance of the dried scab which succeeds a pustule, as in the case of small-pox. Many contagious poisons have no palpable form, so far as has been at present ascertained, but are given off in the various exhalations and excretions of the body, especially in those emanating from the lungs and skin. Some are supposed only to contaminate the breath, such as that of whooping-cough; others seem to be present in all the exhalations, as well as in the various secretions, for example, that of small-pox. The poison of scarlatina is very abundant in connection with the epithelium of the skin which is shed in this disease, and this may be retained in a dormant state in clothing for an indefinite time. Cholera and typhoid fever are probably communicable only through the fæces, which are capable of imparting their specific characters to any excrement with which they are mixed, their contagion coming from the intestines. Hydrophobia is an example of a contagious malady only transmissible through a special secretion, namely, the saliva. It is said that malignant pustule may arise from eating the flesh of an animal affected with this disease. The blood may be the channel by which a contagious poison is directly conveyed. It is important to remember also that the emanations from the body of a person who has died of an infectious disease may give rise to the same malady in another individual, and this power of imparting contagion may continue for some time after death.

The next point to consider is, how the contagium may be transmitted from one animal or individual to another, and how it gains access into the system? In some cases it is necessary to bring the material in which the poison resides into close and intimate relationship with the minute vessels of the tissues, so that immediate absorption may take



place. This is artificially carried out by *inoculation*, that is, by puncturing the skin or otherwise destroying its superficial portion, and introducing the source of contagion into the subcutaneous areolar tissue, or even directly into the vessels. It may be absorbed, however, through any abrasion or ulcer, situated either on the skin or a mucous surface. Hydrophobia, syphilis, and vaccinia are examples of diseases which can only be propagated in this way.

Another mode of communication is by *direct contact*, without any breach of continuity of the surface. This has been specially termed transmission by *contagion*, but it is not advisable thus to limit the use of this word. It is particularly through mucous membranes that contagious poisons enter the system in this way, as is well illustrated by gonorrhœa and purulent ophthalmia. Parasitic diseases are propagated by contact, such as scabies; and it is stated that malignant pustule may arise in consequence of the infected matter soaking through the skin.

Many contagious affections can be conveyed from one individual to another without the necessity of any immediate contact between them. The contagium is given off into the surrounding atmosphere, and thus passes to the unaffected person, being subsequently inhaled or swallowed, or absorbed by the skin. To this mode of communication many would limit the application of the word *infection*. The poison may also become mingled with the food, such as milk, and be thus taken into the system. Further, the contagious elements often become attached to what are termed *fomites*, including articles of clothing, especially those of a woollen, silken, or cotton fabric; bedding and bed-clothes; hair and various other articles; and they are thus propagated. They may retain their activity under these circumstances for long periods, in this way being the means of originating their several diseases after considerable intervals, though they tend to become weakened by lapse of time. Persons passing between the sick and healthy are liable to carry an infectious disease to the latter. A contagious poison may also be conveyed by clothes sent to laundries, or sent home from an infected school; as well as by letters, cabs, and numerous other agencies. Moreover, it frequently becomes attached to furniture, or to the floor and walls of rooms, and thus an infectious disease may break out after an indefinite interval, if an apartment which has been occupied by a patient suffering from the complaint has not been properly disinfected.

Flies and other insects are believed to be the means of disseminating contagious diseases in some instances, by alighting first on diseased, and then on healthy individuals; or they may probably convey contagious poisons directly from excreta. Earthworms are said to have been instrumental in conveying the contagion of splenic fever to animals from those which have died of the disease. Water is a most dangerous medium for conveying the poison of cholera and typhoid fever, in consequence of the excreta of patients suffering from these complaints finding their way into, and becoming mixed with water used for drinking purposes.

Some contagious maladies may be communicated by all the modes just considered, others only through one of them, as will be pointed out when treating of these affections individually. The opinion has been expressed that they are all inoculable if the necessary conditions could be ascertained, but there is no proof of this statement.

After it has reached an individual from the atmosphere a contagious poison attaches itself to the skin, as well as to the mucous membrane of the mouth, nose, throat, respiratory passages, alimentary canal, and

other parts, and it may even find its way into the air-cells of the lungs. It then passes through the more delicate membranes, becomes imbedded in the thick mucous tissue, through which it gradually finds its way, or penetrates the little chinks between the epithelium cells of the skin. Thus it reaches the minute capillaries and lymphatics, into which it enters, and is subsequently carried through the system. Absorption is promoted by a swollen, soft, and moist state of the skin; or by a weak, distended condition of the capillaries; while it is especially facilitated by the presence of wounds or abrasions, and by such a condition as that of the interior of the uterus after delivery (Beale).

**2. Degrees of contagiousness of different diseases, and modifying influences.**—There are marked differences with regard to the facility and certainty of the transmission of contagious diseases. Some, such as small-pox, measles, and scarlet fever, are very readily communicated; others, such as typhoid fever, are uncertain. The contagium of influenza is extremely diffusible; that of typhus fever seems to concentrate about the patient, and is destroyed by dilution with air. Many modifying influences are also at work. The probability of a contagious disease being communicated is, as a rule, in proportion to the quantity and strength of the poison which reaches the system, but it must be remembered that in many instances a very minute quantity is sufficient. The virulence of the contagium also varies often at different periods, either in the course of an infectious malady or of an epidemic. The mode of application has considerable influence, inoculation being obviously the most certain. It is believed that a contagium becomes weakened by passing through several individuals. If any fluid containing a contagious poison is much diluted, there is less chance of successful inoculation. From experiments made with the lymph of small-pox, vaccinia, sheep-fever, and glanders, it would appear that by allowing liquids which contain contagious particles to stand for some time, these particles subside to the bottom, so that the upper layer of fluid may be inoculated without producing any effect. By thorough filtration they may also be separated. This proves that the liquid portion does not contain the contagium, but that the latter resides in the deposited particles.

Much will depend frequently on the temperament, constitution, state of health, and previous habits of the individual to whom the contagium is applied, as to whether the disease will be transmitted or not. A previous attack of a contagious malady usually protects against a second, but not invariably, and it may appear a third time or even more frequently in exceptional instances; when a second attack does occur, it is generally of a mild character. Syphilis cannot be produced even by inoculation after this has been done a certain number of times. It is exceptional, but not impossible, for two infectious diseases to be present in the same person at the same time, and should such an event occur, they generally modify one another; in some instances one affection of this kind exercises a protective influence against another, either temporarily or permanently, or greatly modifies it, as is well exemplified in the relation which exists between small-pox and cow-pox. Not uncommonly a patient suffers from two or more contagious complaints in succession. Some individuals seem quite insusceptible to certain infectious disorders, without any apparent reason for this. In such cases it has been suggested that the disease has occurred during intra-uterine life.

External conditions have a very important influence in the dissemination of contagion. Unfavourable hygienic circumstances undoubtedly increase the virulence of many contagious poisons, such as those of typhus and relapsing fevers. It is believed that water intensifies the virulence of the contagium of typhoid fever and cholera, and some maintain that these contagia are harmless when they first escape, and only acquire virulent properties after a certain interval. Climate and season have a considerable modifying influence, some diseases requiring a high temperature for their development, others being checked by much external heat. The direct application of great heat or extreme cold, as well as of certain chemical agents, is of the highest importance in destroying contagious poisons and rendering them inert, as upon this effect depends in a great measure the power which we possess of checking the spread of the diseases originated by these agents. Among the most efficient chemical substances acting thus are chlorine, iodine, hypochlorite of lime, chloride of zinc, sulphurous acid and sulphites, creosote, carbolic and cresylic acid, Condyl's fluid, and chloralum. The bearing of certain experiments made with contagious germs, by which the character of the diseases they produce is modified, will be presently alluded to.

**3. Nature of Contagion.**—There are certain contagious affections which are evidently dependent upon definite parasitic plants or animals, and in each individual disease of this class, the particular animal or plant which originates it obviously constitutes its contagious element. With regard to the ordinary infectious diseases, it is assumed that in connection with each of them there is a "specific morbid agent or poison, capable of exciting and propagating this one disease and no other, and without the action of which upon the system it cannot possibly arise." This supposed poison has been variously named a *contagium*, *virus*, *zyme*, or *ferment*; and its nature we have now to consider.

Some contagious diseases, and especially infective forms of inflammation, are associated with distinct morbid products, such as pus. It is commonly believed that in these affections the contagium is something distinct from such morbid materials, and that these only form a vehicle for its transmission, but some high authorities maintain that in the contagious inflammations the inflammatory products themselves constitute the *contagion*.

The theories as to the nature of contagion which are at all worthy of consideration at the present time may be classed as:—**1. Chemical or Physico-chemical.** **2. Vital or Germ-theories.** Under the former, two chief views deserve to be mentioned, namely:—(a.) That the contagium in each disease is a specific chemical compound, probably of an organic nature, and either solid, liquid, or in the form of a volatile gas. (b.) That it is albuminoid matter in a state of rapid chemical and physical change—in short, mere decomposing organic matter. The *germ-theory*, or theory of *contagium vivum*, is now in the highest favour, although still not universally accepted. According to this hypothesis, it is affirmed that all contagious diseases arise from the action of *living organisms* or *germs*, which are specifically distinct from each other in the several individual affections. There is by no means an agreement in the opinions held as to the nature of these supposed disease-germs, or as to the precise mode in which they exercise their morbid influence. It is generally believed that they are *living microscopic organisms* or *germs*, which according to the best authorities belong to the vegetable



kingdom, though some refer them to the animal kingdom, and variously described as bacteria, bacilli, vibrios, micrococci, microzymes, zoogloea, &c. There are also different views entertained with respect to the part which these organisms play in the propagation and development of contagious maladies. It is usually supposed that they themselves constitute the *contagium*, and that "their powers of producing disease are due to their organic development." Dr. Burdon Sanderson has suggested "that they may serve as carriers of infection from diseased to healthy parts, or from diseased to healthy individuals, and yet be utterly devoid of any power of themselves originating the contagium they convey." Sir Joseph Lister has advanced the view "that the fungi and their relations, bacteria, may contain in themselves some chemical compound absolutely peculiar to them, and forming part of their substance, which may act upon albuminous compounds after the manner of a ferment." Dr. William Roberts has brought forward the hypothesis that organisms become contagious through a process of "variation" or "sporting" under certain circumstances, from organisms originally harmless, as is frequently observed in the vegetable kingdom.

Dr. Beale entertains a very distinct germ-theory from that just considered. He maintains that the germs are not parasites, but that they are extremely minute particles of *living germinal matter* or *bioplasm*, which present no differences in appearance in different diseases, even under the highest powers of the microscope, but have an essential difference in vital power. He says "a disease-germ is probably a particle of living matter, derived by direct descent from the living matter of man's organism." Dr. MacLagan's opinion is that "the organisms which produce the phenomena of disease are not those which we see and describe as bacteria, but other and much more minute organisms."

The main arguments upon which the germ-doctrine rests may be briefly stated as follows:—First, the foundation for this doctrine as applied to contagious diseases originated in a supposed analogy between ordinary fermentation and the mode of action of contagia; and it was maintained that fermentation is invariably associated with, and dependent upon the growth of low organisms. Moreover, special kinds of fermentation are attended with the formation of special organisms; thus, vinous fermentation is set up by, and is accompanied with the development of the *torula cerevisiæ*, while Lister has shown that a specific organism, which he terms the *bacterium lactis*, always leads to the lactic acid fermentation. It has not yet been definitely settled, however, that organisms are essential for the fermentative process; while those who are opposed to the germ-theory of disease will not allow that there is any real analogy between fermentation and contagion. Secondly, the germ-doctrine is said to explain more satisfactorily than any other the phenomena which contagious diseases present, the rapid multiplication of contagium within the body, as well as its power of retaining its vitality for long periods, and of resisting destructive influences. Thirdly, infective inflammations are found to be attended with the development of abundant bacteria in the affected tissues; and these organisms have been proved to be concerned in originating septicæmia. Bastian and others, however, look upon the bacteria present in the tissues which are the seat of infective inflammation, not as the cause of the morbid process, but as pathological products, being developed from pre-existing germs, the tissues undergoing

a bacterial degeneration. Fourthly, the connection between certain diseases and specific organisms now seems to have been definitely traced. The disease called "splenic fever" in animals, and "malignant pustule" in man, has been proved to be caused by an organism which has been named *bacillus anthracis*. Relapsing fever is connected with the development of an organism called the *spirillum*. Klein has discovered that the disease named pig-typhoid is due to another specific organism. Vaccinia, small-pox, sheep-pox, and a few other contagious diseases have been attributed to certain minute particles, named micrococci, and supposed to be of the nature of germs. A specific *bacillus* has been described in typhoid fever, ague, and recently in cholera, by Koch; while organisms have also been referred to measles, diphtheria, erysipelas, &c.

Remarkable experiments have been made, mainly by Pasteur, which are strongly corroborative of the germ-theory as applied to certain particular diseases, while of great practical value in other respects. Without entering into details, it must suffice to state that, by a certain method of cultivation, under the influence of a moderate supply of oxygen, of the organism producing a very fatal form of infectious disease in fowls, known as "chicken-cholera," Pasteur could so attenuate it and mitigate its virulence that, when inoculated, it caused only local mischief, with slight constitutional disturbance, and this mild attack was protective, at any rate for a year, against future attacks. He obtained similar results with the *bacillus anthracis*, which had been previously experimented upon by Dr. Greenfield. He cultivated an attenuated poison, by which he could produce a mild form of splenic fever in cattle, quite free from danger, but absolutely protective against subsequent inoculations of the disease in its most virulent form. Still more recently this eminent experimenter has produced remarkable results by the inoculation of the poison of hydrophobia, attenuated by cultivation, in preventing or mitigating the disease. The bearing of such investigations upon the mitigation of infectious diseases in the human subject is obvious, but this is a result to be looked for in the future.

**4. Effects of the action of a contagium upon the system, and the changes it undergoes.**—The effects of a contagium may be entirely local and superficial, as in the case of scabies, and probably gonorrhœa; or in a few instances they are at first obviously local, but subsequently become general or constitutional, for example, syphilis, vaccinia, and small-pox by inoculation. As a rule, however, the primary action of the morbid principle seems to be on the general system, and this is usually followed by local lesions.

When specific diseases begin with local effects, as in syphilis or inoculated small-pox, the lymphatic glands next above speedily become specifically affected. At or about the time that these changes are at their height, febrile symptoms appear, and soon after the characteristic eruption is developed. In the case of vaccinia there is no such eruption. In those affections where there are no obvious primary local effects, it is generally believed that the several contagia act first upon the blood, or, as some think, upon the nervous system. The view held by some pathologists, however, is that even in these diseases the poison produces in the first instance specific local processes at the spot or spots where it reaches the body, followed by changes in the next lymphatic glands, and then by general infection of the system. This idea has been particularly applied to diphtheria, cholera, and typhoid fever. In any case the general system and the blood become affected more or less speedily



in the several diseases. The favourite notion is that a fermentative or *zymotic* process is set up, and that there is an analogy between ordinary fermentation and the mode of action of contagia. Dr. Bastian rejects the idea of any such analogy, and maintains that a contagium has merely a chemical or physical action upon the blood and tissues. Dr. William Roberts is inclined to the belief that in regard to specific contagia we shall find more guiding analogies in parasitism than in fermentation.

After a contagium gains access into the system it undergoes rapid increase, and according to the germ-theory the germs multiply abundantly, probably at the expense of the albuminoid elements of the blood, walls of the vessels, and tissues, so that the minutest quantity of a contagious poison introduced into the body may generate an enormous amount of the same. The contagious elements are thus universally diffused by the blood through the body, and reach those structures which afford a suitable soil for their further development or growth. At first there is no evident sign of their action upon the system, but a *period of incubation* ensues, differing in duration in different diseases, though having tolerably defined limits in each several malady. This incubation-period is generally considered as lasting from the time of entrance of the contagium into the system until the first onset of definite symptoms, there being in the meantime either no symptoms at all, or none of any distinct character. It may be very prolonged, as in the case of hydrophobia, which may remain dormant certainly for many months. In the case of the eruptive fevers, however, Dr. W. Squire and others reckon this incubation-period as terminating with the appearance of the eruption, but it will be more convenient in this work to follow the definition given above. The action of the contagious poison, when it originates a *fever*, is first indicated by more or less severe general symptoms of a pyrexial character, the onset of which is usually well-marked, and is characterized by rigors and other phenomena. Frequently there are significant local symptoms in addition. The contagium may act so violently upon the system, and its increase may be so rapid, as to cause death at the very outset, and without the production of any evident structural lesion. If this event does not happen, the local manifestations or specific lesions of the disease become developed after a certain time, which may be limited to one tissue or organ, or be observed in several parts, and these constitute its *anatomical characters*. The various eruptions characteristic of many of the contagious fevers constitute important local manifestations of these affections. The local lesions are indicated by corresponding symptoms, but they often aggravate the constitutional disturbance as well. After a certain period has elapsed the symptoms subside, and if there has been fever deferrescence follows according to one or other of the methods already described. The poison ceases to increase, and is finally expelled altogether out of the system. Permanent structural changes may or may not remain.

It is important to observe that in each of these specific diseases there is a considerable regularity and uniformity, not only in the course of the stages above described, but also in their duration, and therefore in that of the entire affection from first to last, and it is necessary to become acquainted with this *natural history* of the several maladies. Complications and sequelæ are, however, very liable to arise, which interfere with the natural progress of events. Great variety is also observed as



regards the intensity of these diseases. In some instances they are very mild; in others they assume a typhoid or malignant type, and are extremely fatal. This difference is sometimes seen running through epidemics.

5. **Elimination of Contagious Poisons.**—As has been already stated, in many instances the contagium ceases to multiply after a time, and it passes out of the system, this also occurring during the entire course of the malady. The main theories as to the modes in which this removal is effected are:—*a.* That the living particles by their activity make their own way out of the vessels and through the tissues, and thus reach the surface. *b.* That they are conveyed outwards suspended in the fluid which transudes from small vessels. *c.* That the poison is directly eliminated by the agency of epithelial and secreting cells, especially those of the skin, kidneys, and intestines. According to this idea the cells attract and separate the contagium, and are then cast off, being replaced by new elements. Those who believe in this theory look upon the eruptions, epithelial desquamation, diarrhoea, and similar phenomena, as efforts of nature to eliminate the poison, and on this they found a special treatment, by which they propose to assist nature in this eliminatory process. There are strong objections, however, against such a view. Beale argues not only that the cells have no eliminatory power, but that the poison actually destroys them, and that this is the cause of the profuse shedding of epithelium which is observed at the close of some of the contagious diseases, such as scarlet fever.

## II. ON EPIDEMICS.

Diseases are divided into three classes, according to the manner in which they are disseminated amongst the population, namely:—1. *Sporadic*, or those which occur in an isolated and scattered manner, and do not attack large numbers of people at the same time, *e.g.*, bronchitis. 2. *Endemic*, or those which are peculiar to certain districts, or which are constantly prevalent in these districts to a greater or less extent, *e.g.* ague and goitre. 3. *Epidemic*, or those which suddenly attack large numbers of people, and spread rapidly amongst them, often causing great devastation, this event occurring at irregular intervals, and lasting a variable time, but being of limited duration, *e.g.*, cholera, enteric fever, or small-pox. These three classes, however, are not absolutely distinct. Sporadic cases of epidemic diseases are common enough, and these are also frequently endemic in a district; for instance, typhus fever often prevails in the filthy quarters of large towns; and yellow fever, plague, and other endemic affections may become epidemic. The terms *miasmatic* and *zymotic* are in common use to designate certain diseases. *Miasmatic* has been applied to all *specific fevers*, but it is often limited to those of *malarial* origin. The word *zymotic* does not imply any fermentation-theory of disease, but is now made to include all epidemic, endemic, and contagious maladies, which are capable of being prevented by proper attention to hygienic and other conditions.

Attention will now be directed to the subject of epidemics. An epidemic may often be distinctly traced to the direct influence of contagion, aided frequently by unfavourable hygienic conditions, or to some other obvious cause, such as famine, but in many instances its origin

cannot be thus definitely fixed. Certain diseases prevail as epidemics, the infectious nature of which is much disputed, for instance, influenza. Various theories have been suggested to explain the occurrence of epidemics under these circumstances. They have been attributed to an *epidemic influence* or *constitution*, which has been supposed to reside in the atmosphere around us, and to depend upon the influence of the heavenly bodies; upon gases emitted in connection with volcanoes and earthquakes; upon the electrical condition of the air; upon the quantity of ozone in it; or upon the rapid development and migration of microscopic animalcules. All these, however, are mere hypotheses. It is not improbable that certain conditions affecting the vitality and development of contagious germs account for the occurrence of some epidemics. When an epidemic of a contagious disease arises as the result of evident anti-hygienic conditions, or from some other obvious cause, it is believed either that the specific poison is increased in quantity or rendered more virulent; or that the constitution of individuals becomes so altered as to render them more amenable to its influence, and less able to resist it.

The chief facts which have been observed in relation to epidemics may be stated under the following laws:—1. Epidemic influence chiefly affects those diseases which are infectious, rendering them more prevalent and more dangerous; or diseases which have a malarial origin. As a rule, only one of these complaints is epidemic at the same time, but sometimes there seems to be a tendency to the prevalence of several of the acute specific diseases simultaneously. Occasionally other maladies appear to assume an epidemic character; and now and then an entirely new disease makes its appearance in this way. Sometimes it is only the type of ordinary diseases which is influenced; or there is a tendency to the implication of special organs. 2. The prevalent epidemic affects more or less the characters of other diseases. This is well illustrated in the case of cholera and influenza, choleraic diarrhœa being very common during the existence of the former, catarrhal affections during the prevalence of the latter. 3. The extent of an epidemic varies much. If this is very large, the disease usually attacks different places in succession, becoming milder in one region as it invades another. It may be confined to a certain limited district, being then usually due to some evident local cause. Amongst the diseases which often occur in extensive epidemics may be mentioned influenza, measles, scarlatina, and small-pox. Typhus and relapsing fevers are limited to districts where certain anti-hygienic conditions exist; cholera, typhoid fever, and diphtheria often occur in irregularly scattered local outbreaks. 4. The progress of an epidemic is also subject to variations. Generally it advances regularly onward in a definite direction, and in this way may make the circuit of the globe. It may advance very rapidly, or exceedingly slowly and gradually. Sometimes an epidemic seems to leave a place and then return, as if falling back upon itself; or it passes over particular regions without affecting them; or it goes out of its course in a lateral direction, attacking parts not in the line of progress. Epidemics are not under the influence of winds, as they frequently advance in a course directly contrary to these. 5. The mode of invasion may be sudden, or more or less gradual; usually the latter. An epidemic disease also generally gives indications of its approach by the occurrence of cases presenting some of its symptoms in a mild form; thus, cholera is generally preceded by cases of diarrhœa, or a few sporadic cases may occur, giving warning of its advent. 6. The intensity of an epidemic is subject to much variety,

the disease being in some instances exceedingly fatal, in others comparatively mild. It is most virulent as a rule at the early period, judged by its characters and fatality. This is partly explained by the fact that probably those are first attacked who are most predisposed. 7. The mode of disappearance is generally gradual also, cases becoming by degrees less severe and fewer in number; but it may be rapid, either from some evident cause or without any apparent reason. 8. The duration of an epidemic is very uncertain. It may persist, with intermissions, for several years, as in the case of cholera. 9. Cycles of epidemics are frequently observed, one disease being after a certain time followed by another, and this by a third, and so on. The theory has been advanced in explanation of the occurrence of epidemics, that there is what is termed a *pandemic wave*, under the influence of which a series of oscillations of febrile diseases occur, these following each other regularly over the globe. 10. It is most important to notice that epidemics are greatly under human control, and they can be prevented or made much less severe by attention to proper hygienic and other measures, which will be presently considered. With the advance of civilization some epidemic, as well as endemic diseases, have been entirely eradicated from countries and districts where formerly they were exceedingly rife; and by the aid of well-directed efforts there is no reason why many others should not be completely expelled from our midst. 11. Epidemic influences seem to affect other animals at the same time as human beings, and it is not at all improbable that this is also true with regard to plants.

### III. ON THE HYGIENIC MANAGEMENT OF CONTAGIOUS FEVERS; AND THE PREVENTION AND LIMITATION OF EPIDEMICS.

A most important object to be kept in view, when treating a patient suffering from an infectious fever, is to prevent its extension to others, and the means which promote this end are also most useful as regards the well-being of the patient. The measures to be attended to will now be briefly considered.

1. *Separation* from other individuals is necessary as far as possible, and in many cases almost complete *isolation* is demanded. At all events anything like overcrowding must be avoided, and only those persons who have any business in the sick-room should be admitted. They should wear clothes to which the contagium cannot easily adhere, and go as little as possible into the midst of healthy people. Medical men ought to take adequate precautions against conveying any contagious affection.
2. *Proper ventilation* is essential, and this is best carried out by placing the patient in a large room, and opening the windows more or less according to the weather, by night as well as by day, care being taken of course to protect the patient against draughts. A good fire in the room materially assists ventilation.
3. All excessive curtains, bed-clothes, carpets, and other objects which might act as *fomites*, ought to be removed. In this way ventilation is also promoted.
4. *Cleanliness* must be thoroughly attended to, as regards the patient, bed, clothing, bedroom, &c.
5. Those who come into contact with the patient should avoid inhaling the breath or exhalations, and should afterwards not swallow their saliva, but cleanse out the mouth and nostrils.
6. One of the most important matters to attend to is the *disinfection* or *complete*



*destruction* of everything which is capable of conveying contagion. In the first place all *exhalations* and *discharges* should be at once *disinfected*. Anything coming off from the skin is best destroyed by frequent sponging with some suitable disinfectant, such as a weak solution of Condyl's fluid or carbolic acid, or by applying carbolic oil. The air of the room should also be somewhat impregnated with some volatile material of this nature, such as chlorine (from chloride of lime), carbolic acid, or sulphurous acid. It is further recommended to place across the doorway a sheet moistened with dilute carbolic acid, Burnett's fluid, Condyl's fluid, or chloralum. Secretions from the nose or mouth ought to be removed by means of disinfected rags, these being immediately burnt. Excretions *should be received into utensils containing some disinfectant*, and thoroughly mixed with this before being removed from the room. This is especially needful in the case of those diseases which are known to be propagated chiefly by the stools, namely, cholera and typhoid, and if possible a separate water-closet should be used for the reception of the excreta in these affections, which should be frequently flooded with some disinfecting fluid. The best disinfectants for this purpose are carbolic acid and carbolic powder, chloride or sulphate of zinc, chloride of lime, or chloralum. All dirty clothing, bed-clothes, &c., must be put at once into vessels containing some disinfecting fluid, especially Condyl's or chloride of lime, before being taken out of the room for the purpose of being washed. The clothes previously worn by a person suffering from a contagious disease ought also to be disinfected. The floor, doors, windows, &c., should likewise be frequently washed with some disinfectant. 7. *Food*, such as beef-tea, must on no account be allowed to remain for any length of time in the sick-room, and should never be taken by any one who is not habitually in the apartment. 8. After the patient has left the sick-room, it should be thoroughly *cleansed and disinfected* in every corner, and then whitewashed, or re-papered and painted. Sulphurous acid, chlorine, and carbolic acid are the most useful agents for disinfecting an unoccupied apartment. Heat is very efficient for the purpose of disinfecting bedding and bed-clothes.

It is necessary to carry out these measures more or less thoroughly in proportion to the degree of contagiousness which the particular disease presents. For example, they demand strict attention in the case of scarlatina and small-pox.

When an epidemic has appeared in, or threatens to invade, a district, additional precautions are called for, as regards attention to proper hygienic conditions and other matters. Under these circumstances it is requisite to instruct ignorant individuals as to what course to pursue; and to appoint competent persons to visit from house to house, to see that the necessary measures are properly carried out, especially in low and crowded parts of towns and cities. The chief practical points to be noticed are as follows:—

1. *Cleanliness* must be strictly observed in every particular. Frequent washing and whitewashing of premises is required.
2. All *overcrowding* must be prevented, and *free ventilation* insisted upon. It is particularly necessary to look to this among the poor, and in common lodging-houses or crowded alleys.
3. Persons who are likely to spread infection *must not mingle with others* in places of public resort.
4. Special attention must be paid to all *decomposing organic matter*, especially *house-refuse*. Everything of this description should, if possible, be at once removed, having been previously disinfected; or if it cannot be got rid of,

abundance of disinfecting material must be mixed with it. House-drains and sinks, street-drains and sewers, water-closets, cesspools, privies, ditches, &c., require careful and frequent examination, so that they may be kept in order. The earth in the neighbourhood of dwelling-houses is often saturated with organic matters, and therefore demands attention. During the removal of organic matters from houses it is well for the inhabitants to keep away as much as possible. 5. *Disinfectants* should be freely employed in and around houses, especially where there is much filth. 6. It is most important to look to the source of the *water-supply*, particularly that which is used for drinking purposes, and to see that no organic matter finds its way into it from sewers, drains, cesspools, polluted ground, &c. The waste-pipe of cisterns and baths often opens into drains, and, owing to an imperfect state of the traps, organic matters or injurious gases become mixed with the water. This matter must be especially attended to during an epidemic of typhoid fever or cholera. On no account should water be taken which contains any organic matter, and it ought always to be filtered. 7. It may be advisable to *remove healthy persons* to some place where they would be free from the danger of infection. 8. If there is any known *preventive* of an epidemic disease, this must be at once resorted to, and fully carried out. Thus vaccination or re-vaccination should be thoroughly enforced during an epidemic of small-pox, in the case of all who have not previously undergone the operation. 9. It may be requisite to carry out the practice of *quarantine*. 10. It is important that the *general health* of the community should be maintained by every possible means, and all causes which tend to lower the system, such as intemperance or bad living, should be avoided. It is particularly necessary for those who attend upon the sick to take every precaution for the preservation of their health. They should live well, but not indulge too freely in stimulants; and they should never go with an empty stomach near the sick person. They require daily exercise in the open air, at the same time avoiding undue fatigue. They also need sufficient sleep, and must pay strict attention to cleanliness. 11. Any person who presents the *slightest symptoms* of the disease which is epidemic ought to be, *without delay*, brought under medical treatment. 12. On no account should an individual suffering from an epidemic disease of an infectious character be *brought into the midst of healthy persons, or into an unaffected district*, if this can possibly be avoided. The conveying of infected persons by *vehicles used by the public* is a serious crime, which the law now takes cognizance of and punishes. Special conveyances are provided, should it be necessary to remove them to a hospital.

---

### CHAPTER III.

#### ON THE CLINICAL INVESTIGATION OF ACUTE FEBRILE DISEASES.

A LARGE proportion of the cases which come under observation in ordinary practice belong to the class of *acute febrile diseases*, and it is most essential to have a clear understanding as to how to proceed in

their investigation, because it is particularly important that a correct diagnosis should be arrived at in these cases as speedily as possible. The special points which require attention are as follows:—

1. It is well first to inquire what febrile complaints the patient has previously suffered from, if any. It should then be ascertained whether the patient has been exposed to any infectious disease; or whether there has been any other obvious cause at work likely to give rise to a febrile condition, for example, malaria or cold.

2. If possible, the **exact date, even the hour**, of the onset of symptoms must be fixed; and the **mode of invasion** ascertained.

3. The **symptoms** which have arisen in the course of the case are then to be inquired about, as well as the times at which they appeared; those being subsequently noted which are present at the time of examination of the patient. During their *premonitory* or *early stage*, the acute specific fevers are usually attended with certain *peculiar symptoms*, differing in each complaint, and more or less characteristic. Those which require to be particularly investigated are:—The severity of general pains; the existence of any localized pain, especially in the back or epigastrium; catarrhal, throat, stomach, intestinal, and head-symptoms. Of course, if the pyrexia is due to inflammation of some organ or tissue, there will probably be local symptoms indicating the seat of mischief.

4. It is of the greatest importance to determine the **degree of pyrexia**, as well as its **course** and **mode of progress**. This is done by the careful and systematic employment of the thermometer. In this way even at a very early period much assistance is gained in arriving at a diagnosis. In the exanthemata the fever is of the *continued* type. By its degree of intensity and rapidity of onset the nature of the complaint may, in many cases, be foretold at an early stage. Each fever is supposed to have a definite course of temperature, and on the whole this is true; but a good many exceptions are met with in particular cases.

5. Most of the acute specific fevers are attended with a **skin-eruption**. This constitutes one of their chief *anatomical characters*, and generally enables each of them to be at once distinguished from all others. It must be remembered, however, that the eruption is not always present, for there is no doubt but that scarlatina, measles, small-pox, and other exanthemata may run their course without any eruption. When due to inoculation, it may be confined to the spot where the morbid material is introduced, as in the case of vaccinia. The points to be ascertained with regard to each eruption are:—*a.* The *exact time* of its appearance after the first onset of symptoms. *b.* Its *primary seat*, as well as the parts of the body to which it spreads, and the mode and rapidity of its extension. *c.* Its ordinary *amount*. *d.* Its precise *characters*, from its first appearance to its decline, including any changes which it may undergo during its progress. *e.* Its *duration*, both as regards the entire eruption, and its individual constituents. *f.* The cutaneous *sequelæ* which may follow it, such as desquamation. *g.* The chief *varieties* it may present.

6. In all febrile cases it is imperative to make a careful **physical examination** of all the chief organs of the body, and to **examine the urine**. This may reveal the cause of the pyrexia, even when there are no symptoms pointing to any particular organ. Besides, it must be borne in mind that even in the specific fevers complications are very



liable to arise, or some of the organs may be primarily involved, and it is most important to detect any lesion in connection with these organs as early as possible. Hence *daily examination* at least should be carried out, and in many cases it is requisite to examine the principal organs even more frequently than this. The *sphygmograph* is of value for the purpose of indicating the condition of the circulation.

#### ON THE USE OF THE THERMOMETER.

The value of the *thermometer* in the investigation of disease is at present so generally recognized in theory that it is unnecessary to enter into any discussion on this subject; at the same time, it is needful to impress upon all the extreme importance of employing this instrument in *daily practice*, because there can be no doubt that even now many do not use it to the extent which it deserves.

Neither is it requisite to give any detailed description of the instrument used. The thermometer should be sensitive and accurate; of a sufficient range and marked in fifths; self-registering; easily read; and of a convenient size to be carried in the waistcoat-pocket. These conditions are fulfilled in the *clinical thermometers* which are sold in most respectable instrument-shops. It is well, however, to compare the one used with a standard thermometer from time to time. Longer instruments than those usually employed are made for taking extreme temperatures; extremely sensitive thermo-electric apparatus are used for special researches in temperature; and self-registering thermographs have also been invented for continuous observations. For taking surface-temperatures mercurial thermometers are made of special shape, having a long cylindrical bulb coiled up in a plane at a right angle to the stem; thermo-electric and other instruments are likewise employed for this purpose.

**Mode of use.**—The regions usually employed for taking the temperature by means of the clinical thermometer are the axilla, the inner side of the upper part of the thigh, the mouth, the rectum, or the vagina. Sometimes it is requisite to determine and to compare *local* temperatures. The instrument must be kept in close contact with the surface, and completely covered. When the temperature is taken in either axilla, which is the most convenient place in most cases, the patient should lie on the same side, and press the arm firmly to the side; or it may occasionally be necessary to strap the thermometer to the surface by means of plaster. If there is much perspiration the skin should be wiped dry before inserting the thermometer. The mouth does not afford accurate results, but is convenient for giving approximate information, the thermometer being placed under the tongue, and the mouth firmly closed. The rectum and vagina are the most reliable, but, for obvious reasons, cannot be made use of in ordinary cases, but under certain circumstances the temperature should be taken in one or other of these cavities, as in restless children, very emaciated adults, or patients who are unconscious. Care must be taken that the instrument does not slip into the rectum, and that it is not broken by restless movements. The patient should lie on his side, the thermometer being then introduced for about two inches, and held there with one hand, while the other hand is placed on the patient's hip. With regard to the time required for the instrument to be retained in its position, there is

a difference of opinion. With proper precautions *five minutes* is usually sufficient, especially if "two observations at intervals of one or two minutes give exactly the same result" (Aitken). To be strictly accurate, however, the mercury ought to *remain stationary for five minutes*. Baumler gives, in order to be scientifically correct, and in obscure cases, where a trifling elevation of temperature may be of importance in diagnosis—for the rectum, three to six minutes; mouth, nine to eleven minutes; axilla, ten to twenty-four minutes. More time is required if the circulation is weak. The time needed in the rectum may be much shortened by heating the thermometer to a degree a little below that which is expected; and in the case of the axilla, although usually, and in first observations, the index should be shaken down to or below normal before the temperature is taken, it may often be previously raised in the same way. The time required in the axilla and mouth may be materially shortened by keeping these cavities closed for from ten to fifteen minutes before the thermometer is introduced.

It is desirable, if possible, that the individual upon whom the observation is made should have been at rest in bed for at least an hour previously. Not unfrequently, however, the thermometer has to be employed without any such preparation.

The intervals at which the temperature should be taken will vary according to the nature of the case. Often only one observation is required. In most instances twice a day is sufficient, namely, in the morning and evening, and in many once daily is enough. Sometimes, however, it is most important to note the temperature at very frequent intervals, or even to take continuous observations. Should this be needful, it is advisable to teach the nurse or some other intelligent person how to use the instrument, by whom it might also be employed if any unusual symptoms should arise. In all febrile cases it is requisite to have recourse to the thermometer until convalescence has been firmly established, for reasons to be presently indicated.

In using the thermometer the points to be observed are:—1. The *exact temperature*, as indicated by the *end of the index most distant* from the bulb of the instrument. 2. The *rapidity with which the mercury rises*, this being in proportion to the height of the temperature. It is often important to take a note at the same time of the *frequency of the pulse and respirations*; and in some cases to make a *quantitative analysis of the urine*, in order to determine whether there is a relation between the temperature and the amount of urea, uric acid, and other waste-products discharged. All these observations should be recorded on proper forms or charts, of which several have been planned, the temperature being indicated by angular lines or curves. It may be mentioned here that Fahrenheit's scale is followed throughout this work.

**Temperature in health, and chief modifying influences.**—In the axilla the temperature in health averages about 98·4° F. It may range, however, from 97·3° to 99·5° or even 100°, but if it goes beyond this in either direction, and remains persistently above or below the normal, there must be something wrong. The chief circumstances which influence the temperature in health are as follows:—1. *The part of the body* in which it is taken. It is higher in internal parts, such as the rectum or back of the mouth, than in external parts; in sheltered regions of the body than in those which are exposed; over the trunk than over the limbs. 2. *Age*.—The temperature, according to most

observers, is higher in children and young persons than in adults. It is also said to rise in old age. 3. *Time of the day*.—During the day the temperature rises until evening, and then falls slowly till early morning, when it again ascends. In this way there is a variation of about  $1.5^{\circ}$  during the twenty-four hours in adults, but the range is greater in children. 4. *Climate and exposure to heat or cold*.—In the tropics the average temperature is a little higher than in temperate or cold climates, and it may reach  $99.5^{\circ}$  or even  $100^{\circ}$  F. Long exposure to great heat or cold will also influence it to a slight degree. 5. *Food and drink*.—After a full meal the temperature at first falls, but it rises as digestion proceeds. Fasting lowers the temperature. Alcohol seems to cause a speedy fall, but this is only temporary, and a considerable quantity is required in order to influence the temperature materially. Certain articles of diet in daily use produce some effect, such as tea and coffee. 6. *Exercise* increases the temperature, especially that of the extremities, provided it is not sufficient to induce great fatigue. 7. *Mental conditions*.—Prolonged study and other forms of mental effort cause a slight depression. 8. The late Prof. A. B. Garrod found that the temperature rises on stripping off the clothes and exposing the surface of the body, and the difference is greater in proportion to the coldness of the surrounding air. When the temperature of the air is above  $70^{\circ}$  F., there is a slight fall, but a rise to the previous temperature soon takes place.

**Uses of the thermometer in disease.**—In the great majority of cases disease tends to raise the temperature to an abnormal height, there being more or less *pyrexia*; and it is for the purpose of accurately determining the degree of this increased bodily heat that the thermometer is chiefly employed. Occasionally it sinks below the normal, or may be unequal in different parts of the body, but these deviations are not nearly of so much consequence as a general rule.

At present it is only intended to sum up concisely the circumstances under which the thermometer may prove serviceable. The peculiarities which individual diseases present as regards temperature will be pointed out when these are severally discussed.

The information afforded by the thermometer may give valuable assistance in:—1. Diagnosis; 2. Prognosis; 3. Treatment.

1. Much help is constantly derived from the thermometer with respect to *diagnosis*, and the following remarks may serve to gather up the circumstances under which it is thus useful. *a.* In many cases which present themselves in ordinary practice, where symptoms exist which might or might not belong to the premonitory stage of some acute illness, all doubt may often at once be cleared up by taking the temperature. Thus I have frequently found in the out-patient room, that when symptoms suggestive of scarlatina or small-pox were complained of, by the help of the thermometer we have been enabled to negative the supposition of either of these diseases being present, or, on the other hand, to corroborate such a suspicion. In short, the instrument enables us at once to determine *whether pyrexia is present or not*, as well as its *degree*, and thus becomes a most valuable aid to the practitioner, which he should ever keep in mind. *b.* Occasionally by one, or at most two observations, it is possible to ascertain positively the *nature of a fever*. For instance, if the temperature suddenly rises to  $104^{\circ}$  or  $106^{\circ}$  F., the patient having been quite well on the previous day, he is probably suffering from some form of malarial fever, and this



is certain if the temperature falls rapidly, so that it becomes normal in a few hours. *c.* Many febrile disorders are now known to have tolerably *regular and uniform ranges of temperature* throughout their entire course, and to present peculiar diurnal and nocturnal variations, the temperature being, as in health, generally higher by night than by day. It is therefore essential to become acquainted with this portion of the *natural history* of each of these affections, and to employ the thermometer regularly in investigating them, so that they may be thus distinguished from each other, and from all complaints which may simulate them. *d.* The *habitual use* of the thermometer may lead to the discovery of disease when there is no obvious sign of its existence, for the fact of a patient presenting a temperature above the normal should always call for a more minute examination, which would probably lead to a satisfactory diagnosis. This has been frequently observed by those who employ the thermometer in lunatic asylums, who have thus detected phthisis in insane patients when they could not otherwise have suspected it. *e.* *Complications* occurring during the progress of fevers or during the period of convalescence, as well as *relapses*, are indicated either by a disturbance of the typical range, by delayed defervescence, or by a rise in temperature after it has once subsided; and either of these deviations may be the first thing observed. Hence the necessity of taking a daily note of the temperature until the patient has perfectly recovered. *f.* In certain diseases the thermometer gives information as to the *activity of the progress* of a morbid process, for instance, in pulmonary phthisis. Further, it may occasionally help in distinguishing between *different forms* of this complaint. Again, in connection with hæmoptysis, the thermometer is useful in indicating that inflammation has been set up by blood extravasated into the respiratory organs. The same remark applies to the effects of an apoplectic clot in the brain. *g.* *Depression of temperature* is an important sign of certain conditions, as in collapse from various causes, when it may sink considerably and rapidly; some cases of severe injury to the upper part of the spine; certain diseases of the brain and cord; starvation; great loss of blood; and some chronic wasting diseases. It is somewhat below the normal also in certain chronic pulmonary and cardiac affections, and in chronic Bright's disease; and it must be remembered that in these conditions the temperature may not rise to the usual height when febrile complaints supervene. Sometimes the temperature is low on the surface, but high internally, as in cholera. *h.* *Inequality of temperature* in different parts is sometimes of aid in diagnosing paralysis or other nervous disorders. In a paralyzed limb the temperature may be higher or lower than in the corresponding limb on the opposite side; in hemiplegia the paralyzed side is often  $\frac{1}{2}^{\circ}$  to  $\frac{3}{4}^{\circ}$  higher than the healthy one. Neuralgia may be accompanied with marked local rise of temperature; and a similar condition may be observed in hysteria. These differences all depend on vaso-motor influences. Local stagnation of blood and its consequences lower temperature. Of course local differences of temperature may be due to external causes. Attempts have been made to use local temperatures in the diagnosis of pulmonary phthisis, pneumonia, pleurisy, brain-affections, articular diseases, tubercular formations, and other conditions, but the results are not reliable.

A word of caution is necessary with regard to children. In these subjects the temperature may run up rapidly to a considerable height,

when there is nothing particular the matter, and therefore care must be taken not to jump to a hasty diagnosis of some serious disease, simply because the thermometer indicates much bodily heat. It often falls with equal rapidity.

2. The temperature may be of use in assisting towards a *prognosis*, either in itself; from its relation to the pulse, respirations, or amount of excreta; or from its association with other symptoms. *a.* The *degree of heat* observed during the early period of a febrile disease, especially when taken in conjunction with the prominent symptoms, will often give a good idea as to whether the particular case under observation is likely to be a severe one or not. If the temperature is at all high, it shows that a sharp attack may be anticipated, and that complications resulting from the presence of products of decomposition in the blood are liable to arise; therefore a guarded prognosis should be given. *b.* A *very high* temperature, especially when it exhibits a tendency to a continuous and rapid rise, is extremely dangerous, especially if the excretions are deficient. *b.* A *sudden change* in the temperature may be premonitory of some coming event, even for some days before this actually occurs. Thus a marked fall in cases of typhoid fever not uncommonly precedes hæmorrhage from the bowels, and gives warning of its approach. *d.* If the temperature *does not increase*, or if it *falls from morning to evening*, this is a favourable sign; if it is *higher in the morning* than on the previous evening, this shows that the disease is advancing, and the prognosis is consequently more grave. *e.* In many pyrexial diseases the fever usually subsides on certain days, often by *crisis*; if in a particular case the expected fall takes place, and defervescence goes on regularly and continuously, the prognosis is favourable; if the contrary happens, or if the decline of the fever is irregular, an unfavourable course is indicated. *f.* Should the temperature *decline rapidly* in certain acute febrile affections, such as pneumonia or typhus fever, while the pulse and respirations increase in frequency, and the other symptoms show no signs of improvement, but on the other hand become worse, the prognosis is very serious. A markedly low temperature is in itself an evil omen in these complaints. *g.* A *very low* temperature, as in collapse, is more or less dangerous, and should it sink below 93°, the termination is almost always fatal.

It must be remembered that accidental circumstances may temporarily modify the temperature in disease as in health, such as food, exercise, excitement, &c. It may be increased by sources of irritation, such as retained urine or fæces, on the removal of such irritants being often markedly reduced. Defervescence may proceed so far that the animal heat is brought below the normal, sometimes considerably. After convalescence from severe continued fevers, the temperature often remains low for some time. The same condition is also observed during the apyrexial periods of intermittent fever; and in the remissions of the remittent variety.

3. The value of the thermometer as affording indications for *treatment* may be gathered from the remarks already made, and it will be only necessary to give two or three illustrations. A *very high and ascending* temperature calls for prompt recourse to the use of cold, as already described under the treatment of pyrexia. In *ague*, after this disease has apparently subsided, it is found that the temperature still rises at the usual intervals, and until this has become quite normal for two or three days, treatment must not be discontinued. During

*convalescence from fevers* an increase of the bodily heat may be due to something wrong in the diet or in the use of medicines, and such an event should lead to a careful inquiry on all matters which might tend to raise the temperature, so that appropriate measures might be adopted to remove the source of disturbance.

---

#### CHAPTER IV.\*

### SIMPLE CONTINUED FEVER—FEBRICULA—SYNOCHA.

A CONSIDERABLE number of cases come under observation in ordinary practice, which present the usual symptoms of *fever*, but which cannot be referred to any of the contagious fevers, nor can the symptoms be traced to any adequate local cause. Besides these, anomalous forms are not uncommonly met with, to which various names have been applied. Though many of these cases scarcely come under the class of acute specific fevers, they may be conveniently described here.

**ÆTIOLOGY.**—Simple febricula does not seem to be contagious, or to depend upon any specific poison as a rule. It may result from cold; excessive heat, such as prolonged exposure to the sun; over-eating or drinking; or great fatigue. In many instances no distinct cause can be made out. Probably some cases of so-called febricula result from the action of one of the contagious poisons, modified by the constitutional condition of the individual, or by the quantity entering the system being very minute. I have known a severe epidemic of typhoid fever to be preceded by cases which would be classed as mere febricula.

**SYMPTOMS.**—Febricula is characterized by the ordinary signs of *fever*, in their most typical and simple form, but of variable intensity. The *invasion* is indicated either by chilliness or slight rigors, with general pains, lassitude, and headache. Afterwards the skin becomes hot and dry; and the pulse frequent and full. Severe headache is usually complained of; the face is flushed; while the patient is restless, and sometimes a little delirious at night. There is thirst, with a furred tongue, loss of appetite, and constipation. The urine presents febrile characters. Frequently symptoms are present indicative of catarrh of the mucous membranes, but these are not sufficient to account for the pyrexia. Roseolar or erythematous eruptions have been observed in some instances, and in others certain bluish spots on the skin have been described.

The *temperature* rapidly ascends, usually to a moderate height, but it may reach 102°, 103°, or even 104° in a few hours. This high temperature, however, if it occurs, only lasts for a short time usually, often but a few hours, or at most one or two days, and then it falls rapidly.

**DURATION AND TERMINATION.**—The *duration* of febricula is generally about three or four days, but a week or ten days may elapse before convalescence is complete. Defervescence usually takes place by *crisis*, the

\* In the description of the individual *Acute specific diseases* in the following pages, I have deemed it expedient to omit the consideration of the *diagnosis* under each particular affection, and to devote a separate chapter to a general summary of this subject.



temperature falling to the normal in from 24 to 36 hours, there being also a copious discharge of urine, with abundant deposit of lithates; free perspiration; and sometimes diarrhœa or epistaxis. Occasionally defervescence takes place by *lysis*, convalescence being consequently delayed. The *termination* is always in recovery.

TREATMENT.—All that is required is to keep the patient in bed; to give a diet of milk and beef-tea, with cooling drinks; to open the bowels freely; and to administer some simple saline mixture, such as one containing citrate of potash or liquor ammoniæ acetatis. If there is much heat of skin, tepid sponging is very useful. During convalescence quinine may be given.

---

## CHAPTER V.

### TYPHUS FEVER.

ÆTIOLOGY.—Typhus fever is generated by a *specific poison*, and is highly contagious. The nature of this poison has not been definitely determined, but micrococci have been described. It is principally given off in the exhalations from the skin and lungs, being afterwards inhaled or swallowed. The cutaneous exhalations have a peculiar odour. Infection is far more likely to happen in the case of those who are brought into close and frequent contact with the sick, and hence nurses and medical men are very liable to be attacked. It must be borne in mind, however, that if there are a number of cases congregated together, so that the poison is concentrated, a very short, even a momentary exposure may cause the disease to be transmitted. The contagious influence does not seem to spread to any great distance, and is much weakened by dilution with air. In well-ventilated private houses typhus fever rarely spreads, and it never extends from hospitals to adjacent streets. It is more likely to pass from a low storey to a higher one, than in the contrary direction. Fomites, such as clothing, bedding, furniture, or the walls of rooms, may retain the contagion for some time if not properly disinfected, and may thus subsequently originate the disease in the same place, or be the means of conveying it to other districts. Woollen and dark-coloured materials are said to take up the poison most readily. Typhus fever is stated to be most contagious during the period of convalescence, but infection probably lasts from the end of the first week until convalescence is established. A second attack is an exceedingly rare event.

The opinion is strongly held by some eminent observers, that typhus fever may be developed *de novo*, independently of any infection, in consequence of great overcrowding and destitution; others, however, maintain that these conditions only encourage the development, and increase the virulence of disease-germs already existing.

*Predisposing causes.*—There are certain circumstances which either greatly intensify the action of the typhus contagium, or render individuals more liable to be attacked. These are:—1. A low physical condition, induced by intemperance and bad feeding, or by chronic disease. 2. Overcrowding and deficient ventilation, especially overcrowding of

dwelling-houses, or of individuals in the same house or room, along with bad ventilation. 3. Want of cleanliness, domestic and personal. 4. Mental depression, from overwork or anxiety, or fear of contagion. 5. A temperature not too high. In consequence of the action of these causes, typhus fever is infinitely most prevalent amongst the poor; in the crowded parts of large towns, especially in those places where the sanitary arrangements are inefficient; in low regions; in crowded camps or dwelling-houses, such as the lower class of lodgings; and in cold and temperate climates. Epidemics are also very apt to arise during periods of distress and famine from any cause. Typhus seems to be much more rife in Great Britain and Ireland than in other countries, and it is stated not to occur within the tropics. Mental causes appear chiefly to affect persons belonging to the better grades of society. Something may probably be attributed to individual susceptibility, certain persons being more prone to be attacked than others.

**ANATOMICAL CHARACTERS.**—The blood is much altered in fatal cases of typhus fever. It either remains fluid or forms very soft clots, and tends to decompose rapidly. The fibrin is diminished, and the red corpuscles, which are increased in number at first, afterwards become deficient. The salts are in excess, while urea and ammonia are present, the latter being supposed to result from the decomposition of the former. Under the microscope the red discs are seen to be irregular in form and crenated, and they collect in amorphous heaps. The colouring matter transudes, tinging more or less the various tissues and the fluid contained in serous cavities.

The body does not usually present much emaciation, but decomposes speedily. The maculæ on the skin which are observed during life are frequently persistent after death.

The voluntary muscles are of a dark colour and softened. Their fibres often exhibit signs of degeneration under the microscope. Sometimes they are the seat of hæmorrhages. Similar softening with fatty degeneration is observed to a marked degree in the heart. It is believed also that the same change occurs in connection with the involuntary muscular tissue generally.

There is nothing characteristic in the brain. There may be some congestion and excess of serum, and occasionally slight arachnoid hæmorrhage has been observed. In some epidemics the morbid appearances of cerebro-spinal meningitis have been described.

All the organs are commonly hyperæmic, softened, friable, and enlarged, especially the liver and spleen; the latter may be quite pulpy, but does not reach a very great size. The salivary glands are frequently inflamed, and may be the seat of suppuration or gangrene. Acute nephritis is sometimes observed. The cells of organs tend to become the seat of acute fatty degeneration.

In the alimentary canal the appearances which may be met with are redness and softening of the gastric mucous membrane; congestion or inflammation of that lining the intestines, especially the colon; and enlargement of the glands, which is particularly noticed in children. There is nothing characteristic in these appearances, nor is there ever any deposit or ulceration, such as typhoid fever presents.

There may be various lesions of the nature of *complications*. Bronchitis is very commonly observed, as well as hypostatic congestion of the lungs, which may end in hypostatic pneumonia. Sometimes ordinary acute pneumonia is present.

**SYMPTOMS.—1. Incubation-stage.**—The *period of incubation* in typhus fever is usually from nine to twelve days, but it may not go beyond six days. During this time there may be such symptoms as chilliness, general pains and malaise, restlessness, headache, and loss of appetite, but these may be entirely absent, and are not significant.

**2. Invasion-stage.**—The invasion may be singularly sudden, as I know from personal experience, and it is usually tolerably marked, but may be more or less indefinite. The disease begins either with a series of slight or moderate rigors, or with one severe and prolonged fit of shivering, followed by pyrexial symptoms. The rigors often recur for two or three days. There is a marked sense of depression and exhaustion, the patient speedily taking to his bed, and presenting an aspect of weariness and heaviness, or even of considerable prostration. General muscular pains are complained of, and the limbs tremble on movement. Nervous symptoms are prominent. These are dull frontal headache, often severe, with a feeling of heaviness in the head and throbbing; giddiness; more or less dulness of hearing, with noises in the ears; flashes of light and photophobia; sometimes an unpleasant smell; restlessness and disturbed unrefreshing sleep, though the patient is at the same time often very drowsy. The mind soon begins to wander, and becomes confused as to time, place, and surrounding circumstances and individuals, distinct delirium setting in from the fourth to the eighth day, which, however, is not constant at first, while the patient can be roused to answer questions. The delirium is most frequently of a dull and muttering character, but may be extremely active and excited at the outset, the patient being sometimes very violent. The expression is heavy and indifferent; the eyes are injected and suffused; and a more or less dusky flush covers the cheeks, the complexion having a dingy and dirty appearance.

Nausea and vomiting are sometimes present, and may be distressing symptoms. The tongue is at first covered with a thick white fur, but tends to become speedily dry and brown; it is often tremulous. There is much thirst, with total anorexia, and a disagreeable slimy taste in the mouth. The bowels are generally confined, but diarrhœa is not very uncommon, the stools, however, presenting no peculiar characters, and being usually dark. Some degree of enlargement of the spleen can often be detected.

The skin feels hot and pungent. The pulse becomes frequent, rising steadily to 100 or more, and being often large and full, but very compressible; it may be small and weak, or dicrotic. The urine is markedly febrile.

Commonly there are signs of more or less catarrh of the nasal and respiratory mucous membranes, accompanied with cough and expectoration, and some dry râles may be heard over the chest.

**3. Eruption-stage.**—Two forms of eruption are observed in typhus fever, viz.:—a *subcuticular mottling*; and distinct *maculæ* or *mulberry spots*. Usually both are present in variable proportions, but the mottling is not unfrequently observed without the spots, though the latter very rarely appear without the former. Children often do not exhibit any rash, and in them the mottling is most marked. The eruption usually appears on the 4th or 5th day, but may come out at any time from the 3rd to the 7th or 8th day. The back of the wrists, borders of the axillæ, and epigastrium exhibit it first; it then spreads rapidly over the trunk and limbs, but is rarely seen on the face and neck. *The rash*



is all out within one, two, or three days, and no fresh spots are developed after this, while each spot is perceptible until the entire rash disappears. The amount varies considerably, but the maculæ are frequently very numerous, and may cover the skin almost completely.

*Characters and Course. a. Maculæ or Mulberry Rash.*—A number of distinct spots are first observed, varying in size from mere points to two or three lines in diameter, the larger being formed by the union of smaller ones. They are irregularly roundish, the larger spots being the more irregular, and their margin is ill-defined. They are quite superficial, and at first are often slightly raised, but this elevation subsides in a day or two. The colour is described as resembling the stains of mulberry juice, being as a rule at the outset of a brightish or pinkish-red. It is deeper at the centre than at the margin, and completely disappears under pressure, returning again when the pressure is removed. In a few days the hue deepens, and may become purple or dark crimson or livid, especially towards the centre of the spots, which at the same time become more defined at their edge. This is especially observed over the back and other dependent parts. Pressure only diminishes the colour after three or four days, a light-yellow stain being left, and finally it does not affect it at all, the spots being in fact converted into true petechiæ, of an uniform hue. The eruption, viewed as a whole, has not an equal depth of colour.

*b. Subcuticular Mottling.*—This is most marked in dependent parts, and is described by Dr. George Buchanan as “a faint, irregular, dusky red, fine mottling, as if below the surface of the skin some little distance and seen through a semi-opaque medium.”

The duration of the rash varies. It usually subsides from the 14th to the 21st day. The mottling disappears more readily and sooner than the spots, and the latter remain longer if they become petechial. No desquamation follows the disappearance of the eruption.

The skin of dependent parts is more or less congested, especially that of the back. Miliary vesicles or sudamina may appear about the end of the second week, usually over the groins, subclavicular regions, or epigastrium. A peculiar odour is given off from the skin of persons suffering from typhus.

During the eruption-stage most of the symptoms previously existing become worse, and tend to be of a low, adynamic or typhoid character. The headache, however, usually subsides on or before the 10th day, and if it should continue along with marked delirium, this is a sign of danger, indicating some cerebral complication. Debility and prostration become very marked, the patient lying helplessly on his back, with the eyes closed or half-closed, in a state of muttering delirium, from which it is difficult or impossible to rouse him. Somnolence often sets in, which may be followed by complete stupor and coma. Muscular twitchings and trembling, rigidity, and picking at the bed clothes are frequently observed, and occasionally convulsions with strabismus. Sometimes coma-vigil is noticed, the patient lying with the eyes wide open, apparently awake, but staring vacantly into space. The complexion becomes muddy-looking, and a more dusky flush covers the face, which may be almost livid. The conjunctivæ are extremely injected and suffused, the pupils being often contracted. The skin of the extremities becomes cold and perspiring. The tongue is dry, brown and cracked, or frequently even covered with a thick blackish crust, and immovable; its surface is red and tends to bleed; while sordes cover

the lips and teeth. Patients usually drink with avidity, but deglutition is difficult. The nostrils are stuffed up. Tympanites is sometimes a prominent symptom. The pulse rises to 120, 140, 150, or more, but remains stationary after reaching a certain point; it becomes small and weak, and may be irregular. The heart's impulse and sounds are feeble, especially the systolic sound; and capillary stasis is very liable to arise. Respiration is much hurried and disturbed, being also frequently unduly abdominal. The breath has a peculiar and most unpleasant odour. Physical examination of the chest reveals bronchitic râles, or more serious complications may be detected. Incessant hiccup is sometimes a distressing symptom.

The urine is not uncommonly albuminous, or contains a little sugar; it may be retained or passed involuntarily along with the stools. Bed-sores are very liable to be produced over parts which are pressed upon.

The severity of the symptoms of typhus fever varies much in different cases, but if a case goes on to a fatal issue, prostration becomes more and more complete, the heart's force is exhausted, and the nervous symptoms indicate that the nerve-centres are still more disturbed. Before death the temperature may rise or fall rapidly, and in some instances the pulse suddenly falls. Complications may arise to hasten the fatal result.

4. **Stage of Defervescence.**—This sets in in cases of recovery from the 13th to the 17th day, generally at the end of the second week. There is a remarkable and sudden *crisis*, which often occurs at night, the patient falling into a deep sleep, lasting for many hours, on awaking from which a wonderful improvement is observed in his aspect and in the symptoms generally. The temperature falls considerably, as well as the pulse, which gains in strength. The skin is soft and perspiring, the eruption less marked, and the complexion clearer. The tongue becomes moist, and cleans from the edges, either in patches or molecularly, and some inclination for food may be felt. Delirium ceases, the patient recognizes those around, but the mind is still confused, and entirely unconscious of all recent events. There is a sense of extreme weakness, and the limbs feel as if they did not belong to the body. Unless complications or sequelæ impede the favourable progress, convalescence begins at once, and the strength is regained comparatively rapidly, but it is some time before this is completely restored. The tongue soon cleans, and the appetite becomes perfectly ravenous; only those who have experienced the feeling can realize the extreme sense of hunger which is felt. Much sleep is indulged in, and the mind does not regain its normal vigour for some time. A relapse of typhus is extremely rare.

**Temperature.**—Different observers have described different ranges of temperature in typhus fever, and this appears to depend partly upon the nature of the epidemic. The *ascent* is steady and continuous up to the 4th or 5th evening, without any morning remission. The maximum temperature is rarely under  $104.9^{\circ}$  to  $105^{\circ}$ , often reaching  $107^{\circ}$ , or even above this. It may rise to  $105^{\circ}$  on the 3rd or 4th evening in severe cases; in slighter cases it may not be above  $103^{\circ}$ . A slight morning remission is observed on the 6th morning, and a well-marked fall occurs on the 7th day, unless the case is very severe. After this a rise takes place again, but rarely to the former maximum. In fatal cases, however, it may go up to  $108^{\circ}$  or  $109^{\circ}$ . The temperature is *continuous* up to the *period of defervescence*, with a distinct but not considerable morning

remission. This is more marked in cases where the temperature is high, and may average from  $100^{\circ}$  to  $101\frac{1}{2}^{\circ}$ . Dr. Buchanan states that it ranges from  $100^{\circ}$  to  $101\frac{1}{8}^{\circ}$  until the middle of the 2nd week, and is afterwards about  $101\frac{1}{2}^{\circ}$ . Defervescence is very rapid and sudden, setting in from the 13th to the 17th day, and the temperature may fall to or below the normal in 12, 24, or 48 hours. This event is often preceded by a rise above the temperature of the previous day. Occasionally after the sudden fall there is a rise of  $2^{\circ}$  or  $3^{\circ}$ , and then defervescence extends over some days; in short, a combination of *crisis* and *lysis* is observed. As already mentioned, in fatal cases there is frequently a rapid elevation or sinking of temperature, and it may reach  $109^{\circ}$  on the one hand, or  $95^{\circ}$  on the other.

Some observers have found a relation between the temperature and pulse in typhus, but this is by no means constant or uniform, and the one may be high while the other is low.

VARIETIES.—Cases of typhus fever present considerable differences as regards their intensity and their prominent symptoms, to which special names have been applied. The *nervous*, *circulatory*, or *respiratory* system may appear to be most implicated. In some epidemics there has been a great tendency to gangrene, hence named *putrid fever*. Typhus fever may kill in a few days, by the direct action of its poison upon the system, before any local lesions have been developed. Niemeyer described mild cases, in which the earlier symptoms of typhus occurred, without any eruption or enlargement of the spleen, and in which convalescence set in at the end of a week.

COMPLICATIONS AND SEQUELÆ.—These should always be looked for and guarded against, as they may arise without any evident symptoms. The most important are:—1. Affections of the respiratory organs, viz., bronchitis; pulmonary hypostatic congestion or consolidation; pneumonia; gangrene of the lung (very rare); pleurisy; phthisis; laryngitis with œdema glottidis. 2. Affections of the circulatory organs and blood, including cardiac softening and degeneration; phlegmasia dolens; scurvy. 3. Partial paralysis, as a sequela, which is usually soon recovered from. 4. Dysentery in some epidemics. 5. Gangrene of the toes, nose, and other parts, especially during the winter; or cancrum oris in children. 6. Erysipelatous affections of the skin, throat, or deep tissues ending in suppuration. 7. Suppurative inflammation or bubos of the parotid or submaxillary glands, beginning in the cellular tissue around. 8. Inflammation and abscesses in various parts of the body. 9. Suppurative inflammation in joints. 10. Acute nephritis, which may terminate in chronic renal disease.

TERMINATIONS AND DURATION.—Most cases of typhus fever end in recovery. The *mortality* varies in different epidemics, but the average number of deaths is stated to be about 1 in 5. The average *duration* is about 14 days, but may extend to 21 days; if it is beyond this, the prolonged course is due to complications. On the other hand, the disease may run a much shorter course. Death may result either from coma or cardiac failure, or, most commonly, from both causes combined; or it may be due to complications.

PROGNOSIS.—This is always grave in typhus fever, and a very guarded opinion should be given. The chief general circumstances which increase the danger of any particular case are as follows:—1. The patient being of middle or advanced age. 2. The male sex to some degree. 3. A low condition of the system, whether constitutional, or due to privation or fatigue, intemperate habits, previous diseases, or other causes. The



presence of the gouty diathesis is highly dangerous. 4. Mental depression, and a presentiment of death on the part of the patient. 5. Improper hygienic conditions, especially bad ventilation and overcrowding. 6. Neglect of proper treatment until a late period.

The *symptoms* and *complications* present afford most important indications as regards prognosis. Those of unfavourable import are:—1. Extreme prostration, with a dry, hard, and brown tongue; marked tympanites; or persistent hiccup. 2. Excessive feebleness of the heart's action, as evidenced by its impulse and sounds, and by the pulse; or very excited action, with a weak pulse; or an exceedingly frequent pulse, which is at the same time extremely feeble, irregular, or intermittent. 3. Severe and early cerebral and other nervous symptoms, especially continued sleeplessness with delirium; deep coma or *comavigil*; muscular tremors, twitchings or rigidity, carphology, subsultus tendinum, *convulsions*, early relaxation of the sphincters, strabismus, and *great contraction of the pupils*. 4. A very high temperature, without any remission on the 7th day, especially if it persists and shows a tendency to rise; or a *sudden fall*, the other symptoms not improving. 5. A large amount and dark colour of the eruption, especially if mingled with numerous petechiæ, and if there is lividity of the face and limbs, with marked congestion in dependent parts. 6. Suppression or retention of urine; deficient elimination of its solid ingredients; the presence of much albumen or of blood in the urine, especially if associated with casts; and particularly the early occurrence of these symptoms. 7. Signs of collapse. 8. Pulmonary inflammation, gangrene, erysipelas, and other dangerous complications.

**TREATMENT.**—The principles already laid down with regard to the treatment of *fever* are those which must be followed in the management of cases of typhus. There is no possibility of checking the disease in its course, and all that can be done is to avert the tendency to death, and to assist nature towards a satisfactory termination.

1. **General management.**—It is most important to attend strictly to all the *hygienic conditions* which demand attention in contagious fevers, and particularly to look after every point connected with the sick-room, including the nursing. These matters are often of greater moment than any medicinal treatment, and always aid considerably in conducting a case to a successful issue, while they prevent the dissemination of the disease.

It may be laid down as an invariable rule of practice that patients suffering from typhus fever *will not bear any kind of lowering treatment*, but that they always need to be supported more or less, and their strength must be husbanded in every possible way. They should take to bed *at once*, and use no exertion whatever, on no account being permitted to get up to stool, but a bed-pan being provided for their use. From the first a nutritious and easily assimilable *diet* must be administered, chiefly consisting of liquids, such as milk, beef-tea, or chicken-broth. It is essential to give these at regular intervals, in considerable quantity, and *not to neglect them during the night*.

*Alcoholic stimulants* are required in the great majority of cases, but they ought not to be given recklessly, the nature and quantity of the stimulant to be employed being determined by a careful consideration of each individual case. Port or sherry wine, or some spirit, especially brandy, answer best as a rule, and they should be given in stated doses at regular intervals, by night as well as by day. It is well to begin

with a small quantity, gradually increasing it as circumstances require, the amount being again reduced as the symptoms improve. Stimulants are not usually called for during the first few days, but in the case of the aged, the intemperate, and those who are much debilitated from any cause, they are demanded at the very outset. The signs which chiefly indicate the necessity for alcohol are:—1. A feeble state of the circulation, as shown by the pulse, the heart's impulse and sounds, a tendency to capillary stasis, or any disposition to syncopal attacks. 2. The existence of typhoid symptoms, the amount needed being usually in proportion to the severity of these symptoms. 3. A large amount and dark colour of the eruption, with abundant petechiæ. 4. Profuse perspiration, the other symptoms not improving. 5. Coldness of the extremities. 6. The existence of complications of a low type. On the other hand, alcohol is contra-indicated or requires to be cautiously administered should there be a very hot and dry skin; symptoms of much cerebral excitement; or conditions of the urine pointing to deficient elimination on the part of the kidneys. In all cases the propriety of continuing or increasing the amount of stimulants must be judged of by the effects produced.

**2. Therapeutic treatment.**—If a case of typhus is seen at an early period many recommend the administration of an emetic. The bowels should be kept open daily by some mild aperient, or by the use of simple enemata. In order to maintain free elimination, it is advisable to allow the patient plenty of drink, which may contain in solution citrate of potash, nitre, cream of tartar or chlorate of potash. Tea, coffee, and salt are also recommended to be given freely for this purpose. The medicines which have obtained most repute in the treatment of typhus are the *dilute mineral acids*. Nitric, hydrochloric, nitro-hydrochloric, sulphuric, and phosphoric acids are all employed. Either of these may be made into a drink, or given in doses of  $\text{m} \times \text{xxx}$  every three or four hours, along with tincture of bark. Sulphuric acid answers best when typhoid symptoms set in. Quinine in moderate doses is another valuable remedy, and may be very advantageously combined with one of the mineral acids. Tincture of iron has also been highly recommended.

Various *antiseptics* have been tried, such as carbolic acid, sulphocarbolates, creosote, sulphites, Condy's fluid, and peroxide of hydrogen, but it does not appear that they can be relied upon.

**3. Symptomatic treatment** often demands considerable attention in cases of typhus fever. The symptoms which are likely to call for interference are excessive heat of skin; nausea and vomiting; thirst; constipation or diarrhœa; and head-symptoms, namely, headache, sleeplessness, delirium, stupor, or coma. Hiccup sometimes causes much distress. For the relief of this symptom the best remedies are sal volatile, ether, spirits of chloroform, hydrocyanic acid, camphor, and musk, in various combinations. A sinapism may be applied over the epigastrium; or the ice-bag may be tried if necessary.

Should there be a tendency to marked prostration, it is necessary to administer diffusible *stimulants* freely, such as sulphuric or chloric ether, camphor, musk, and carbonate of ammonia, along with alcohol. Sometimes patients become so low that they cannot swallow, and then recourse must be had to nutrient and stimulant enemata, which should be persevered in to the last; or even subcutaneous injection of ether may be resorted to in extreme cases. It is always very important to look to the bladder, and to draw off the urine if necessary.

4. The various **complications** met with must be watched for, and every care taken to prevent them, especially *pulmonary complications* and *bed-sores*. As regards the treatment of inflammatory affections, it must be borne in mind that *stimulants* and *tonics* are indicated when such complications supervene during the course of typhus.

5. Much care is requisite during the stage of **convalescence**, all over-exertion being avoided, as well as excessive eating. Tonics and change of air are highly beneficial at this time. It is especially necessary to guard against any sudden effort during the early period of convalescence, as this is liable to cause coagulation of blood in some of the principal veins of the lower extremities. Any sequelæ which may arise must, of course, be attended to, and treated by appropriate measures.

---

## CHAPTER VI.

### TYPHOID OR ENTERIC FEVER—PYTHOGENIC FEVER— ABDOMINAL TYPHUS.

**ÆTIOLOGY.**—Typhoid fever originates from a *specific poison*, which is quite distinct from that causing typhus. Formerly they were looked upon as identical diseases, and a few still hold to this opinion, but without any adequate reason. Klebs, Eberth, Koch, Coats, Crooke, Bouchard and others have attributed the disease to specific *typhoid bacilli*. Sokoloff and Fischel observed similar organisms in the blood of the spleen after death; and Maragliano of Genoa has stated that he has found them in blood taken from the spleen during life, as well as in that of the general circulation. Micrococci have also been described. Guerin maintains that the toxic element in enteric fever results from the fermentation of faecal matters, retained and accumulated at the end of the small intestine, behind the ileo-cæcal valve.

There is abundant evidence to prove that typhoid fever is infectious, and when once it finds its way into the midst of a number of individuals it tends to spread amongst them. It is most important, however, to understand clearly how the disease is mainly conveyed. The exhalations do not appear to contain the contagium, and there is very little danger from merely coming into the vicinity of patients suffering from typhoid. Indeed, the probability is that the malady cannot be transmitted in this way, and medical men or nurses rarely take it from attending upon patients. It is in the *fæces* that the poison is chiefly contained, and by their agency the disease is propagated. The atmosphere may become impregnated with the emanations from the excreta, either because the latter are thrown into some open space, or because the water-closets, privies, sewers, &c., are imperfect, and undoubtedly the poison may thus find its way into the system by inhalation of the tainted air. *Water* is, however, the great channel by which it is conveyed, and numerous epidemics and endemics, as well as sporadic cases of typhoid fever, have been traced to some special water-supply. The materials may soak through the soil from cess-pits, or in consequence of being



merely thrown upon the ground, thus obtaining access into wells, the water of which is used for drinking purposes; or they may find their way into cisterns through the waste-pipes. The occurrence of typhoid fever is very often traceable to defective sanitary arrangements in particular houses. It has also been clearly proved that *milk* is not uncommonly the vehicle by which the typhoid poison reaches the system, either in consequence of water containing it being mixed with the milk, or used for washing milk-cans; or from this article of diet becoming tainted in some other way with the excreta of patients suffering from the fever. The opinion has been advanced that typhoid may be communicated through drinking the milk of cows fed on soil containing much sewage matter; or from eating the flesh of animals suffering from the disease. An outbreak of enteric fever in Germany was attributed to eating veal thus infected. Fomites may convey the disease, but only if they should become contaminated with the typhoid stools.

The name "pythogenic" was given to typhoid fever by the late Dr. Murchison, who, with others, was strongly of opinion that this complaint is as a rule *spontaneously* originated in connection with ordinary sewer emanations and putrefying animal matter, and he believed that even in the specific stools the poison is always a product of decomposition. That it can be thus spontaneously originated seems by no means improbable, at any rate when water is contaminated with sewage-matter, or with the gases emanating therefrom; but, as a rule, cases can be traced distinctly to the action of the typhoid poison. Typhoid has been attributed to other sources of decomposing organic matter, and even to recently-exposed mud.

It is important to recognize the fact that the typhoid-contagium in the *faeces* becomes more virulent after a time, and some even think that when first discharged it is comparatively, if not quite, harmless. Its amount and virulence are increased by certain conditions, namely, stagnation, accumulation, and concentration of the infected *faeces*; seclusion from open air; and a certain degree of heat. A kind of fermentation takes place, and hence a very small proportion of a typhoid evacuation may lead to the infection of large quantities of excreta; the same process is believed to occur in milk.

*Predisposing causes.*—Age materially influences the occurrence of typhoid fever. It is by far most common during youth and adolescence, being very rare in young infants, and in persons beyond 45 or 50 years of age. Individuals under 30 are twice as liable as those over 30, and half the cases occur from 15 to 25 (Murchison). Sex does not seem to have any effect. Cases are most numerous during autumn, especially after a dry and hot summer, and the facts mentioned above explain this. Over-crowding is not a predisposing cause of typhoid, but deficient ventilation may have some influence. The disease attacks persons in all classes of society, and is not at all more prevalent among the poor; if anything, the contrary is the case. Individual susceptibility is believed to predispose, and some families are very liable to the disease, and to have it in a severe form. Recent comers into an infected district are said to be most liable to be affected. Persons in good health are often attacked before others; but overwork, mental depression or shock, and debility from any cause, are said to predispose. Various chronic and acute diseases, as well as pregnancy, seem to afford some protection against typhoid fever. Habitual use of impure water has, in some instances, rendered epidemics of typhoid fever very severe; in other

cases it has almost appeared to confer an immunity from the complaint.

**ANATOMICAL CHARACTERS.**—The general condition of the body in fatal cases of enteric fever will vary with the time at which death occurs. Usually there is more or less emaciation; rigor mortis is distinct and of moderate duration; while the excessive *post-mortem* congestion, rapid putrefaction, and dark colour with softening of the muscles, characteristic of typhus, are not observed in typhoid. The muscles, however, undergo more or less granular degeneration. The eruption is not persistent after death; but there may be the remains of bed-sores, gangrene, erysipelas, or sudamina.

**Alimentary Canal.**—It is here that the most important morbid changes are found in enteric fever. The pharynx and œsophagus may be congested, inflamed, covered with diphtheritic deposit, or the seat of ulceration. The ulcers are generally very superficial, and are never met with before the third week. They are not the result of any morbid deposit. The stomach occasionally presents hyperæmia, mamillation, softening, or superficial ulceration, but is usually normal.

The small intestines are only rarely distended with gas; but they contain more or less of the materials similar to those passed in the stools. Increased vascularity of the mucous membrane may be observed, either uniform or in patches, but this is by no means necessary; it is most evident towards the lower part. At a later period the colour may be greyish or slate-coloured. The membrane is sometimes swollen and softened, the latter being probably a *post-mortem* change.

The characteristic lesions of enteric fever consist in certain morbid changes in connection with *Peyer's patches* and the *solitary glands*. These structures present different appearances, according to the time at which death occurs, the morbid changes being divided into certain well-defined stages.

1. *Stage of deposition or enlargement.*—The first alteration observed is an enlargement of Peyer's patches and of the solitary glands, owing to the presence of a morbid substance, supposed by some to be of the nature of a specific deposit from the blood undergoing a peculiar development, but generally considered to be the result of a proliferation of the cell-elements previously existing. The material is made up chiefly of granular matter and oil-globules, with a variable number of cells, having no special characters. It is probably first formed within the glandular sacs, but these may burst and discharge their contents into the surrounding cellular tissue, or there may be an increase of cells here also.

It is a matter of doubt at what period the glandular enlargement commences, and whether it is preceded by hyperæmia. Murchison states there is no previous congestion, and that a deposit has been met with on the 1st or 2nd day, but Trousseau gives the 4th or 5th day as the time of its appearance.

Peyer's patches appear to be unduly prominent, being raised one or two lines or more above the level of the mucous membrane, and having steep edges, with a smooth or granular surface; they are more or less firm, though the membrane covering them is often softened; while they vary in colour from pinkish-grey to different hues of red, the mucous membrane over them being sometimes purplish, and each patch is surrounded by a vascular ring. The corresponding peritoneum is also injected. The substance seems to be adherent to the mucous and

muscular coats. On section it appears as a soft, greyish-white or pale-red material.

Two forms of patches are described, the *plaques molles* and the *plaques dures*, but there are gradations between them, and they may coexist. The latter are more prominent and firmer, and the membrane over them is smoother and more uniform. The differences between the two kinds are, that in the *plaques molles* the deposit is less abundant, and is confined to the glands, which in the *plaques dures* have burst and discharged their contents (Murchison).

The solitary glands are not always involved. On the other hand, in exceptional instances they may be alone affected. They vary in size from a millet-seed to a pea, and sometimes look like pustules.

2. *Stage of destruction*.—In some instances it appears that a kind of resolution takes place, and the material is absorbed, without any breach of surface occurring; this may happen in patches high up in the bowel, while those lower down are ulcerating. Almost invariably ulceration takes place, the average time at which this process begins being about the ninth or tenth day, but it may commence much earlier or later than this. Each ulcer is commonly the result of the death of a Peyer's patch and of the membrane covering it, the whole separating as a single slough or in separate irregular portions. These sloughs are usually yellowish or yellowish-brown from staining, or they are sometimes discoloured by blood; they are occasionally seen in process of separating, hanging loosely. There may be mere superficial abrasion and softening of the mucous membrane preceding ulceration. Sometimes the glands simply rupture and discharge their contents, giving rise to a net-like appearance, and Aitken believes that this is the most frequent mode of elimination of the softened deposit. The solitary glands undergo a similar destructive change; and it may also spread to the mucous membrane between the glands.

3. *Stage of ulceration*.—The characters presented by *typhoid ulcers* are as follows:—In length they usually vary from a line to  $1\frac{1}{2}$  inch, but if several ulcers join a surface of some inches may be involved. The shape is oval or elliptical, round, or irregular, according as the ulcer corresponds to a Peyer's patch, to a solitary gland, or to several patches or glands united. There is never any thickening or hardening of the edges or floor, nor is any morbid deposit observed here. The margin consists of a "well-defined fringe of mucous membrane, detached from the submucous tissue, a line or more in width, and of a purple or slate-grey colour: this is best seen when the bowel is floated in water" (Murchison). The floor is formed either by the submucous, muscular, or peritoneal coat, the ulcer accordingly varying in depth. Those ulcers which correspond to Peyer's patches occupy the part of the intestine most distant from the mesentery, and their long diameter is longitudinal, and not transverse, as regards the direction of the intestine.

4. *Stage of cicatrization*.—This stage commences usually about the end of the third week, but may be delayed considerably beyond this period, the ulcers becoming chronic or atonic. Each ulcer takes about a fortnight to cicatrize. Healing is accomplished without any puckering, contraction, or constriction of the gut. A thin transparent layer of lymph forms on the surface of the ulcer, by which the mucous membrane becomes gradually attached to its floor from the periphery to the centre, and into which at last it passes imperceptibly. The cicatrix is slightly depressed, thinner at the centre than at the circumference, pale,



smooth, and translucent. After a time the mucous membrane may become movable, and it is said that villi may form, but if the glands are destroyed it is very unlikely that these ever become renewed.

The changes above described begin first, and are most extensive and most advanced in that portion of the intestine in which Peyer's patches are most evident, namely, in the lower portion of the ileum, and from this point they gradually extend upwards, until ultimately they may occupy the lower third of the small intestine. The extent of the disease is, however, very variable, the number of patches involved ranging from 2 or 3 to 30 or 40. Generally at a *post-mortem* examination different stages of the morbid process are visible in different parts, it being most advanced below, where the most extensive ulcerations are observed. At the upper part there is usually a somewhat abrupt transition from diseased to healthy patches, and all those below the first diseased patch are generally involved. The solitary glands are, as a rule, only implicated in the lower 12 inches of the gut, and they ulcerate later than the patches. Occasionally the small circular ulcers associated with these structures constitute the chief or only lesion, especially when Peyer's patches are absent. They are more liable to be attacked in children.

*Intestinal perforation* is an event to be dreaded in connection with typhoid ulcers. It may be brought about in the following ways:—1. Most frequently by molecular disintegration or an extension of ulceration, producing one or more minute, round apertures, like pin-holes. 2. By more or less extensive sloughing, involving the peritoneum, the slough separating partially or completely, and leaving an opening of variable size. 3. By rupture or laceration, leading to an elongated perforation, and this may happen even after cicatrization has been completed. Generally there is but one perforation, but occasionally two, three, or more have been observed. In most cases the opening is in the lower portion of the ileum, but it may be higher up, or in the large intestines.

If a case of typhoid fever has a very prolonged duration, it is said that the coats of the intestine, as well as the glandular structures, become considerably atrophied.

*Large intestines.*—These are usually distended with gas, sometimes to an extreme degree. The mucous lining may be congested or softened. Deposit and ulceration not unfrequently occur in connection with the solitary glands, these morbid changes being, as a rule, limited to the cæcum and ascending colon. The ulcers are generally small and circular, but may be  $1\frac{1}{2}$  inch long, with the long diameter transverse. In one fatal case which came under my notice the morbid appearances were observed chiefly in the cæcum and ascending colon, where there were at least twenty ulcers, some as large as half-a-crown, while in the small intestines there were not altogether above half a dozen, and these were confined to the solitary glands, Peyer's patches presenting but little alteration. I have since met with a somewhat similar case.

**Absorbent Glands.**—The *mesenteric* glands always present important changes, these being associated with the morbid conditions in the intestines, and being most marked in those glands which correspond to the part of the gut most diseased. They become enlarged at the very outset, not merely as the result of irritation, but from an increase in their lymphatic elements, similar to that which occurs in the intestinal glands. They continue to enlarge until from the tenth to the fourteenth day, present a red or purplish colour, and feel tolerably firm. On section, little opaque, pale-yellow, friable masses are sometimes seen. Subse-

quently these may soften into a pus-like fluid mixed with sloughs, and the glands in rare instances actually burst into the peritoneum. After the softening process begins in the intestinal glands, the mesenteric glands also soften and become smaller. Ultimately they frequently become tough, contracted and shrivelled, pale or of a grey or bluish colour, and occasionally they calcify. The *mesocolic* glands are similarly altered when the colon is involved. Other glands may enlarge from irritation.

**Spleen.**—This organ is almost always much enlarged, especially in young persons, very dark in colour, and softened. Sometimes it contains opaque yellowish-white masses. It may be quite pulpy, and has been known to rupture.

**Liver and Gall-bladder.**—The liver is sometimes congested or softened. Its cells always undergo more or less granular degeneration, and in severe cases this is very marked. The gall-bladder may be the seat of catarrhal or diphtheritic inflammation, or of ulceration. After three or four weeks the bile is often thin, watery, colourless, and acid in reaction.

**Peritoneum.**—Peritonitis may occur in typhoid fever, either extensive or circumscribed, limited abscesses sometimes forming. It may arise from mere extension of irritation from the bowel; from intestinal perforation; from rupture of glands or of the spleen; or from perforation of an ulcer in the gall-bladder.

**Urinary Organs.**—The kidneys are sometimes congested; or they may have their tubes choked up with detached epithelium. Granular degeneration of their gland-cells occurs in various degrees. The mucous coat of the bladder may be congested or inflamed.

**Blood and Organs of Circulation.**—The blood is dark and fluid, and does not coagulate, if there have been typhoid symptoms before death, but these characters are rare in typhoid fever as compared with typhus. The white corpuscles are increased, and disintegrating red corpuscles are sometimes seen. The heart is more or less softened, and presents granular degeneration of its fibres, which in grave cases becomes a marked change, and may lead to fatal syncope.

**Respiratory Organs.**—There may be congestion, various forms of inflammation, œdema, or ulceration of the larynx, the last-mentioned not being due to any specific deposit. Signs of bronchitis, hypostatic pulmonary congestion or œdema, pneumonia, or pleurisy may be evident. The bronchial glands are sometimes enlarged.

**Nervous System.**—This presents no particular alterations. There may be excess of serum in connection with the brain and its membranes.

**SYMPTOMS.**—1. **Incubation-stage.**—The *period of incubation* in typhoid fever is of doubtful duration. It most frequently extends beyond ten days, and may be much longer than this. There are no distinctive symptoms. Sometimes the incubation-period appears to be very short if the poison is concentrated, the disease setting in with vomiting and purging, attacking a number of persons at the same time, and giving rise to a suspicion of irritant poisoning.

2. **Actual attack.**—It is scarcely possible to divide this affection into distinct stages, but at the same time it often presents periods in its progress, which are marked by tolerably characteristic phenomena. The *invasion* is ordinarily *very indefinite and gradual*, and the patient cannot fix the exact date of the commencement of the attack. Frontal

headache, with giddiness and noises in the ears ; general pains in the limbs, with a feeling of lassitude and illness ; restlessness and disturbed sleep ; slight, irregular chills ; diarrhœa, with loss of appetite, furred tongue, and not uncommonly nausea and vomiting, are the ordinary symptoms at the outset. Sometimes there is much abdominal pain. Diarrhœa may be the only prominent symptom for some time. The tongue may be quite clean for some days, even when pyrexia is high. Occasionally repeated epistaxis occurs. Soon signs of pyrexia appear, increasing towards evening. It frequently happens that the patient does not feel sufficiently ill to take to bed for some days, but follows his occupation, and it is not an uncommon event for patients to come to the out-patient room of a hospital after having been poorly for many days, thinking that there is not much the matter with them, while they sometimes walk about during the entire illness. It is extremely important, from a diagnostic point of view, to bear in mind this ill-defined character of the onset of enteric fever, and to be prepared to look for the disease when its symptoms are anything but characteristic. Murchison met with cases in which at first the symptoms resembled those of ague.

*Early stage.*—The disease being established, the symptoms present during the first week or ten days are as follows :—

The general appearance does not indicate any great prostration, and although a certain degree of depression is felt, it is not very marked. The expression presents nothing peculiar, and the face is normal in colour, or pale, or a pink circumscribed flush may be noticed on one or both cheeks, varying in depth of tint, and not constant. There is pyrexia, the skin being hot and usually dry, but sometimes moist ; while the pulse is accelerated to 100 or 120, and somewhat weak and soft ; it varies in frequency in the same patient, being readily quickened, and it is generally more rapid at night. The tongue presents usually a thin whitish or yellowish fur, is moist at first, small and pointed, red at the tip and edges, with large papillæ. In exceptional cases it is large and thickly coated ; or red, smooth, and glazed. The lips are parched and dry, and the mouth feels slimy. There is more or less thirst, with loss of appetite, and not uncommonly nausea and vomiting.

Abdominal symptoms are prominent as a rule. These are pain and tenderness, especially in the right iliac fossa ; more or less flatulence or tympanitic distension ; small gurgling on pressure in the right iliac fossa ; and diarrhœa. Physical examination reveals enlargement of the spleen. Sometimes intestinal hæmorrhage occurs. The diarrhœa varies considerably in severity, the stools numbering from two to twelve, twenty, or more within the twenty-four hours. Usually they range from three to six. At first the fæces present no peculiarities, but after a few days they assume special characters, becoming thin, yellow, pulaceous, and somewhat resembling "pea-soup" in appearance ; very offensive, and often ammoniacal ; and alkaline in reaction. Uniform throughout when first passed, they separate on standing into an upper watery layer, of a yellowish or brownish colour, containing albumen and salts in solution, the latter including chloride of sodium and carbonate of ammonia ; and a lower layer or deposit, consisting of the remains of food, epithelium and mucus corpuscles, blood, small yellow flocculi, shreds of slough, and crystals of triple phosphates.

Head-symptoms are not very marked at this time. Frontal headache persists, with dizziness and buzzing in the ears. Sleep is restless and disturbed, but the mind is clear, though inactive, and there is no



delirium even at night. Epistaxis is not an uncommon symptom during this period.

The urine presents well-marked febrile characters; urea and uric acid are in excess; and chloride of sodium is diminished.

Frequently there are slight bronchitic symptoms, dry râles being also heard over the chest.

*Eruption.*—A specific eruption is present in the great majority of cases of enteric fever, but not invariably. It is not unfrequently absent in very young patients, and in persons over thirty. It first appears usually from the 7th to the 12th day, but may in rare instances be seen as early as the 4th, or not until the 20th day. The abdomen, chest, and back are the regions which it generally occupies, but it is occasionally observed on the limbs, especially the thighs, or very rarely on the face. It does not appear all at once, but comes out in successive crops, each spot lasting from two to five days, and then fading away completely. The amount of eruption present at one time is never great, the number of spots rarely exceeding from 12 to 20 or 30, and there may be but two or three. They continue to come out often until the 28th or 30th day, or sometimes even much later than this. Murchison found the average total duration of the eruption to be about  $14\frac{1}{2}$  days. It appears earlier, and lasts a shorter time in children.

The typhoid rash consists of separate spots, which are round, lenticular, or oval in shape; and vary in diameter from  $\frac{1}{2}$  a line to 2 lines. They are slightly but distinctly elevated as a rule; rounded on the surface; having a well-defined margin, and a soft feel. They present a pinkish or rose colour, which throughout their whole course disappears completely on pressure, and which gradually fades away. They never become petechial. In very rare instances the spots are minutely vesicular. They never persist after death.

*Advanced stage.*—The symptoms thus far described may continue without any particular change until convalescence sets in, the tongue remaining moist throughout, and there being no marked prostration or severe nervous symptoms. Usually, however, the phenomena change more or less. The patient emaciates and becomes much weaker, being sometimes very prostrate at last. When the pectoral muscle is gently tapped with the end of the finger, it sometimes rises in a small swelling—*myoidema*, lasting from twenty to thirty seconds, due to contraction of the degenerated muscular fibres. The face is more flushed, the conjunctivæ may be injected, and the pupils dilated. The fever continues; and the pulse becomes more frequent, but weaker. The heart's action and sounds are feeble. The tongue tends to become dry and brown, or red, shining, and deeply fissured; while sordes collect on the lips and teeth, and the breath has a very disagreeable odour. Labial herpes is not uncommonly observed. There is no diminution in the abdominal symptoms, which indeed are often intensified; and hæmorrhage from the bowels is liable to occur at any time, and may be very abundant. Sometimes the stools are passed involuntarily. The spleen also becomes larger.

The nervous symptoms undergo a marked change. From the 10th to the 14th day the headache and general pains cease, but there is more giddiness, with deafness. The mind also becomes affected, as indicated by more or less somnolence, mental confusion, or delirium. The latter is at first only nocturnal, but may become continuous, though it is usually worse at night, while drowsiness is more marked by day.

The delirium is generally of an active, noisy, and talkative kind at the outset, and may be very violent, the patient throwing off the bed-clothes, trying to get up constantly, and having various delusions. Sometimes the patient lies in an apathetic state, with half-closed eyes, appearing to understand what is said and done, but unable to make intelligible replies. Epistaxis is not uncommon at this time.

Sudamina may appear, usually in the third or fourth week, especially over the chest and abdomen, and on the sides of the neck. Bed-sores are liable to form in parts which are pressed upon.

The respirations become hurried and shallow, and there are more marked signs of bronchial catarrh; while air enters imperfectly into the lungs, and there is a danger of hypostatic congestion. The urine becomes more abundant, lighter in colour, and of lower specific gravity; while slight albuminuria may set in, but is not very common. Sometimes the urine is retained, or is passed involuntarily with the stools. Rarely it contains blood, renal epithelium, or casts.

In some cases the ordinary symptoms characteristic of the *typhoid state* are developed, petechiæ at the same time occasionally making their appearance, but this is an unusual course of events.

When typhoid fever ends in recovery it presents a *gradual* subsidence of the symptoms, defervescence taking place by *lysis* and not by *crisis*. Convalescence makes slow progress, and is liable to be retarded by one or more relapses, as well as by complications or sequelæ.

**Temperature.**—Typhoid fever presents some very characteristic features as regards its temperature. The *ascent* is quite regular and gradual, and continues for four or five days. *The evening temperature is about 2° higher than that of the morning, and there is a remission each morning of about 1° compared with the previous evening, so that there is a daily rise of about 1°, and at last the evening temperature comes to be from 103·5° to 104°.* This mode of ascent is quite distinctive of typhoid.

The *stationary* period varies greatly in duration, according to the severity of the case. The temperature ranges usually between 104° and 106° in the evenings, and only a *slight morning remission* is observed. It may reach 107°, 108°, or even above this.

The *decline* is also peculiar. Defervescence takes place gradually, and is first indicated by a more distinct morning remission; in three or four days the evening temperature falls, and the morning remissions become very considerable, a difference of 2°, 3°, or even more being observed. The time taken to reach complete defervescence, so that the evening temperature is normal, varies much. Complications and sequelæ also not uncommonly lead to irregularities; and a relapse may cause the temperature again to rise in the same regular manner as at first.

**VARIETIES.**—Remarkable differences are observed in cases of typhoid fever, both as regards their degree of severity and the prominent symptoms which they present. There may be no abdominal or other characteristic symptoms from first to last; while the abdominal symptoms bear no necessary proportion to each other. Instead of there being diarrhœa, constipation may be marked throughout; and I have more than once had a series of cases in succession under my care in which this symptom required regular treatment.

Murchison divided typhoid fever into the following varieties:—

1. The **mild form**, under which would be included the *abortive* variety of certain writers, which ends in the second or at the beginning

of the third week ; as well as some cases considered to be of the nature of simple febricula.

2. The **grave form**, which, according to the prominent symptoms present, is subdivided into—*inflammatory, ataxic, adynamic, irritative, abdominal, thoracic, and hæmorrhagic*.

3. The **insidious or latent form**, also called **ambulatory**, because the patient often walks about during the entire attack. Sudden death may occur in such cases, from perforation or hæmorrhage.

In addition to these varieties, there can be no doubt but that many of the cases of so-called *infantile remittent fever*, as well as those of *gastric or bilious fever*, are merely cases of modified typhoid fever.

RELAPSES.—Typhoid fever is very subject to relapses, and they may occur even three or four times. Sometimes there is a recurrence of pyrexia only, but a true relapse is attended with a return of the characteristic symptoms and lesions. Generally it appears about ten days after the temperature has become normal, and may or may not be attributable to error in diet. A relapse is almost always shorter in duration than the original attack, and the advanced stage is very often not observed. Recovery generally follows. Relapses have been attributed to re-infection from the mesenteric glands, but on no sufficient grounds.

COMPLICATIONS AND SEQUELÆ.—Affections of the respiratory organs are apt to occur during the course of typhoid, as well as in connection with typhus fever; those which are more common in the former than the latter being pneumonia, pleurisy, and acute tuberculosis. Various other complications mentioned under typhus are occasionally met with; and also thrombosis of the veins of the leg, embolism and its consequences, or meningitis. Those that are specially to be dreaded in typhoid, however, are *perforation of the intestine* and *peritonitis*. Perforation generally happens in the third or fourth week, but it may occur as early as the eighth day, or not until the patient is apparently almost convalescent. It is very frequent in the *latent* variety. Usually it is attended with the ordinary symptoms of perforation (to be described hereafter), but sometimes these are very obscure. Peritonitis may be general or local. There may or may not be prominent symptoms of this complication. *Intestinal hæmorrhage* is sometimes regarded as a complication, but in reality it is a symptom which may come on at any time after about the 10th day, being most common from the 14th to the 24th day. It may be independent of any cause, or is brought on by improper food or by exertion.

The most important *sequelæ* of typhoid fever are phlegmasia dolens from thrombosis, phthisis, mental weakness or insanity, temporary general or partial paralysis, neuralgia, otorrhœa, anæmia, and a general state of ill-health, with much debility and wasting, which may never be recovered from. The last-mentioned condition is due to destruction of the villi and glands of the intestines, accompanied with shrivelling of the mesenteric glands.

DURATION AND TERMINATIONS.—It is often difficult to fix accurately the *duration* of cases of typhoid, on account of its insidious mode of onset. Generally it ranges from three to four weeks, rarely extending beyond the 30th day. Many cases terminate on or about the 21st or 28th day. The mean duration of fatal cases seems to be about 22 days, but many run a much shorter course, and death may occur within the first few days. On the other hand, the complaint may be more prolonged, and Murchison mentions an instance in which fresh spots



appeared up to the 60th day. Complications and sequelæ may also protract the disease, as well as one or more relapses.

Typhoid fever may terminate in complete recovery; in death; or in a permanent state of ill-health. The average *mortality* is said to be about 1 in 5·4 cases, ranging usually from 15 to 25 per cent., but it differs in different epidemics, and also with the mode of treatment and other circumstances. The causes of death are:—1. Gradual asthenia, or this condition combined with anæmia. 2. Direct loss of blood, from epistaxis or intestinal hæmorrhage. 3. Poisoning of the blood, as the result of imperfect excretion, or of absorption of septic matters. 4. Hyperpyrexia. 5. Complications, especially perforation of the bowels, or peritonitis.

PROGNOSIS.—Until a patient is quite convalescent after an attack of typhoid it cannot be considered that all danger is past, and a guarded opinion should always be given as to the ultimate result, even in the mildest cases. The prognosis is rather worse in females, in those advanced in years, and in persons who have come recently to an infected district. It is favourable in the young. Family constitution seems to have some influence. Previous debility does not materially increase the danger from enteric fever.

Many of the conditions mentioned as being unfavourable in typhus are to be similarly regarded in typhoid fever, especially severe nervous symptoms, and great prostration, but the pulse and tongue are not so much to be relied upon, and abundant eruption is not a bad sign in typhoid. The pulse and cardiac action must, however, be watched, and great frequency, with marked weakness and compressibility of the pulse, and physical signs of cardiac failure, are to be recognized as more or less grave. The chief indications of danger are severe abdominal symptoms, with excessive diarrhœa; intestinal hæmorrhage, especially if profuse; signs of perforation; symptoms of peritonitis; profuse epistaxis; marked muscular tremors, the mind being clear, which is said to indicate deep ulceration; sudden extreme prostration; aggravation of all the symptoms after a temporary improvement in the second or third week. A relapse rarely proves fatal.

The value of the thermometer in guiding the prognosis of typhoid fever requires particular notice. During the second week the temperature shows whether a case is likely to be severe or not. In mild cases a marked morning remission is observed, which begins early and increases; the evening exacerbation is late; and soon there is a permanent fall, the stage of defervescence setting in. In severe cases the opposite conditions are observed. The prognosis is unfavourable in proportion to the height of the temperature, and to the duration of this increased heat, especially if there are but slight morning remissions. Either a sudden rise, or a rapid and extreme fall, is a bad sign. Considerable irregularity in the ordinary course of the temperature indicates the existence of complications. A marked fall often gives warning of the approach of intestinal hæmorrhage.

TREATMENT.—1. The remarks made with regard to the **hygienic management** of cases of typhus apply equally to those of typhoid fever, but there are some points which require special notice. Remembering the origin and chief modes of propagation of the poison of typhoid every attention must be paid to the *disinfection of the stools*; to the *removal of all filth*; and especially to the *water-supply*, in accordance with the rules already laid down when speaking of the prevention of epidemics.

2. **General management.**—In all cases a patient suffering from typhoid fever should take to bed from the first, and remain there until fairly convalescent. As a rule it is best to use the bed-pan from the first, so that the patient may become habituated to it.

The greatest care is necessary as to *diet*, which should be entirely liquid, nutritious, and non-irritating, and administered at stated intervals, but not too frequently. Good milk is by far the most important article of diet, but beef-tea, which may be thickened with arrowroot, beef-juice or essence, and custards are also serviceable. The patient may drink toast-water, barley-water, or mucilaginous liquids, and may also have tea or coffee, if desired. Fruits are not to be permitted in any quantity, but good grapes in moderation are sometimes welcome, their seeds and skin being removed, but their effects must be watched. This caution in diet is to be observed in all cases, but especially when there is any reason to suspect extensive ulceration. By proper attention to this matter many cases of enteric fever may be brought safely through, without the administration of any medicine whatever. It is very important to see that the milk is good, and obtained from a proper source, as it has sometimes happened that milk infected with the typhoid poison has been given, and thus great harm has been done; moreover, it must not be given in too large quantity, else it will curdle and do mischief, and to guard against this, the stools must be watched. It may be desirable to give the milk with soda or lime-water; or with arrowroot or gelatine. Much difference of opinion prevails as to the employment of *alcoholic stimulants*. It is certain, however, that their indiscriminate use may do a great deal of harm, and that they are not nearly so much needed as in typhus, or at such an early period. Often they are not at all required, and it is only in the more advanced stages, when the strength has been reduced and the circulation is feeble, that they are usually called for. Their effects must be closely watched.

3. **Therapeutic treatment.**—Mineral acids and small doses of quinine are the general medicinal remedies most in repute in the treatment of typhoid, but they are not nearly so efficacious in this disease as in typhus, and usually are not required; indeed, in a large number of cases no medicine is needed throughout.

4. **Symptomatic treatment** calls for the chief attention in the large majority of cases of enteric fever. The ordinary symptoms associated with fever must be treated as previously described, and the supervention of the "typhoid state," hyperpyrexia, or other conditions, as well as of complications, calls for appropriate treatment. Digitalis has been found decidedly useful for improving the action of a failing heart, and diminishing its frequency. Subcutaneous injection of ether has been found efficacious in extreme cases. Epistaxis sometimes requires the use of local styptics; or it may become necessary to plug the nares.

A few special remarks are needed with regard to the abdominal symptoms, which are so often a source of trouble and anxiety during the progress of typhoid fever.

If there is a tendency to much pain or tympanites, the assiduous and early application of linseed-meal poultices or fomentations may give relief. Occasionally turpentine stupes or sinapisms are needed; and if the pain is very severe at an early period in young and plethoric patients, it has been recommended to apply three or four leeches over the right iliac fossa, or a small blister. Opium or morphia internally may also be required for the relief of pain.

Diarrhœa ought to be checked if it is excessive, or if the patient is very weak, but it is by no means always desirable to try to stop it, and care must be taken not to favour accumulation of fecal matters in the intestine, when its walls are becoming paralyzed. Enemata of starch and opium are highly efficacious for the purpose of keeping diarrhœa within bounds. Internally the best remedies are Dover's powder, either alone or combined with carbonate of bismuth; sulphuric acid with tincture of opium; or chalk mixture or some preparation of bismuth, combined with tincture of opium and vegetable astringents. Acetate of lead, sulphate of copper, or nitrate of silver have been employed in obstinate cases.

In those exceptional cases which are attended with constipation great care must be exercised in the use of aperients. A teaspoonful of castor-oil, or a simple enema every third or fourth day, as recommended by Murchison, answers well under these circumstances.

Excessive tympanites is best relieved by the use of enemata containing turpentine or assafoetida, along with the external applications already mentioned. The passage of a long œsophagus-tube into the rectum sometimes gives marked relief as regards this symptom.

Intestinal hæmorrhage, if not checked by the remedies mentioned for diarrhœa, demands the internal administration of full doses of tannic or gallic acid, turpentine, or tincture of iron; or the hypodermic injection of ergotine. Ice may be given to suck constantly, and also applied over the right iliac region.

Should perforation or peritonitis occur, the treatment must consist of absolute rest; the total withdrawal of food, or its administration in very small quantities; and the free use of opium. Should constipation ensue, on no account must aperients be given.

5. **Special treatment.**—It is requisite to notice certain special modes of treatment which have been advocated for typhoid fever.

1. *Antiseptic treatment.* Various antiseptics have been recommended internally, especially carbolic acid or the sulpho-carbolates, and salicylic acid or salicylate of soda, mainly on the assumption that the disease depends upon specific organisms, which might be destroyed by the agency of these remedies. There is no adequate proof that these agents are really useful, and certainly they ought not to be relied upon solely; at the same time there would be no objection to the administration of harmless antiseptics along with other remedies, as they might have a directly beneficial effect upon the intestinal lesions, and at the same time tend to prevent the formation of septic matters, and consequent blood-poisoning.

2. *Hydropathic treatment.* On the continent, and particularly in Germany, typhoid fever is now extensively treated by means of baths, and this has led to a great reduction in mortality there, but the results do not show that the treatment brings the death-rate below what is usual in this country. This plan is, however, supported by some high authorities here. The advantages claimed are that the treatment by baths prevents the temperature from reaching an injurious height, and limits the intestinal lesions. The exact plan of carrying it out is different with different practitioners, the bath being used at 65° to 70°, at 75°, or from 80° to 90°, and then gradually cooled to 70° or 65°; the patient is kept in it for periods varying from 10 to 25 minutes: it is repeated from three to eight times during the twenty-four hours; and the treatment is continued for two or three weeks or more. Brandy is often given at the same time, and some use also large



quantities of quinine or salicylic acid. There are obvious objections to such a routine treatment as this, and relapses seem to be more frequent under its employment. From personal experience I cannot recognize the necessity for it in ordinary cases of typhoid, but am content with milder measures. Tepid or cold sponging of the skin every night and morning, or more frequently, is most comforting and beneficial; and I have had several cases under my care which have done exceedingly well, where wet compresses were applied continuously over the abdomen and chest, but changed at short intervals. The wet-pack and the ice-cap have also been used with advantage. Another serviceable plan is to place the patient on a water-bed containing cold water. Should any tendency to hyperpyrexia be manifested, energetic hydropathic treatment should be at once resorted to, in order to bring down the temperature. 3. *Eliminatory treatment.* Some practitioners, instead of endeavouring to check the diarrhoea in typhoid, encourage it by means of aperients, with the view of assisting the elimination of the supposed poison. This plan of treatment is obviously attended with much danger, and in my opinion is most objectionable; at the same time a gentle aperient at the outset may be of use, and some good authorities recommend a dose or two of grey powder or calomel.

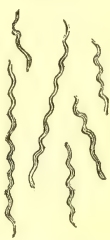
6. *Convalescence.*—During convalescence much careful supervision is needed for some time, especially with regard to food, and the employment of purgatives. The diet must be very gradually improved, and only taken in moderate quantities. No solid food should be given until the temperature has been normal for at least a week, or even ten days. It is the more necessary to insist upon this, because patients are often much inclined to indulge to excess in all kinds of food, and do not understand why they should be restricted. Wine is valuable at this time, if properly employed. If an aperient is required, a small dose of castor-oil or a simple enema answers best. Tonics and change of air have a very beneficial effect in promoting recovery and restoring strength. Cod-liver oil is also useful, if much debility and wasting remain.

## CHAPTER VII.

### RELAPSING FEVER.—FAMINE-FEVER.

*ÆTIOLOGY.*—Relapsing fever is an acute specific disease, originating in a *specific poison*, and it is highly infectious. Some suppose that this affection is merely a milder form of typhus fever, but unquestionably they are distinct diseases. Relapsing fever often spreads rapidly, and this happens the more readily where there is freedom of intercourse between the sick and healthy. Dr. de Zouche observed that when it gained access into a house it usually attacked every inhabitant therein. The contagium is chiefly given off in the breath and exhalations, and those who come much into contact with patients suffering from relapsing fever are most liable to be attacked. It may be conveyed by individuals or fomites, and clings tenaciously to a house for months. With regard to the nature of the contagium, this seems to have been definitely associated with certain organisms named *spirilla*, a form of bacterium. These were first discovered by Obermeier in 1872, and their

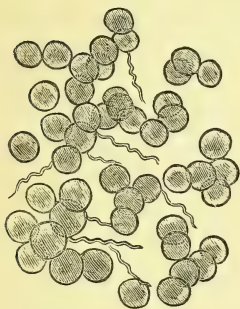
FIG. 9.



Spirilla of Relapsing Fever.  $\times 500$ .

existence has since been fully confirmed by other observers. They are in the form of moving spiral fibrils, extremely delicate, and from two or six times the diameter of a blood-corpuscle in length ( $\frac{1}{1500}$  to  $\frac{1}{500}$  inch). These spirilla are only found in the blood, not having been

FIG. 10.



Spirilla amongst red blood corpuscles.

detected in any of the other fluids of the body, or in either of the secretions. Moreover, they are only present at certain times, namely, during the febrile paroxysms, for they disappear entirely shortly before the crisis, and are absent during defervescence and in the apyrexial intervals. It has been proved by experiment that relapsing fever is easily communicated by inoculation of the blood, but only during the paroxysms, when the spirilla are present. The complaint cannot be produced by inoculation of any other fluid of the body.

Heydenreich has proved experimentally that spirilla are very short-lived at even the normal temperature of the blood, but still more so at febrile temperatures, and probably their variable prevalence in the same attack is associated with the development and disappearance of successive generations. In 1877 an organism apparently identical with the spirillum was found in the blood of patients suffering from famine-fever in Bombay. Dr. Vandyke Carter has made an interesting series of observations upon this organism, of which he gave an account at the meeting of the International Medical Congress in 1881.\* His conclusions are summed up thus:—

The following data point to a real connection of spirillar blood contamination with the pyrexial attacks of relapsing fever:—1. Infection is always followed by fever. 2. With the advent and progress of pyrexia the blood parasites increase. 3. They disappear with the cessation of fever. 4. By contact with the sick, and by inoculation of blood containing the spirillar organisms or their germs, the disease may be conveyed to new or old subjects.

The following data point to conditions modifying at least the connection above implied:—1. The presence of the blood parasite during several hours or for one or two days prior to fever. 2. The sudden outburst of pyrexia is not preceded by or attended with a proportionate visible augmentation of the spirillum. 3. The absence of any fixed relation between variation in form and intensity of fever and varying numbers of the organism. 4. The persistence of the parasite during pseudo-crises and defervescence by lysis.

*Predisposing causes.*—The anti-hygienic conditions which promote the spread of typhus have a similar influence in the case of relapsing fever, particularly want of food, overcrowding, and filth, and Murchison believed that the disease may be spontaneously developed, especially as the result of destitution. It prevails generally during periods of famine, and has hence been called famine-fever. This fever is most frequently met with in the British Islands, especially in Ireland, but is far less prevalent than was formerly the case. A severe epidemic occurred in Liverpool a few years since. Males are attacked in larger proportion than females; and the complaint is most common between 15 and 25 years of age.

\* See "Transactions of International Medical Congress," 1881, Vol. I., page 334.

**ANATOMICAL CHARACTERS.**—There are no *post-mortem* appearances at all characteristic of relapsing fever. Petechiæ and jaundice are persistent if they have been observed during life. The blood frequently contains much urea, and the white corpuscles are increased; occasionally it is dark and fluid. The presence of spirilla in the blood has been already discussed. The spleen is usually much enlarged during the febrile paroxysm, and softened; sometimes it contains fibrinous deposits. The liver is also considerably enlarged and congested, but neither this organ nor its duct exhibits any condition likely to account for the jaundice present. There may be signs of various morbid conditions of the nature of complications.

**SYMPTOMS.**—The *period of incubation* in cases of relapsing fever lasts from about four to ten days usually, but may be very much shorter, the attack even appearing to commence almost simultaneously with exposure in exceptional instances.

The *invasion* is remarkably sudden, the patient usually feeling perfectly well immediately before, and being able to fix upon the exact moment of attack. The first symptoms are often felt on awaking in the morning. Generally, however, there has been constipation for some days previously (De Zouche). The attack is commonly ushered in with a severe rigor, and a sense of great weakness, but there may be only slight shivering. Sharp frontal headache is complained of at once or very soon, rapidly increasing in intensity, with giddiness, and pains in the back and limbs, often exceedingly severe. After a variable time marked pyrexia sets in, with dry pungent skin, flushed cheeks, frequent pulse, and excessive thirst. In two or three days profuse and general perspiration follows in some cases, but gives no relief. The rigors are repeated at intervals, and may alternate with sweating, simulating ague. In children the disease may commence with a "heavy sleep." Vomiting and retching are early symptoms, the vomited matters being yellow, yellowish-green, or green, and consisting of bile and gastric secretions; sometimes they are black. Epigastric uneasiness or pain, and pain or tenderness over the liver and spleen, are also complained of, these organs, especially the latter, being obviously enlarged. Appetite is quite lost as a rule, and there is great thirst throughout. The tongue is at first moist and covered with a white or yellowish fur, and generally remains in this condition throughout, but it may become dry and brown, with sordes on the teeth. It is often transversely fissured and red at the edges, with enlarged papillæ. In bad cases patches of ulceration are observed on the tongue and inside of the cheeks. Constipation generally persists throughout, the stools being normal in colour or dark. Sore-throat is frequently complained of, the fauces being reddened, and one or both tonsils being enlarged.

The appearance of the patient is often quite characteristic. "The eyes appear somewhat sunken, from the dark circle which surrounds them; they are clear, but have a despairing, woe-begone look, not easily to be forgotten if once seen. The whole face expresses the consciousness of pain and helplessness" (De Zouche). More or less jaundice is observed in many cases, and sometimes the skin exhibits a bronzed hue. Various eruptions have been described in individual cases, but there is nothing of this nature at all specific in connection with relapsing fever.

The pulse rises rapidly to 100, 120, 140, or even 160. It may be full and strong, but in bad cases tends to become weak, intermittent, or irregular, the impulse and sounds of the heart being at the same time



feeble. The urine is febrile; it may be much diminished in quantity or suppressed, and urea is often deficient. In exceptional cases the urine contains albumen. Headache continues very severe throughout, with a throbbing sensation, and there is much restlessness and sleeplessness. Delirium is uncommon, but is occasionally observed, especially towards the crisis, when it may be violent in character.

The symptoms become intensified towards the period of the *crisis*, which event happens in most cases on the 5th or 7th day, but it may take place at any time from the 3rd to the 10th day. They are often alarming at this time, and dyspnœa may be prominent. Crisis is almost always accompanied with profuse sweating, the perspiration pouring off for some hours. Sometimes sudamina appear, and occasionally watery diarrhœa or vomiting occurs. Hæmorrhages are not uncommon, especially epistaxis, and now and then menorrhagia or hæmorrhage from the bowels. In severe cases, and in weak individuals, a copious and general petechial eruption is often observed at this time. The symptoms generally rapidly abate; the pulse and temperature fall even below the normal, the former frequently continuing below par; the tongue cleans; and the patient often feels quite well, only being a little weak. Most patients soon get up, and some try to work.

In exceptional cases there is not a complete cessation of symptoms, but only a partial remission. In other instances severe muscular and arthritic pains are complained of over the body generally, the metacarpal and phalangeal joints being most liable to be attacked. These painful sensations prevent sleep, and may make the patients cry out. At this time the disease is liable to be mistaken for acute rheumatism, especially as some of the joints occasionally become swollen. Bronchitis may set in during the intermission, with much spasmodic cough, and expectoration of viscid tenacious mucus, or even of blood; the symptoms sometimes resemble those of whooping-cough, especially in children.

*Relapse.*—Occasionally no relapse occurs, particularly towards the end of an epidemic, or it is scarcely noticeable. De Zouche states that he was always able to ascertain on close questioning that a relapse had taken place. It may set in any day from the 12th to the 17th, generally on the 14th. Its onset is equally sudden with the primary attack, and the symptoms are precisely the same, their intensity being, however, usually less, though they are in exceptional cases more severe. The average duration of the relapse is from three to five days, but it may vary from a few hours to seven or eight days, and it terminates by crisis, usually in the same manner as after the first attack. A second, third, fourth, and even fifth relapse has been sometimes observed.

In rare instances extreme prostration suddenly comes on, with signs of collapse, the face being of a purplish colour, especially the nose, the limbs cold and livid, the pulse very feeble, and the patient becoming unconscious. In other cases typhoid symptoms are developed, accompanied with suppression of urine.

*Temperature.*—There is a continuous ascent in relapsing fever for four or five days, without any evident morning remission, the temperature finally reaching 104°, 105°, 106°, or even 108°. It may then remain stationary, with slight morning remissions, until the period of *crisis*, when it falls below the normal. At the *relapse* it again rapidly rises, and may reach even a higher point than during the first attack; it once more suddenly falls at the second crisis.

**COMPLICATIONS AND SEQUELÆ.**—The most important are bronchitis or pneumonia; various hæmorrhages; sudden syncope; pains in the muscles and joints, occasionally with effusion into the latter; a peculiar form of ophthalmia, preceded by amaurosis; diarrhœa or dysentery; œdema of the legs, due to debility and anæmia, which may also cause an anæmic murmur; parotid or other buboes; and abortion. De Zouche observed in many cases fine desquamation of the cuticle about the second week after crisis, and also falling-off of the hair.

**TERMINATIONS.**—The great majority of cases of relapsing fever recover; in a large number collected by Murchison the mortality was only 4·75 per cent. Convalescence, however, is often tedious, and a state of marked debility sometimes remains, which may ultimately prove fatal. Death may result from syncope, collapse, or nervous exhaustion; excessive diarrhœa or dysentery; uterine hæmorrhage, especially after premature labour; uræmia; excessive vomiting in children; or from pneumonia, peritonitis, or other complications.

**PROGNOSIS.**—This is generally favourable in relapsing fever, but is less so in aged persons, and in those who have been lowered by disease, intemperance, or privation.

The chief signs of danger are marked jaundice; severe hæmorrhages, especially uterine; extensive petechiæ or purpuric spots; sordes and ulcerations about the tongue and mouth; incomplete defervescence after the first crisis; suppression or great diminution in the quantity of urine; severe cerebral symptoms; signs of syncope; and the presence of grave complications. It must be remembered that serious symptoms may come on quite suddenly, even in a mild case. Convalescence is often considerably delayed, on account of sequelæ.

**TREATMENT.**—1. During the **first febrile paroxysm** it is desirable to keep the bowels regularly open, but not to purge excessively. If the case is seen early, an *emetic* seems to be useful. Attention must be paid to the urine, to see that excretion is properly taking place; and saline *diaphoretics* and *diuretics* may be given. A drink containing 3 i or 3 ij of nitre to the pint is recommended by Murchison. Tincture of aconite and Warburg's tincture have been well spoken of in the treatment of relapsing fever. Cold or tepid sponging is very useful.

Opium is a most valuable remedy to relieve headache, sleeplessness, vomiting, and the severe pains present. De Zouche found hydrate of chloral of use. He only considers it desirable to moderate vomiting, should this symptom be excessive. Other symptoms must be attended to, if required; as well as complications.

The *diet* should be light, though nutritious, as a rule, but if the patient is low, a more supporting diet is indicated. *Alcoholic stimulants* are not often required, but should be given from the first if there is much debility, if an anæmic murmur is present, or if there is any tendency to syncope. They are also called for in the case of old people, and of young infants who cannot take the breast; and are in most cases necessary during the exhaustion following the crisis, as well as during convalescence.

2. During the **interval** the patient should be kept in bed, if possible. Various remedies have been tried with the view of preventing the relapse, but without success. Quinine in five-grain doses may be given. The *relapse* must be treated on the same principles as the primary attack.

3. During **convalescence** good diet and *tonics*, especially quinine,

mineral acids, and iron, are indicated. These also seem to be the best remedies for the various sequelæ. The application of leeches and blisters behind the ears, the administration of calomel internally, and the local use of atropine are recommended for the ophthalmia which follows relapsing fever.

---

## CHAPTER VIII.

### SCARLATINA—SCARLET FEVER.

**ÆTIOLOGY.**—Produced by a *specific poison*, scarlatina is highly infectious. The nature of this poison has not yet been positively ascertained, but micrococci have been described. It is especially abundant in connection with the epithelium which is shed from the skin, by means of which it may be conveyed to a considerable distance. Infection may arise from merely going into the room where a patient is lying ill of scarlatina, or being in the same house, or even in the neighbourhood. The apartment also frequently retains the poison lurking in various parts for an indefinite time, unless it has been thoroughly disinfected, and therefore may be the means of originating the disease after a long interval. The infected epithelium-particles easily cling to clothes, letters, and other fomites, and by their aid are often carried far and wide. They are also sometimes conveyed by milk and other kinds of food. It is important to remember that insusceptible individuals may disseminate scarlatina, if allowed to pass from the sick-room and to mingle with healthy persons. The disease has been produced by inoculation. Some believe that it may originate spontaneously, but this is highly improbable. A second attack rarely occurs, and still more exceptionally a third. As to the time that infection lasts in a patient, there is no certainty, but it is safer to consider it as beginning with the incubation-period, and not ceasing until desquamation has been thoroughly completed.

Young children are chiefly attacked, from eighteen months to six years of age, but especially those from three to four years old. In addition to the immunity afforded by a previous attack, the liability to scarlatina decidedly diminishes with advancing years. Both sexes are equally affected. The complaint is more prevalent in large towns, and among the poor. Cases are said to be most frequent in autumn, especially from September to November; there are many exceptions to this statement, however, and epidemics are but too common at other times.

It is a well-ascertained fact, originally brought prominently into notice by Sir James Paget, that patients who have been operated upon are peculiarly liable to a scarlatinal rash, and sometimes the disease assumes a typical and very severe or even fatal form. It has been a disputed point whether this complaint is actual scarlatina, but Mr. Howard Marsh\* has brought forward strong evidence to prove that it is a true, though often modified form of this disease, and he considers it an illustration of how specific diseases are liable to be modified by the circumstances under which they are developed. Scarlet fever seems to stand alone in its tendency to attack patients directly after operations.

\* See "Transactions of International Medical Congress," 1881, Vol. IV., page 177.



**ANATOMICAL CHARACTERS.**—The changes found after death from scarlatina vary according to the severity of the attack, and the structures involved. One of the ordinary anatomical characters consists in erythematous inflammation of the skin, with superficial œdema, constituting the scarlatinal rash, which is more or less persistent after death. Klein has investigated the anatomical changes observed in certain organs in scarlet fever, and has described them with much minuteness.\* Here only a brief summary can be given.

The **kidney** presents changes which differ according to the duration of the disease. In early cases the vascular apparatus, especially that of the cortical portion, shows the following alterations:—1. Increase of nuclei (probably epithelial nuclei) covering the glomeruli of Malpighian corpuscles. 2. Hyaline degeneration of the elastic intima of minute arteries, especially of afferent arterioles of Malpighian corpuscles. 3. Multiplication or germination of the nuclei of the muscular coat of minute arteries, and a corresponding increase in thickness of the wall of these vessels. The glandular part of the kidney gives indications of parenchymatous nephritis, consisting in cloudy swelling of the epithelial lining of some convoluted tubes, germination of the nuclei of epithelial cells, granular disintegration of epithelium, and in some cases detachment of epithelium in the larger ducts of the pyramids. At first these changes are slight and limited, in some cases requiring careful examination to detect them. After about the ninth or tenth day the parenchymatous nephritis becomes more intense, indicated by crowding of urinary tubes with lymphoid cells, granular and fatty degeneration of epithelium of urinary tubes, and cylinders of different kinds in the tubes. Interstitial nephritis also sets in, evidenced by the infiltration of the connective tissue of the kidney with lymphoid cells, commencing around the large vascular trunks. In one case Klein found emboli in arteries in the foci of very intense interstitial inflammation. The intensity of the parenchymatous change seems to be dependent upon the degree of the interstitial nephritis. Other observers have regarded the presence of interstitial nephritis as unusual in scarlatina, but Klein looks upon it as the general rule in cases that die after about nine or ten days. Klebs has described a condition of glomerulo-nephritis in cases of scarlatina which die with symptoms of anuria and uræmic poisoning, where the kidney does not show any marked changes. On microscopical examination of the glomeruli, the whole space of the capsule is seen to be filled with small angular nuclei embedded in a finely granular mass. The vessels of the glomerulus are almost completely covered by nuclear masses. Dr. Bryan Waller has described similar appearances, and regards the intra-capsular cell-accumulation as composed partly of proliferated connective-tissue corpuscles, and partly of migrated leucocytes, which have passed through the walls of the glomerular capillaries. Klein remarks “a very curious fact is the deposit of lime matter in the epithelium and lumen of urinary tubes, first of cortex, then also of pyramids, at an early stage of scarlatina, when the kidney otherwise shows only very slight change.”

The structures of the **fauces** are the seat of more or less inflammation, which may terminate in destructive lesions, and Klein found peculiar changes in the following lymphatic structures, namely, the lymphatic follicles at the root of the tongue and pharynx, those in the mucous membrane lining the posterior surface of the epiglottis, those

\* See “Pathological Transactions,” Vol. XXVIII., page 30.

forming the tonsils, those in the mucous membrane of the larynx and trachea, and those in the submaxillary lymphatic glands. He observed that in the central portion of these follicles the ordinary uninuclear lymph-cells forming their chief bulk were greatly decreased in number, their place being taken by large granular cells, containing from two to twenty or more germinating nuclei. In the glands of the neck he also found fibrinous thrombi in the veins; and at first hyaline, then fibrous degeneration of adenoid tissue, and the appearance of giant-cells.

The **liver** is slightly enlarged, and Klein observed in this organ granular opaque swelling of liver-cells; filling of some of these cells with fat-globules, of others with pigment; similar changes in some of the arteries to those described in the kidney; but, above all, indications of acute interstitial hepatitis, namely, great thickening of the connective tissue of Glisson's capsule, and collections of round cells in the intertubular connective tissue. In some cases these cells were also found within the acini.

The **spleen** is not uncommonly enlarged and hyperæmic. Klein noticed as constant changes—enlargement of Malpighian corpuscles; hyaline degeneration of intima of arteries; multiplication of the nuclei of the muscular coat of ultimate arterioles, and hence increased thickness of their walls: hyaline swelling and degeneration of adenoid tissue around degenerated arteries; and changes in the central parts of many Malpighian corpuscles, somewhat similar to those described in the lymph-follicles of the throat. The mesenteric glands may be enlarged and congested.

The **blood** in cases of scarlatina is generally deficient in fibrin and in coagulability; sometimes, on the other hand, fibrin is in excess.

Various morbid conditions of the nature of complications are frequently met with in fatal cases.

**SYMPTOMS.**—Scarlatina presents several important and well-marked varieties in its clinical history, but before alluding to these a typical case—**Scarlatina Simplex**—will first be described.

1. **Incubation-stage.**—The *period of incubation* lasts in most cases from three to five days; it may not be longer than one or two days, or it may extend to six or eight days, but not beyond this (Squire). Generally there are no symptoms, but the patient is sometimes a little ailing, languid, and restless.

2. **Invasion-stage.**—The onset of scarlatina is usually distinct. Chilliness is felt, but not severe rigors, followed by pyrexia, varying in its degree, but the temperature generally rises rapidly to 104° or more. The skin feels hot and dry, the face is flushed, and the pulse is very frequent. At the same time sore-throat is complained of, the fauces being reddened or dry, while the neck feels stiff, and tenderness is noticed about the jaws. Vomiting is often a prominent symptom, with much thirst, and total loss of appetite. The tongue is usually furred, and red at the tip and edges, presenting also enlarged papillæ. Pains in the limbs, lassitude, frontal headache, and restlessness are generally present. There may be a little delirium at night, and in young children scarlatina is sometimes ushered in by sudden convulsions or coma.

3. **Eruption-stage.**—The scarlatinal *rash* generally appears on the second day, but sometimes it comes out within twelve hours, or not until the third or fourth day. Its primary seat ordinarily is the neck and upper part of the chest, but it spreads rapidly to the face, as well as over the trunk and limbs. Sometimes it appears first on the legs.

The eruption begins as minute bright red spots, which speedily coalesce to form uniform patches of greater or less extent, so that large portions of the surface may be covered with the rash. The precise tint varies, but it is usually bright scarlet, or of a boiled lobster or raspberry hue, though it becomes darker as the case progresses. The colour is more marked in the centre of each spot, and disappears completely on pressure, soon returning again on the removal of pressure, being preceded by a yellowish hue. The patches are usually very distinct in the flexures of the joints. The spots are not at all elevated as a rule, but occasionally separate ones are slightly papular. The rash reaches its height usually about the fourth or fifth day from the commencement of the illness, and begins to fade from that to the sixth day, this occurring first in the parts first invaded by it. It has generally disappeared before the ninth or tenth day, and then desquamation sets in.

Sudamina are frequently observed if the rash is intense, especially in adults. They are seen about the neck and chest, in the axillæ or groins, or occasionally over the whole body. The skin feels dry, and often in some parts rough, presenting the condition known as *cutis anserina*. Not unfrequently the eyelids, hands, and feet are puffy. The patient experiences a sensation of heat or burning, and there may be much itching or a sense of tingling.

*State of the throat.*—There is more or less general redness of the fauces, with œdema and swelling. The surface is dry or covered with viscid mucus, and thick opaque secretion is often seen on the tonsils. These may be slightly ulcerated, or the seat of suppuration. There are the usual subjective symptoms of sore-throat, with pain and difficulty of swallowing. The glands about the angles of the lower jaw are enlarged and painful, and the subcutaneous tissue is sometimes œdematous and puffy. The mucous membranes of the nose and mouth, as well as the conjunctivæ, are often red and inflamed.

*Temperature* usually continues to rise until the rash attains its height; then it remains stationary, and subsides as the eruption begins to fade, either by crisis or gradually. It ranges as a rule from 104° to 106° F., but may reach 107°, 108°, or even higher in exceptional cases. There is a slight morning remission.

The pulse is frequent, and may reach 120, 130, 160, or more; it varies in its force, but is usually strong and full. It falls as the temperature lowers. The tongue is furred, and presents the so-called “strawberry” appearance, owing to the papillæ being much enlarged and red, and projecting through the fur, so that the surface may actually feel rough. As it cleans, the tongue is seen to be red, and the papillæ remain prominent, sometimes for a considerable period. Appetite is quite lost, but there is much thirst. The bowels are usually constipated. More or less headache continues; while the patient is restless and sleepless, or has some nocturnal delirium.

The urine is febrile, and deposits sediments of uric acid and urates, which, as well as urea, are usually increased. Chloride of sodium and phosphates are diminished in quantity. Albumen is often present, and renal epithelium is visible under the microscope. Sometimes the urine contains blood.

4. *Desquamation-stage.*—The symptoms subside more or less rapidly, and then the epidermis begins to separate, this process lasting a variable period, while the amount of desquamation also differs much, being usually in proportion to the intensity of the rash, and the number



of sudamina. The skin feels dry and inelastic before desquamation commences. The process begins as a rule in those parts where the rash first appears. Where the skin is thin, the epidermis comes off in small, branny scales; in other regions it forms small patches; but where the cuticle is very thick, as over the palms and soles, it peels off in extensive pieces, sometimes forming a mould of the fingers or hand.

During this period the pulse and temperature frequently fall below the normal for some days. The urine becomes abundant and watery, as well as deficient in phosphoric acid, and it contains a considerable amount of renal and vesical epithelium. The throat may remain sore and the tonsils enlarged for some time.

VARIETIES.—Such being the ordinary course of scarlatina, it is important to point out the chief varieties which may be met with.

1. In some instances the symptoms are very trifling, the temperature not being at any time higher than  $101^{\circ}$  or  $102^{\circ}$ , and only a slight rash and sore-throat being present, which soon disappear. This is a mild form of **Scarlatina Simplex** or **Benigna**.

2. **Scarlatina Anginosa**.—In this form the condition of the throat is grave, and gives rise to severe and prominent symptoms. There is extensive and deep inflammation of the tissues, the redness tending towards a dark hue, the tonsils and uvula being much swollen, while sticky mucus and secretion cover the surface, or sometimes diphtheritic-looking patches are visible. Ulceration then often sets in, or occasionally gangrene, which may spread extensively, and may even involve the larynx. Dangerous hæmorrhage is thus liable to be induced. The glands about the jaw and the other structures of the neck swell considerably, and may suppurate or slough to a variable extent. In some cases the salivary glands are involved. Much difficulty is experienced in opening the mouth and examining the throat, which is very painful, while deglutition is exceedingly difficult and distressing, and fluids are liable to enter the posterior nares during the act of swallowing. The breath is extremely disagreeable.

The rash is usually delayed in its appearance; is less marked or diffused; disposed to fade and return again; and its final departure is later than usual.

Often there is much swelling of the nasal mucous membrane, with offensive and irritating discharges from the nostrils, or blocking-up of these passages by secretion. The mouth and lips are also sore and cracked; and the tongue has a darker hue than in ordinary cases.

The general symptoms are prone to be of a low type, this being partly due to interference with respiration, and to the absorption of putrid matters from the throat. When adynamia is very marked from the first, the variety is sometimes named **Scarlatina Anginosa Maligna**.

Nausea, vomiting, diarrhoea with irritating discharges, and tympanites may result from swallowing morbid materials from the throat.

If the case terminates in recovery, the temperature continues high after the rash has disappeared, owing to the state of the throat.

3. **Scarlatina Maligna**.—As just stated, the symptoms may take on an exceedingly adynamic or malignant character in connection with a bad state of the throat, and the same thing may happen during any severe attack of scarlatina, especially if the patient has been previously in a weak or unhealthy condition. Nervous symptoms are then prominent from the first. There is much prostration, with restlessness, insomnia, and muttering delirium; followed by convulsions, stupor, or

coma. The pulse is very feeble, rapid, small, and irregular; the circulation is impeded, as evidenced by duskiness of the face, and capillary congestion in dependent parts: while petechiæ are frequently observed, and sometimes hæmorrhages occur. Respiration is much hurried. The tongue is dry and brown.

An important class of *malignant* cases are those in which there is extreme prostration from the first, with intense nervous depression, the poison seeming to act powerfully upon the nervous system. The child becomes faint and sick, is pale and cold or almost collapsed, exhibits great restlessness and anxiety, or may be delirious. The pulse is extremely rapid, weak, small, and irregular. The depression speedily increases; the face becomes very pale, livid, or mottled; and coma or convulsions set in. The breathing is quick and irregular. The skin becomes cold, or alternately hot and cold, and clammy perspirations break out. Death may take place before the eruption has time to appear; or a slight irregular rash comes out, should the child live long enough.

Several cases of this character are sometimes met with in the same family, its members being rapidly carried off one after another.

4. **Scarlatina sine eruptione.**—In some cases there is fever, with a sore throat, but no eruption appears. This is liable to happen in second attacks.

5. **Latent.**—There may be no symptoms whatever, and the fact of a patient having suffered from scarlatina may be known only by desquamation of the cuticle taking place; or albuminuria and dropsy setting in.

In addition to these varieties others have been described depending upon the characters of the eruption, such as *papulosa*, *variegata*, *pustulosa*, *pemphigoidea*, &c., but these are of no practical consequence.

COMPLICATIONS AND SEQUELÆ.—1. The most frequent and important complication, and the one that requires special notice, is *acute desquamative nephritis* and its consequences. Many authorities, indeed, look upon this morbid condition as *part of the disease*. It is imperative in all cases of scarlatina to examine the urine at frequent intervals, even for some time after apparent convalescence. There is often a certain amount of albuminuria, as in other febrile diseases, and the kidneys are always more or less affected, but these conditions disappear as the fever subsides. The renal affection generally appears during or after desquamation, and though exposure to cold seems to excite it in some instances, as a rule it comes on quite independently of any such obvious cause. It is due to the deficient action of the skin, which involves excessive activity on the part of the kidneys, these organs having at the same time to remove large quantities of waste-products. Probably the scarlatinal poison itself has some direct influence upon the renal epithelium. The symptoms are similar to those of ordinary *acute Bright's disease*, scarlatina being in fact one of the most frequent causes of this affection. The urine becomes diminished or suppressed; is highly concentrated; contains more or less blood, or has a smoky tint; is highly albuminous; and presents blood-corpuscles, renal epithelium, and epithelial, blood, or granular casts under the microscope. Dropsy sets in, beginning usually in the subcutaneous tissue, and it may spread with great rapidity, involving the serous membranes, larynx, and lungs in some cases. More or less pyrexia is usually present, as well as frequent vomiting, constipation, headache, and drowsiness; while there is always a danger

of distinct uræmic symptoms setting in. The symptoms may subside and disappear; or a form of chronic Bright's disease may remain as a sequela. In some instances this complaint seems to commence as a chronic affection. Dr. George Johnson is of opinion that in the presence of certain casts in the urine, which he terms "exudation cell-casts," or "white-cell casts," and which he thinks may probably be made up of leucocytes, we have the means of diagnosing the existence of the glomerulo-nephritis of Klebs.

The remaining *complications* or *sequelæ* calling for notice are:—  
 2. Dropsy without albuminuria, and having no obvious cause. 3. Ulceration of the throat, either a continuation and extension of that originally present; or a new form, spreading rapidly with much sloughing, and affecting the tissues of the neck widely. Pus may then find its way into the chest; or the vessels of the neck may be opened, causing fatal hæmorrhage. 4. Affections of the joints, either of a rheumatic character; or ordinary inflammation, occasionally ending in suppuration or in chronic disease. The inflammation is sometimes around, rather than in the joints, or it may be set up in connection with muscles. 5. Serous inflammations, with a tendency to the formation of pus, especially pleurisy and pericarditis, being usually, but not necessarily, dependent upon renal disease or rheumatism. 6. Bronchitis and pneumonia, and possibly phthisis. 7. Endocarditis, which may lead to permanent organic affections of the valves and orifices of the heart. 8. Affections of the ear. These are by no means uncommon, and include otorrhœa; inflammation of the tympanum ending in suppuration, followed by rupture of the tympanic membrane; inflammation or ulceration of the Eustachian tube, with subsequent closure, and consequent deafness; necrosis of the bones, which may cause meningitis, abscess of the brain, or facial paralysis. 9. Abscesses in various parts, especially in connection with lymphatic glands. 10. Gangrene occasionally. 11. Inflammation and destruction of the cornea in rare instances.

**PROGNOSIS.**—The number of deaths from scarlatina varies greatly at different periods, but the mortality from this disease is considerable every year. A careful prognosis should always be given, and account must be taken of the possible *complications* and *sequelæ*. The complaint is most fatal among very young children; and in large towns. The chief circumstances which render the prognosis grave are a severe epidemic type of the disease; family predisposition to a fatal termination; great depression at an early period; typhoid symptoms at any time; late development of, or a tendency to duskiness in the eruption, especially if accompanied with petechiæ or hæmorrhages; prominent nervous symptoms; extensive sloughing or ulceration about the throat, hæmorrhage, and other dangerous local complications; renal inflammation and its results; and severe diarrhœa or vomiting. Special allusion must be made to *pregnancy*. The occurrence of scarlatina in connection with this condition is extremely dangerous, and life may be destroyed in a few hours.

**TREATMENT.**—1. In the first place, the general treatment for the **prevention of infection** must be thoroughly carried out in every particular in cases of scarlatina, and it is always advisable to keep patients under observation, and to exercise every precaution, until desquamation has entirely ceased, and all danger of the development of renal disease has passed away.

2. In ordinary cases but little **medicinal treatment** is required.



The bowels should be acted upon occasionally; and some saline mixture may be administered, such as one containing citrate of potash or liquor ammoniæ acetatis. Barley-water, lemonade, or iced water may be given freely as a drink, and a diet of milk and beef-tea allowed. The skin should be carefully sponged with lukewarm water twice a day, different parts being exposed in succession and then dried; to this water may be added a little carbolic acid, Condyl's fluid, or camphor, to act as a disinfectant. Some practitioners recommend that oil or grease should be rubbed in, or carbolized oil applied. When the fever and rash have subsided, warm baths should be used about every other day, the patient being well scrubbed with carbolic acid soap, in order to get rid of all the infectious epithelial scales.

3. Some of the principal **symptoms** which may call for active interference in cases of scarlet fever will now be considered.

The *throat symptoms* are best relieved in ordinary cases by sucking ice or inhaling steam. If there is much redness and swelling these measures should be steadily persisted in, or the throat may be gargled frequently with lukewarm water, if the patient is old enough to perform this act, heat and moisture being at the same time applied externally over the neck. It may possibly be advisable to apply a few leeches about the angles of the jaw, but very seldom. For ulceration and gangrene *antiseptic* gargles should be freely used, or if these cannot be employed, the application may be made with a brush, or in the form of spray. The best antiseptic remedies are carbolic acid, creosote, chlorate of potash, Condyl's fluid, or sulphurous acid; some prefer chloride of lime, chlorine water, dilute hydrochloric acid, common salt, or peroxide of hydrogen. It may be requisite to touch ulcers with nitrate of silver or its solution.

If the throat is in a serious condition, it is most important to attend to *general treatment*. Nourishing food, in the form of soups, meat extracts, milk, &c., as well as stimulants, especially port wine or brandy, must be given more or less freely in proportion to the state of prostration of the patient, which is often considerable in these cases. At the same time tincture of steel should be administered in full doses— $\mathfrak{m}$  xx- $\mathfrak{x}$ l every three or four hours, alone or combined with quinine or mineral acids. In some cases ammonia and bark are preferable. Carbolic acid and sulphocarbates, creosote, hypochlorite of soda, the hyposulphites, and other *antiseptics* have been much commended in *scarlatina anginosa*, and there is no harm in giving either of these at the same time as the remedies just indicated. Chlorate of potash may be allowed freely as a drink. When the nostrils are blocked up, and there is much nasal secretion and discharge, it is well to wash out the meatuses occasionally with some weak disinfectant solution, or a weak solution of nitrate of silver may be required.

In actual practice it is not uncommonly exceedingly difficult to carry out the instructions just given, especially when we have to deal with young children who cannot or will not swallow; much must then depend upon the judgment, sagacity, and firmness of the practitioner, but it must be borne in mind that the only hope of recovery in a large number of this class of cases lies in free support of the patient, and if the necessary materials cannot be introduced into the stomach, they must be administered by enemata.

The other conditions which are likely to call for attention are hyper-

pyrexia; adynamic symptoms; restlessness, sleeplessness, or delirium. These must be treated as in other febrile diseases.

4. **Complications** and **sequelæ** often require special treatment, and allusion must be particularly made to the management of the *renal* affection. This should be combated by free dry-cupping over the loins, or it may in some cases even be advisable to take a little blood from the renal region; the application of hot poultices over the same region, frequently changed; the employment of hot-air or vapour baths, in order to excite skin-action; purgation by means of jalap and cream of tartar; and a plentiful supply of diluent drinks, with a mild saline mixture. When the acute symptoms have subsided, some preparation of iron is most valuable, especially the tincture of the perchloride; quinine is also a useful remedy at this time. Hæmorrhage from the throat may be troublesome or dangerous, and Mr. Pepper has successfully tied the common carotid artery for bleeding taking place in connection with a post-pharyngeal abscess after scarlatina.

5. For those extremely malignant cases of scarlatina which are attended with early and severe cerebral symptoms no treatment is of much avail, for they generally prove fatal. The use of a hot *mustard-bath* or *cold-water affusion* seem likely to do most good. A blister may be applied to the nape of the neck; and stimulants administered by enemata.

## CHAPTER IX.

### RUBEOLA—MORBILLI—MEASLES.

**ÆTIOLOGY.**—Measles is decidedly infectious, especially when the eruption is out, and its contagium passes off abundantly in the exhalations of a patient, but especially in the breath, the air around being thus contaminated, though it is not very diffusible. The disease spreads readily in houses, or where children are collected together, as in schools. The cough in the catarrhal stage helps its dissemination. It is also conveyed by fomites. Children have undoubtedly taken the disease from sleeping in a bed or room previously occupied by a patient suffering from measles. Inoculation has been accomplished through the blood, serum, nasal and other secretions. Infection probably lasts from the incubation-period for at least a month, and it may be two months. The time it clings to fomites or rooms is uncertain. A second attack very rarely occurs.

Microscopic elements have been described in connection with measles, supposed to be of the nature of specific organisms. First they were obtained by Dr. Ransome from the expired air of patients suffering from the disease. Subsequently Drs. Braidwood and Vacher observed them in the breath, in the true skin, and in the lungs. Dr. Keating has stated that Dr. Formad and himself have found special micrococci in the blood of children suffering from measles of a malignant type; and also constantly in the papules. It cannot be said at present, however, that measles has been definitely associated with any specific organism.

Children are most frequently affected with measles, but those only a

few months old often escape during an epidemic, while no age is exempt. Epidemics are more liable to occur during cold and damp seasons.

**ANATOMICAL CHARACTERS.**—The chief of these is the specific eruption, but this often disappears to a great extent after death. It is due to hyperæmia of the skin, with some inflammatory exudation, and in some forms minute extravasations of blood occur. The conjunctivæ, and the mucous lining of the nasal cavities, their communicating sinuses, the throat, and the air-passages, are in a state of catarrhal inflammation. More or less bronchitis invariably sets in, but in fatal cases it is usually extensive and of the capillary variety, being associated with collapse of the lungs and lobular pneumonia. Laryngitis is not unfrequently present. The blood is dark and fluid in fatal cases.

**SYMPTOMS.—1. Incubation-stage.**—This generally lasts about eight days after exposure to infection. By inoculation measles has been produced in seven days. Out of 50 cases observed by Dr. Squire, 45 had full rash from the tenth to the fourteenth day; in only one case did it appear earlier than this, and in another it was delayed to the eighteenth day. Subtracting four days from these, this will give six to fourteen days as the extremes of the incubation-period. As a rule there are no symptoms during this stage.

**2.—Invasion-stage.**—Measles usually sets in somewhat acutely, with chilliness, actual rigors, or occasionally convulsions. Then follow the ordinary symptoms of pyrexia, but these are not of great severity in the majority of cases, though the temperature rises rapidly to  $101^{\circ}$  or  $102^{\circ}$ , and now and then even to  $104^{\circ}$ . A child attacked with the complaint is languid, irritable, and restless, or may be somewhat delirious at night. The most striking symptoms, however, are those of so-called catarrh or coryza. The eyes are injected and watery, with a feeling of soreness and sandiness, and a dislike for light, the eyelids being also red and tumefied. There is a constant, irritating, watery discharge from the nose, with frequent sneezing, and occasionally epistaxis takes place. A sense of tightness, fulness, or actual pain is experienced over the frontal sinuses. The throat is generally a little sore and red, and the voice somewhat husky or hoarse, but these symptoms are not prominent. There is more or less laryngeal and tracheal catarrh, as well as bronchial catarrh, which is indicated by a sense of tightness and uneasiness over the chest; frequent cough; quick breathing; and wheezing or dry rhonchal sounds or rhonchal fremitus. A little epigastric pain and tenderness is sometimes complained of, or even general abdominal pains, and there may be much vomiting. The bowels are generally constipated, but may be relaxed.

**3. Eruption-stage.**—The rash of measles makes its appearance in most cases on the fourth day, but may come out from the first to the seventh or eighth day. It begins usually on the face, especially on the forehead, then spreads to the trunk, and lastly to the limbs, appearing in these parts in almost distinct crops on three successive days, but occasionally it is first evident on the limbs. Commencing as small scattered red points, these enlarge to the size of a millet seed or a small pea, and become perceptibly raised and papular, eventually being flattened at the summit. They are distinct to the touch, and sometimes have a hard feel.

The eruption tends to form patches of a crescentic, semilunar, or irregularly-circular shape. In some cases it is so abundant as to form extensive, irregular, though well-defined patches, almost uniformly red,



but with elevations upon them; in others it is only sparsely scattered, especially on the limbs, or it may be limited to the face and upper part of the chest. The tint is variously described as rose-coloured, dark raspberry, lilac, and yellowish-red. It is more marked and brighter on exposed parts, such as the hands and face. Pressure removes the colour temporarily, leaving a slight yellowish hue. If the rash is very intense minute vesicles may form, and small petechial extravasations are sometimes observed. It goes on increasing for about twelve hours, and then declines in the same order in which it appeared, the elevations subsiding, and only a reddish or coppery discoloration remaining, the latter continuing for some time. The cuticle desquamates slightly, separating in the form of fine powdery scales, especially on the face and where the eruption has been considerable; rarely does it come off in patches. Occasionally the eruption suddenly recedes.

During the height of the rash the face and hands are often somewhat puffy and swollen. There is frequently much itchiness and irritability of the skin.

The catarrhal symptoms generally increase during the progress of the eruption to its height. The conjunctivæ and mucous membrane of the nose, mouth, and throat are more or less inflamed, and various discharges escape, or ulceration is occasionally produced. Deafness is now and then noticed, owing to the Eustachian tube being involved. The tongue is much furred, usually moist, and presents a few enlarged and red papillæ; it cleans in patches; sometimes it tends to be dry and brown. Vomiting and diarrhœa may be prominent and persistent symptoms.

The chest-symptoms and physical signs indicate more or less extensive bronchial catarrh, the cough becoming moist, with muco-purulent expectoration; and rhonchal fremitus, with various rhonchal sounds, being detected on examination.

The urine is febrile, and abundant lithates are deposited on standing; it has a peculiar odour; and not uncommonly there is slight albuminuria, or sometimes blood is passed. The sweat and breath are said to have a peculiar smell.

**Temperature.**—This increases up to the height of the rash, especially during the later period, there being, according to Dr. E. Fox, a fall before the commencement of the eruption-stage. The temperature does not usually reach above 103°. Morning remissions may be slight, marked, or absent. Defervescence takes place from the fourth to the tenth day, as a rule by rapid *crisis*, the temperature falling 2°, 3°, 4°, or even 5° in 12 hours. After one or two slight evening exacerbations the temperature becomes normal, or falls even below this point for a few days. In rare instances the temperature rises to 108° or 109°. It must be remembered that it may be much influenced by complications.

**VARIETIES.**—The varieties of measles are:—1. **Morbilli mitiores, vulgares, or simplices.**—This is the ordinary form already described. 2. **Sine eruptione.**—Sometimes there is fever and catarrh, but no eruption appears. 3. **Sine catarrho.**—There may be no catarrh, and occasionally even pyrexia is absent, so that the disease is merely indicated by its eruption. 4. **Graviores, malignant, black, or hæmorrhagic.**—This form may depend upon the epidemic character of the disease; or upon an unhealthy state of the patient. At first the symptoms may be mild, or they assume a virulent aspect from the outset. The malignant variety of measles is characterized by typhoid symptoms; severe nervous

disturbance; and irregularities in the eruption. There is great depression and prostration, with a very weak, frequent, and irregular pulse, cold extremities, a dry and brown tongue, and sordes on the teeth and lips. Twitchings, picking at the bed-clothes, convulsions, delirium of a low and muttering character, or stupor, are generally observed at a very early period. The eruption comes out only slightly and irregularly, often receding and reappearing. It is distinctly livid, purple, or black, being mixed with abundant petechiæ, especially about the legs; and sometimes hæmorrhages from mucous surfaces take place. Extensive bronchitis, pulmonary congestion, or pneumonia are liable to set in. Death usually occurs from asthenia, coma, or asphyxia.

COMPLICATIONS AND SEQUELÆ.—These include:—1. Affections of the *respiratory organs* more especially, which may become dangerous during, or subsequent to, the attack of measles. They comprise acute laryngitis, catarrhal or croupous; chronic laryngitis; capillary bronchitis; chronic bronchial catarrh; lobular collapse; croupous pneumonia; catarrhal or broncho-pneumonia; acute or chronic phthisis; very rarely gangrene. 2. Acute tuberculosis. 3. Inflammation about the eyes, nose, or ears, tending to become chronic, and being accompanied with discharges. 4. Inflammation of the glands of the neck and other parts, which may remain permanently enlarged. 5. Severe diarrhœa, which may become chronic. 6. Acute Bright's disease rarely. 7. Diphtheritic or gangrenous inflammation of the labia in exceptional instances. 8. A low state of the general health as a sequela.

PROGNOSIS.—As a rule the immediate prognosis of measles is favourable, but the number of deaths varies much in different epidemics, the average being about 10 to 12 per cent. The mortality is greater in very young children and in elderly persons; in large towns; during cold and damp seasons; in cold climates; and during extremely hot seasons in hot climates. It is also increased by unfavourable hygienic conditions, bad feeding, and debility or impaired health from any cause. The supervention of measles in cases of acute or prolonged chronic disease is often very serious; it is also highly dangerous in scurvy and allied conditions. The chief sources of danger in measles are the pulmonary complications. The malignant variety is necessarily exceedingly grave, and should recovery take place convalescence is much delayed. Sequelæ are very liable to follow measles, and these must be borne in mind when giving a prognosis.

TREATMENT.—Decidedly the principal indication in the majority of cases of measles is to ward off any dangerous complications in connection with the respiratory organs. This is best carried out by keeping the patient in bed, in a room maintained at a uniform temperature of from 60° to 65° F., according to the time of the year, into which steam from a boiling kettle may be admitted. All exposure to draughts must be carefully avoided, until the entire course of the disease has been run through, and the bronchitic symptoms have subsided. The patient must remain quiet, and it is well to darken the room somewhat. Only a liquid diet should be given, but it need not be too low. The bowels must be kept regularly open by the aid of mild aperients. A mixture containing liquor ammoniæ acetatis; vinum. ipecac.; a few drops of tinct. camph. co., if the cough is very troublesome, with camphor mixture, may be administered. If there is much oppression and a sense of tightness about the chest a sinapism or hot poultices or fomentations should be applied. Thirst is to be alleviated by sucking ice, or by means

of small quantities of acid drinks. If there is much heat and discomfort about the skin, careful sponging with tepid water may be resorted to, only a limited surface being exposed at the same time. Should bronchitis become extensive, tending to involve the smaller tubes, it is best treated by giving ipecacuanha wine in moderate doses; avoiding all opiates; encouraging cough; and applying linseed-meal poultices or sinapisms freely over the chest. Local removal of blood to a small extent may now and then be indicated in plethoric children. In most of these cases, however, *stimulants* are called for, such as carbonate of ammonia and chloric ether, with more or less wine or brandy, and abundance of liquid nourishment. If signs of suffocation appear, mustard baths or warm baths with cold douching should be resorted to, as well as artificial respiration. All forms of pneumonia require a stimulant plan of treatment.

Laryngeal symptoms should be treated by the constant application of heat and moisture externally over the larynx; and the assiduous employment of inhalations of steam. Other complications must be attended to should they arise.

Any tendency towards the *typhoid condition* must be combated by the free use of stimulants and nourishing food. If the eruption suddenly recedes, it is recommended to try to bring it out again by means of various baths, and by the administration of warm drinks internally, but this must be done with caution.

During *convalescence* after measles it is necessary to exercise much care for some time; to guard against colds; and, in order to restore the health fully, it is often advisable to give quinine, iron, and cod-liver oil, and to recommend change of air to a suitable climate, as well as cold bathing, especially with salt water. The clothing must be warm, and flannel should be worn next the skin. Sequelæ not uncommonly require attention after measles.

---

## CHAPTER X.

### RÖTHELN—RUBEOLA NOTHA—EPIDEMIC ROSEOLA— GERMAN MEASLES.

It is necessary to consider briefly an affection to which the above and other names have been applied, which is now becoming generally recognized as an independent complaint.

**ÆTIOLOGY.**—Rötheln has been regarded either as a mild form of measles or scarlatina; or as a combination of these two diseases, and hence termed *hybrid measles* or *hybrid scarlatina*. There is abundant proof, however, that it is quite distinct from both these complaints, though resembling them in many of its features, and that it is undoubtedly an independent *acute specific fever*, propagated by a specific contagium. The subject was thoroughly discussed at the International Medical Congress, and almost all the speakers agreed in this view. Dr. Robert Livinge believes that rötheln is far less contagious than either measles or scarlatina, but that it is more distinctly epidemic, at least in this



country, even than ordinary measles, and certainly more so than scarlatina. The contagium probably comes off in the breath and cutaneous exhalations. The disease only occurs once usually. Adults may be attacked as well as children.

**SYMPTOMS.**—Rötheln is almost invariably a very mild disease, but in exceptional instances it assumes a serious character, and in the discussion above alluded to Dr. Cheadle gave an account of two epidemics, in which the symptoms were of a severe and even malignant character, but which he considered nevertheless to be of the nature of rötheln.

1. **Incubation-stage.**—This usually lasts about twelve days, but may extend to twenty days. There are no symptoms.

2. **Invasion-stage.**—Rötheln may be ushered in with slight shiverings, pains about the body and limbs, and pyrexia, while sore-throat is almost always complained of, which differs from that of scarlatina in being much less severe, the local inflammation very seldom going on to ulceration. The glands of the neck are enlarged. In many cases, but not nearly so frequently, signs of catarrh are present, such as are observed in measles. The intensity of the symptoms is generally comparatively mild, and not uncommonly they are quite insignificant; though, on the other hand, they may be rather severe, the temperature running up to  $103^{\circ}$ , or even higher in some instances; usually it reaches its height on the second day.

2. **Eruption-stage.**—The eruption of rötheln, which is in the form of a rash, comes out generally on the second day, or even within the first twenty-four hours; it may be delayed, however, until the third or fourth day. Its amount is usually in proportion to the intensity of the early symptoms. It appears more or less simultaneously over the body, but rather earlier on the face and chest, and is less marked on the limbs than elsewhere. At first the rash resembles that of measles, consisting of a number of minute red papules, which become grouped in patches, but these are more irregular and less distinctly crescentic than the patches observed in measles. The colour is also rather brighter than in this disease, and it is deeper at the centre than towards the circumference of the patches; a brownish hue has been described. In some instances the groups coalesce either partially or completely, so that the skin is uniformly red over a greater or less extent of surface, and then the rash becomes more like that of scarlatina. The eruption lasts longer than that of either measles or scarlatina, its duration being never less than four or five days, and it may continue for eight or ten days. Slight desquamation of the cuticle follows, but this is never a prominent feature, being merely of a furfuraceous character. When the rash appears, the general symptoms abate considerably in most cases, but the sore-throat persists, being often the last symptom complained of, and continuing sometimes for several days after the rash has disappeared.

Rötheln seems to be unattended by any particular *complications* or *sequelæ* as a rule. Slight albuminuria is not uncommon, but it soon passes away. In rare instances acute renal disease with dropsy sets in; and I believe that I have met with a case in which a fatal result has thus been brought about.

**PROGNOSIS.**—This is highly favourable, death from rötheln being an exceedingly rare event, while recovery is usually speedy and complete.

**TREATMENT.**—All that is needed is to keep the patient in bed; to give a liquid diet; and to administer an aperient if necessary, with some

simple saline mixture. Should the throat be very sore, it may be gargled with milk and warm water. Of course the treatment must be modified in severe cases, according to the symptoms present.

---

## CHAPTER XI.

### VARIOLA.—SMALL-POX.

**ÆTIOLOGY.**—The *specific poison* which generates small-pox is very easily conveyed from one individual to another by inoculation, contact, and infection. It exists in the blood, in the contents of the eruption, and in the substance of the dried scabs, being also given off abundantly in the various secretions, excretions, and exhalations, especially in those emanating from the lungs and skin. Inoculation can be accomplished through the blood, as well as most effectually through the contents of the eruption, and the dried scabs which follow the pustules. Small-pox is one of the most infectious diseases, and its contagium can be conveyed to considerable distances. It is exceedingly dangerous to be in the vicinity of a patient suffering from this disease, even in its mildest form, as this may originate the most virulent type. The fluid obtained from the small-pox eruption can be kept for an indefinite time between two pieces of glass, or on ivory points, and still retains its contagious properties. The poison also clings tenaciously to fomites, especially to clothes and other articles of a rough texture, and retains its vitality for a long period; therefore it is highly dangerous to go into a room which has been occupied by a small-pox patient until this has been most thoroughly disinfected, while clothing and other articles will certainly propagate the disease unless treated in a similar manner. There can be no doubt but that the disease has been not uncommonly conveyed by the agency of cabs. I have known several instances in which persons suffering from mild variola have come as out-patients to hospitals, and as they may remain for some time among the other patients, it is not at all improbable that the affection is sometimes disseminated in this way.

Experiments have been made by Chauveau, Burdon Sanderson, Braidwood and Vacher, and other investigators, with the view of discovering the exact nature of the contagium of small-pox, and it has been supposed that this consists in certain minute particles which are present in the contents of the eruption. Klein has described peculiar organisms in connection with sheep-pox, which is identical with small-pox, but the observations of Dr. Creighton and others throw much doubt upon their existence.

Micrococci take a prominent part in developing the eruption, and are also present in connection with certain secondary lesions which occur in internal organs, but Dr. Sanderson thinks that we have no evidence that they carry with them the specific contagium of the disease.

The safer conclusion as regards the time during which infection lasts

is to consider it as beginning with the earliest appearance of symptoms, and continuing for some time after the eruption has disappeared. The danger is greatest during the period of suppuration. It is very important to bear in mind that the bodies of those who have died from small-pox may undoubtedly convey infection. A second attack is only rarely met with, but even a third may occur.

*Predisposing causes.*—Variola may be met with at any age. It attacks most commonly and most severely those who have either not been vaccinated or re-vaccinated at all, or only inefficiently. Constitution and race seem to influence the occurrence and characters of the disease. Some individuals resist all infection, and cannot even be inoculated. The negro and dark races generally suffer severely. Dread of infection is said to act as a predisposing cause of small-pox. The lower classes suffer most, for obvious reasons.

*ANATOMICAL CHARACTERS.*—The eruption of small-pox constitutes one of its most prominent anatomical characters, and it is the result of circumscribed inflammation of the skin, extending more or less deeply. There is first congestion, which some believe begins in the follicles of the skin. Then the papillæ enlarge, and the cells of the rete mucosum increase, thus giving rise to papules. A layer of soft whitish exudation is described as forming between the cuticle and the true skin, due to proliferation of the cells of the rete mucosum. Next a clear fluid collects beneath the superficial layer of the epidermis, a vesicle being thus formed, and subsequently pus is produced. The pustules either rupture or dry up. The true skin may be extensively involved and destroyed.

Drs. Braidwood and Vacher examined sections of human variolous skin at different stages, and the following is the description which they give of the appearances seen:—

“Under a lower power the human variolous vesicle on the fourth day of the eruption shows on section corpuscular infiltration of the rete and corium, with breaking down of the middle layers of these tissues in the centres of the vesicles. The capillary and glandular elements of the true skin are seen destroyed, the lymph-spaces and vessels are filled with nucleated cells, the walls of the capillaries thickened, and the sudoriparous glands (in the lowest portion of the corium) filled from over-development of their epithelial cells.

“Examined under a high power, the superficial layer of the rete Malpighii is observed compressed to form a limiting membrane outwards, while the deepest layer of the corium acts similarly in separating the cavity of the vesicle from the subcutaneous areolar tissue. The corpuscular elements infiltrating the true skin are seen as round or oval nucleated cells, with well-defined margins, scattered in the tissue at the circumference of the vesicle, as shrunken or compressed, irregularly-shaped, deeply-tinged cells closely packed together in the centre of the vesicle, with here and there large round spaces enclosing groups of small nuclei or nucleated cells. The process of re-production by gemmation is detectable at this stage of the disease.

“A section of human skin occupied by a variolous vesicle of the seventh day, or stage of maturation, exhibits under a low power the increased density of the deep layer of the corium, sending broad processes into the subcutaneous areolar tissue, whilst the cavity of the vesicle itself appears more or less empty and contracting. The elements of the true



skin are no longer distinguishable, excepting a few remnants of lymph-spaces. Under a high power the variolous vesicle at this date shows its cavity more or less intersected by bands of connective tissue enclosing irregularly-shaped nucleated corpuscles. Along the margins of the hairs and their follicles are observed nucleated cells closely packed together, and some of them elongated into fibres. The deep layer of the corium is seen firmly matted together into broad bands of parallel fibres. The process of reproduction is not so distinct at this stage, but the crowding of the sudoriparous glands with nucleated cells is well seen.

“Human variola of the twelfth day of the eruption exhibits under a low power the separation of the rete from the corium by means of the corpuscular infiltration which had taken place, the destruction of the elements of the tissue, the connective-tissue transformation of the deep layer of the corium, and the engorgement of the lymph-system of the true skin. Under a high power the corpuscular character of the changes in the true skin is well seen. The limiting layer surrounding the vesicle, observed so distinctly on the seventh day of the eruption, is now absent, the hair-follicles and glands are almost entirely destroyed.”

Mucous surfaces are not uncommonly affected in small-pox, especially the conjunctiva, and the mucous membranes of the mouth, throat, and nose. Occasionally the whole extent of the respiratory or alimentary mucous tract is involved. There may be merely inflammation, or it is said the specific eruption is sometimes observed. Other mucous surfaces are also implicated, and the eruption has been described as occurring even on serous surfaces. Various organs are often inflamed, as well as serous membranes, especially the pleuræ, and the exudations resulting therefrom are liable to be of a low type, or of a sanguineous nature. Dr. Weigert observed that early in the suppurative stage of small-pox cylindrical masses are seen under the microscope in various organs, particularly the liver, spleen, lymphatic glands, and kidneys, consisting entirely of capillaries plugged with micrococci, which have found their way from the eruption into the circulation, probably through the lymphatics. Afterwards, although the organs appear quite healthy to the naked eye, each plug becomes surrounded by a zone of altered tissue, the nuclei of the cells being wasted. Ultimately infiltration with leucocytes may take place, and a circumscribed miliary abscess is formed, with a surrounding zone of congestion. This, however, probably depends upon the quantity and progress of the cutaneous suppuration, and on the degree in which the circulation becomes impregnated with septic material (Burdon Sanderson). The heart, kidneys, liver, and voluntary muscles generally are found to be very soft in fatal cases of small-pox, being the seat of acute fatty degeneration. Putrefaction proceeds rapidly after death.

**SYMPTOMS.—1. Incubation-stage.**—After inoculation of the small-pox virus the first symptoms appear in seven days. When communicated by infection, the incubation-period is almost always twelve days, but it may be a day or two longer or shorter. During this period there may be some feeling of general illness, but no definite symptoms are noticed.

**2. Invasion-stage.**—Small-pox usually commences suddenly, with chills or repeated and well-marked rigors, followed by pyrexial symptoms, constituting the *primary fever*. The temperature rises rapidly, and may reach 104° or 106° before the eruption appears. Along with the usual symptoms of fever, which are generally severe,

there are others of a very significant character, namely, a feeling of marked uneasiness and fulness or actual pain in the epigastrium, with nausea and more or less obstinate vomiting; pains over the body generally, but especially in the middle of the back, opposite the lower dorsal, lumbar, and sacral regions, this pain not being aggravated by movement; and considerable debility and sense of illness, with tremulousness of the muscles. Even in mild cases it is often remarkable how prominent these symptoms are. Much headache is usually complained of, while the face is flushed, and the carotids throb. In some cases the disease sets in with marked nervous symptoms, such as restlessness, delirium, somnolence, stupor, coma, or convulsions, the last-mentioned being particularly frequent in children. Occasionally there is considerable sore-throat or coryza. The severity of the symptoms at this period is generally in proportion to that of the subsequent stages.

3. **Eruption-stage.**—Of late years particular attention has been called to the fact that in some epidemics of small-pox the eruption characteristic of this disease is often preceded by “prodromal exanthems.” They are met with very irregularly in different epidemics, and the percentage of cases in which they occur is also very variable. These exanthems appear from one to five days before the small-pox eruption, and are described as presenting two varieties, the *diffuse scarlatiniform*, and the *macular* or *measly*, generally accompanied with petechiæ. In exceptional instances an *urticarial* rash has been seen. Their extent is very variable; the rashes without petechiæ may cover large areas, or even the entire body, but in other instances they are limited to certain regions, and this is usually the case with regard to the purpuric extravasations. The favourite regions are the lower part of the abdomen, the inner surface of the thighs, the lateral region of the thorax along the margin of the ribs, the axillæ, the exterior surfaces of the extremities, especially near the knees and elbows, the backs of the hands and feet, and the genitals. These initial rashes may be useful in the diagnosis of cases of small-pox before the true eruption appears, especially the petechial form, and particularly if occupying the regions mentioned above.

The characteristic eruption of small-pox appears usually during the third day, but may be delayed until the beginning of the fourth. The face is almost invariably its primary seat, especially the forehead, but in rare cases it commences about the wrists. It spreads over the body and limbs in from one to two days, and is described as forming three successive crops. The number of spots or “pocks” varies from just a few to thousands, but as a rule from 100 to 300 are present. On the face they are more abundant than elsewhere. They are either distinct, or run together in different ways, thus giving rise to certain varieties which will presently be alluded to.

*Characters.*—If a typical individual pock be observed, it will be found to pass through the following course. It starts as a minute bright red spot, a little raised; enlarging and becoming more elevated, it forms a distinct papule on the second or third day, well-defined, flattened at the top, and having a peculiar solid, hard, dense feel, compared to that of a shot or mustard-seed under the skin, which is very characteristic. This soon changes into a vesicle, a little clear thin fluid collecting in the centre, under the epidermis. About the fifth day a depression forms on the top of the pock, which thus becomes umbilicated; at the same time the contents gradually assume a purulent character, this change begin-

ning at the circumference, the central part still for some time remaining vesicular, and this vesicular portion is stated to be distinctly separated from the surrounding pus by a transverse partition, so that the two spaces may be severally emptied of their contents. At this time a distinct ring of inflammatory redness appears around each pock. The pus increases in quantity, and after a while the umbilication disappears, the pock becoming either rounded or pointed at the top. Its interior is divided into a variable number of areolæ or compartments, sometimes regularly arranged and of equal size, but usually irregular and unequal, the partitions being formed by the white substance already mentioned. With regard to the cause of the umbilication, it has been attributed to the passage of a gland-duct or hair-follicle through the pock; or to a central organic connection between the epidermis and cutis, which finally gives way.

About the eighth day the pustule is at its height, having arrived at the end of the stage of *maturation*. Then it undergoes retrograde changes. It either ruptures, its contents being discharged and drying up, so as to form a yellowish-brown scab; or it shrivels and dries up without rupturing. The scab separates from the eleventh to the fourteenth day, leaving a stain of a reddish-brown colour, which remains for a variable period. If the cutis is at all destroyed, a pit is left of greater or less depth, which ultimately becomes of a dead-white colour.

The course of the eruption just described is modified by circumstances which will be alluded to presently. It is generally completed on the face before other parts of the body.

The appearances and symptoms associated with the eruption will vary according to its amount. If it is at all considerable, there is tumefaction and puffiness of the scalp, face, neck, and other parts, accompanied with a sensation of throbbing and tightness. The eyelids may be so swollen as to be completely closed. The skin is generally of a deep red colour between the pocks, and feels more or less sore and tender. There is almost always most uncomfortable itching, which induces patients to scratch themselves, thus causing much soreness and ultimate disfigurement. A characteristic, unpleasant, sickly odour is exhaled from the body.

The eruption not unfrequently involves the mucous membrane of the mouth and throat, this being accompanied with soreness, salivation, and dysphagia. There is a discharge from the nostrils, or these passages are blocked up. Sometimes the larynx, trachea, and bronchi are involved, as indicated by hoarseness, cough, and more or less dyspnoea. The urino-genital mucous membrane is often affected, causing much pain and soreness, with dysuria, and sometimes hæmaturia. It is said that the eruption may even be met with in the rectum, or along the whole course of the alimentary canal, but this is a doubtful statement. Diarrhoea is not an uncommon symptom in cases of small-pox.

The conjunctiva is frequently in an irritable and inflamed condition, and hence a burning sensation is experienced about the eyelids, accompanied with inability to bear the light, and increased secretion of tears. Occasionally a pustule forms on the ocular conjunctiva, which may lead to ulceration and destruction of the cornea.

*Secondary fever.*—When the eruption of small-pox appears, the primary fever abates rapidly, so that the temperature falls nearly or quite to the normal in moderate cases, the patient feeling as if convalescent. When suppuration commences, however, *secondary* or



*symptomatic fever* sets in, depending upon and being in proportion to the morbid process in the skin, and subsiding as this subsides. It often begins with rigors or chills. The pulse becomes frequent, and there is much thirst, with dryness of the tongue and mouth. The *temperature* rises to  $104^{\circ}$  or  $105^{\circ}$  in a typical case, reaching its maximum when suppuration is at its height, but it may be considerably above this. Defervescence is gradual, and there may be another elevation of temperature when desiccation occurs.

The urine is febrile so long as the pyrexial condition lasts; sometimes it contains a little albumen; or in cases of a low type more or less blood may be mixed with it.

**VARIETIES.—1. Discrete.**—In this variety the pocks are distinct, and do not run into each other, though some of them may touch. They are never numerous, and there may be but a few scattered about here and there. The symptoms are usually mild, but their severity depends upon the amount of the eruption.

**2. Confluent.**—This is a very serious form, in which the eruption is abundant, and the pustules run into each other. The symptoms of the invasion-stage are severe, and nervous symptoms are often prominent. The eruption-stage commences earlier, and there is not at this time such a distinct remission in the fever as is observed in ordinary cases. The eruption is often preceded by prodromal rashes. Very numerous papules appear, small and but slightly prominent, arranged in groups or irregularly, and they quickly spread over the whole body. The course of the eruption is more rapid than usual, the vesicular and pustular stages being soon reached. The pustules present variable appearances, but they are often extensive and flat, and there may be large bullæ filled with pus. The face appears sometimes "as if covered with one bladder of matter." The contents are in some cases serous and watery, or bloody, and very offensive. Frequently there is no distinct red areola around each pustule, but the general surface of the skin is of a dark red colour. Extensive crusts form after desiccation, often of a dark colour and soft, which do not separate for some time. Confluence is usually most evident and most serious about the scalp, face, and neck; the face may ultimately be covered with a continuous crust, like a mask. The true skin is destroyed more or less, and extensive pits, scars, or seams are left, which tend to contract, thus causing great disfigurement. The eruption is generally abundant on the mucous surfaces, giving rise to the various symptoms already mentioned.

Secondary fever does not stand out so distinctly in the confluent as in the discrete variety of small-pox, and adynamic symptoms are apt to set in, with intense nervous depression. Complications and sequelæ are also very liable to arise, many of them of a serious and dangerous character. This form is exceedingly fatal, and if recovery takes place convalescence is generally very slow. Occasionally, however, cases of confluent small-pox are met with which run a remarkably favourable course.

**3. Semi-confluent or Coherent.**—This is an intermediate variety, in which the pocks are not quite distinct, but do not actually run into each other. It is not a dangerous form.

**4. Corymbose.**—Here the eruption is arranged in clusters like bunches of grapes, and it exhibits a tendency to symmetry. It is said to be a very dangerous and fatal variety.

5. **Malignant.**—Under this term several forms of small-pox which are occasionally observed may be included. In some instances an individual is attacked with severe primary fever, attended with low symptoms, and dies from the intensity of the action of the poison before the eruption can appear. Other varieties of malignant small-pox are named *black* or *hæmorrhagic*; *petechial*; *ulcerative*; and *gangrenous*: these terms sufficiently indicate their several characteristic features.

In the *hæmorrhagic* form there is intense adynamia and nervous prostration from the first, with delirium, great restlessness, somnolence or a tendency to coma. The face is sunken and anxious, and the breathing hurried. The eruption is slow and irregular in its development, sometimes receding; it tends to be livid or black, and if pustules are formed, they contain blood. Petechiæ are observed also between the pocks, and hæmorrhage takes place from various parts.

6. **Benigna — Verrucosa — Cornea — Horn-pock or Wart-pock.**—This is a mild and abortive form, in which the pocks do not become purulent, but shrivel and dry up on the fifth or sixth day. There is no secondary fever, and the duration is shorter than usual. It generally follows vaccination. Another mild variety is that in which the eruption continues vesicular to the end—**crystalline-pock**.

7. **Variola sine eruptione.**—It is believed that in persons well protected there may be primary fever without the subsequent appearance of any eruption.

8. **Anomalæ.**—This term is applied to variola occurring along with other exanthemata, during pregnancy, in the foetus, or under other unusual conditions, and thus presenting irregularities.

There are two modifications of small-pox which call for special notice.

**Inoculated Small-pox.**—When the poison of variola is directly introduced by inoculation, the course of events is usually as follows:—On the second day a slight discoloration is observed at the seat of inoculation, and on the fourth or fifth day this is somewhat inflamed and irritable, a small vesicle forming, which enlarges, and becomes surrounded by an inflamed areola about the seventh day. From this time to the ninth day the ordinary primary fever sets in, and in three or four days more the general eruption appears, the original vesicle having in the meantime become pustular and being now at its height, after which it undergoes retrograde changes. The important fact in connection with the inoculated disease is, that in its entire course it tends to be exceedingly mild, the number of pustules being very limited. Occasionally, however, small-pox thus originated assumes a serious or even fatal form.

**Small-pox after Vaccination — Varioloid — Varicelloid.**—There can be no question as to the fact of variola being prevented or greatly modified by efficient vaccination or re-vaccination. The principal effects of vaccination may be thus stated:—1. It sometimes prevents any eruption, there being merely a slight primary fever for three or four days. 2. It diminishes the number of pocks. In some instances there is marked fever, which ushers in a slight eruption, it may be but a single pock. 3. It modifies and shortens the course of the eruption and thus diminishes considerably the secondary fever and its accompanying dangers, as well as the destructive effects upon the skin, with consequent disfigurement. In some instances the eruption, even if extensive, does not go beyond the papular or vesicular stage; in others, though it

becomes pustular, its course is more rapid, the pustules drying up on the sixth or seventh day. Generally they are small, and do not present umbilication. The unpleasant odour is usually absent. In some cases the eruption is preceded by an erythematous or roseolar rash, and it may appear first on the trunk. As the result of desiccation, flat thin crusts are formed; or hard shining scales; or occasionally little tubercles. There is either no pitting at all, or this is very slight.

COMPLICATIONS AND SEQUELÆ.—These are very frequent, especially in connection with the more severe forms of small-pox, the more important being:—1. Affections of the respiratory organs, namely, low forms of pneumonia; pleurisy, rapid in its progress and very dangerous; bronchitis, or inflammation of the general respiratory tract, with the formation of much thick purulent material; occasionally œdema glottidis. 2. Affections of the alimentary canal, such as severe glossitis, gastritis, enteritis, profuse diarrhœa. 3. Various local inflammations and abscesses, namely, over parts that are pressed upon; in the subcutaneous cellular tissue; or in the deep structures of the limbs: along with which may be included boils, which often come out in large numbers, and carbuncles. The pus is generally very unhealthy and sanious. 4. Gangrene of certain parts, such as the scrotum or labia. 5. Erysipelas, especially of the head and face; ecthyma, rupia, or eczema. 6. Pyæmia or septicæmia, from absorption of septic materials. 7. Affections of the organs of sense, which are not uncommonly very destructive, especially ophthalmia; ulceration of the cornea; otitis with purulent discharge, ending in caries of the bones; and destructive inflammation of the nose. 8. Urinary complications, including cystitis; retention and subsequent incontinence of urine; renal congestion, with albuminuria and casts; or abscess of the kidney. 9. Inflammation of the ovaries or testicles. 10. Various hæmorrhages, especially hæmaturia, menorrhagia, hæmoptysis, and epistaxis, accompanied with petechiæ. 11. Peritonitis in exceptional cases.

PROGNOSIS.—Small-pox is a very grave disease, the proportion of deaths being exceedingly high, averaging about 1 in 3 cases. Death may take place at any period of its course, but occurs most frequently between the eighth and thirteenth days, and especially on the eleventh. The usual causes of death are high fever; adynamia; apnœa; pyæmia or septicæmia; direct loss of blood; or, at a later period, asthenia.

The chief circumstances which influence the prognosis are:—1. *Age*. Small-pox is very fatal in children under five years of age; and in persons who have passed middle life. From 10 to 15 is the most favourable period as regards prognosis. 2. The *hygienic conditions* surrounding the patient, the prognosis being worse if these are unfavourable. 3. The *previous habits and health* of the patient, intemperance, debility from any cause, or any organic disease being injurious. 4. Whether *satisfactory vaccination* has been accomplished or not. 5. The nature and intensity of the *symptoms*. Among the signs of evil import are recognized a very high temperature; persistent and excessive lumbar pain; severe vomiting after the appearance of the eruption; as well as all symptoms of an adynamic or malignant character, with nervous depression. 6. The amount and characters of the *eruption*. The gravity of a case is in proportion to the confluence of the eruption, and to the rapidity of its extension. The corymbose variety is very fatal. Other dangerous signs in connection with the eruption are imperfect development of the pustules, or their sudden subsidence;



lividity, hæmorrhage, or gangrene in connection with them; the presence of petechiæ; or pallor with absence of swelling between the pustules. 7. The *complications* and *sequelæ*. These materially influence the prognosis, and especially the complications referable to the respiratory and nervous systems. Hæmorrhages are also of very serious import. Convalescence is often much delayed by complications and sequelæ. Pregnancy is a peculiarly dangerous condition if associated with small-pox. Abortion usually results, and the termination of the case is generally fatal. In some instances the eruption is observed over the fœtus. 8. *Epidemic constitution*. Some epidemics are comparatively mild, others very grave.

**TREATMENT.**—The indications of the treatment of small-pox may be laid down as follows:—1. To pay strict attention to all hygienic conditions, as well as to diet. 2. To prevent a copious eruption, and endeavour to cause this to pass through its different stages as mildly as possible, checking extensive suppuration and destruction of the skin, especially about the face. 3. To subdue excessive pyrexia. 4. To sustain the strength of the patient during the process of suppuration. 5. To treat symptoms, which are often distressing. 6. To guard as much as possible against all complications, and treat them as they arise. 7. To promote convalescence, and attend to sequelæ.

1. **General management.**—Even in the mildest cases patients suffering from small-pox should be strictly confined to their rooms. Free ventilation is essential, and the apartment should be large and moderately cool, all carpets and curtains being removed, as well as excessive bed-clothes, due care being taken to protect the patient against draughts. Cleanliness is also most important, the linen being frequently changed, and at once subjected to the action of some disinfecting agent. In the early period a low diet is called for as a rule, with plenty of cooling drinks or ice, as well as fruits, especially roasted apples, while stimulants must be avoided. Later on it is generally necessary to alter the diet gradually, and to have recourse to beef-tea, soups, jellies, and such aliments, as well as to alcoholic stimulants, the nature and quantity of these being regulated by the circumstances of each individual case. In all cases of a low type, and especially when there is much suppuration, considerable support is needed in the way of nutritious food and stimulants, and if there are indications that the patient will have to struggle through a long process of suppuration, it is important not to allow the strength to become too much reduced, but to employ supporting measures carefully from the first.

2. The management of the **eruption** has always naturally attracted considerable attention. At one time it was the custom to keep patients suffering from small-pox very warm, and to give them hot drinks, with the view of “bringing out the eruption,” but at the present day the object aimed at is to limit this as much as possible, and to modify its course, so as to prevent the dangers of excessive suppuration, and the subsequent pitting and disfigurement. The skin must be sponged freely with lukewarm water, to which may be added some *antiseptic*, such as carbolic acid, Condyl’s fluid, chlorine water, or sulphurous acid. It has been recommended to apply carbolic acid and oil freely over the surface, but this proceeding is of questionable propriety. Some consider that the eruption is best checked by keeping the patient in a dark room, but this practice has been condemned by others. The puncturing of each pock as soon as pus forms has also been advocated. Many *local applica-*

tions have been made use of in order to prevent pitting, the chief of which are nitrate of silver, either applied in the solid form to each pustule, or brushed over the surface as a solution; mercurial plaster or ointment; solution of corrosive sublimate (gr. ij ad ʒ vi); sulphur ointment; tincture of iodine; gutta percha dissolved in chloroform; and carbolic acid, alone or mixed with glycerine or oil. Most of these are very irritating, and require much care in their employment. Dr. Sansom advocates touching each pustule with carbolic acid, and then applying a mixture of this substance with oil of thyme. All the pustules should not be touched at one time, but they should be attacked on successive occasions. Other plans are to cauterize each pock with carbolic acid on the first or second day of the vesicular stage; or to open the vesicles when at their height, and dab the surface with carbolic lotion. Mr. Marson recommends waiting until the pustules have discharged their contents, and then applying either olive oil, alone or mixed with lime-water or calamine; a mixture of glycerine and rose-water; or cold cream and oxide of zinc. He warns against allowing the scabs to dry, and to remain for some time on the nose and other parts of the face. Of course the patient must be prevented as much as possible from scratching. The irritation excited by the acrid secretions is best relieved by frequent sponging, and by the free use of some absorbent powder, such as flour, starch, hair powder, or calamine. If there is much eruption on the scalp, it is necessary at an early period to cut the hair very short, or even to shave the head.

3. In most cases of small-pox **pyrexia** can be kept within limits by sponging the skin; and by the administration of cooling drinks, with a *diaphoretic saline* mixture. A brisk *purgative* is advisable at the outset, and the bowels should be kept freely open afterwards. If there is a tendency to hyperpyrexia, full doses of quinine (gr. iii-v every three or four hours) seem to answer best in this disease. Venesection is never called for.

4. During the **suppurative stage** *tonics* are needed, such as quinine, iron, or mineral acids with decoction of bark. If there are adynamic symptoms, these remedies must be given freely, along with ammonia, camphor, and other *diffusible stimulants*; as well as with plenty of nourishing food, and wine or brandy.

5. The chief **symptoms** which may require to be treated in cases of small-pox are vomiting or diarrhœa; restlessness, sleeplessness, or delirium; soreness of the throat; and hæmorrhages. It is recommended to give morphia for one or two nights, in order to get the patient into the habit of sleeping. Caution must be exercised in the administration of *narcotics*, should there be much bronchial catarrh or salivation. Delirium in small-pox is frequently an indication for the free use of stimulants. Physical restraint may be required, and benefit is sometimes derived from the use of the warm bath. Sore-throat is best relieved by the use of some mild gargle, or by sucking ice frequently, or taking a little currant jelly. Hæmorrhages call for the administration of full doses of tincture of steel, tannic or gallic acid, turpentine, or ergot of rye, some of which may be given in combination. Retention or suppression of urine is said to occur sometimes, but in the Small-Pox Hospital this symptom has never been found to give any trouble. The catheter must be employed should the urine be retained.

6. The **complications** which it is specially necessary to guard against and to be on the watch for, are those connected with the respi-

ratory organs and the eyes, as well as various abscesses. Inflammatory affections usually call for a stimulating plan of treatment, and rarely is any removal of blood required; occasionally it may be advisable to apply a few leeches. If there is much bronchitis, the patient must be encouraged to cough frequently. All abscesses should be speedily opened. Should there be any purulent discharge, particular attention is required as regards cleanliness.

In order to prevent complications connected with the eyes, it is recommended to apply cold water constantly; or to use compresses of a weak solution of corrosive sublimate (gr. i ad 3 vi). Should either of these complications arise, a supporting treatment is indicated. A blister over the temple often does good if there is much conjunctivitis. Marson recommends the use of poppy-fomentations with alum. It may be necessary to touch an ulcer of the cornea with a pointed stick or a solution of nitrate of silver. A green shade should be worn.

7. During **convalescence** good diet and *tonics* are required, and cod-liver oil is often very useful. As soon as the patient is in a fit condition, warm baths should be employed, carbolic soap being freely used. Sequelæ must be attended to, if they should occur, and certain cutaneous sequelæ may require special treatment.

8. **Special treatment.**—Numerous special methods for the treatment of small-pox have been brought forward, but the only one that calls for separate notice is the treatment by *antiseptics*. On the whole the balance of opinion seems to be in favour of the internal administration of these remedies, but some practitioners have not found them so valuable as others, and there is decidedly no unanimity of opinion as to which antiseptic answers best. Different observers advocate the administration of carbolic acid, sulpho-carbolates, sulphurous acid, sulphites, or hypochlorites. At the same time *tonics*, such as quinine or iron, may be given.

9. **Preventive treatment.**—The rules for preventing the spread of contagious diseases should be rigidly carried out in the case of small-pox. Patients who have suffered from this disease must not be allowed to mingle with healthy persons until they are quite convalescent, and have been completely disinfected. Rooms which have been occupied by such patients, as well as clothing worn by them, must also be thoroughly cleansed and disinfected. Any articles used for cleansing the skin, such as pieces of sponge or rags, should be immediately destroyed. The great prophylactic against small-pox, however, is *satisfactory vaccination and re-vaccination*, as will be pointed out in the next chapter. *Inoculation with variolous virus* has been practised, with the view of producing a mild type of the disease, but this is only justifiable under certain rare circumstances, namely, when small-pox breaks out among a number of people in a confined space, and no vaccine-matter can be obtained, as for instance, on board ship out at sea.

---



## CHAPTER XII.

## VACCINIA—COW-POX.

**ÆTIOLOGY.**—Vaccinia is an acute specific disease, originating from a *specific virus*. In the cow, especially the milch-cow, it occurs as a natural malady, either sporadic or epizootic, running a definite course, and attended with the formation of a vesicular eruption near the udder and on the body of the teats. In the human being it is only induced by direct inoculation, either of the matter taken from the cow or calf, or of that conveyed from one individual to another. Many believe that vaccinia is identical in its nature with variola, only modified by its occurrence in another animal, and many observations and experiments have been made which seem to support this notion. The contagium of this affection has been proved to consist in certain minute granules present in the vaccine-lymph, which are regarded as micrococci. Godlee and others have cultivated them, but Quist is the only one who affirms that he has vaccinated successfully with the cultures. He says that bacilli are also present, and are developed from the micrococci. The experiments of Chauveau, Sanderson, and Braidwood and Vacher, seem to be conclusive as to the inertness of the soluble constituents of vaccine-matter.

**METHODS OF VACCINATION, AND PRECAUTIONS TO BE OBSERVED.**—The great majority of practitioners vaccinate with “humanized lymph,” that is, with vaccine originally derived from cows, it being unknown how the cows were infected, but afterwards transferred from one child to another. Another method is to inoculate cows with human smallpox, and then to inoculate human beings with lymph thus produced, and transfer it from man to man. There seems to be positive proof that the lymph does not deteriorate or lose its protective power after passing through any number of individuals. The matter should, if possible, be inoculated when fresh, being inserted directly from arm to arm. Often, however, this is not practicable, and the lymph has therefore to be collected in glass tubes, on ivory points, or on little plates of glass, and used subsequently. It has been recommended to mix the lymph in a watch-glass with twice its quantity of pure glycerine and water, and to preserve this mixture in capillary tubes, which is said to be equally effective. The matter should always be taken from a perfectly healthy child; from thoroughly characteristic vesicles; and on the eighth day. Several punctures are to be made on the summit of each vesicle, so that no blood shall be mixed with the lymph, and all pressure must be avoided, only such fluid as escapes spontaneously being made use of. If dried lymph is employed for vaccinating, it must be rendered liquid by mixing it with a very minute quantity of water. Inoculation with “calf-lymph” is now practised by many, the humanized cow-pox being inoculated into calves, and then transmitted from calf to calf, or from the calf to man, but not from man to man.

Vaccination ought to be performed when children are very young, that is, from six weeks to three months old, provided other circumstances are

favourable. It is most important that they should be in good health at the time, and especially that they are free from skin-affections, and from acute disorders, such as diarrhœa. If small-pox is in the neighbourhood, however, vaccination should be performed under any circumstances, and at the earliest age, even immediately after birth should there be great risk of infection. If children are weakly, and there is no urgency, vaccination may be delayed for a year or two. Of course no subject is too old to be vaccinated, if the operation has not previously been satisfactorily done. When vaccination proves unsuccessful, it should be repeated after a short interval.

The part selected for inoculation is the outside of the arm, over the insertion of the deltoid muscle, the skin covering this region being made tense. The chief methods of performing the operation, which should be always carried out thoroughly and carefully, are as follows:—1. By a single or double puncture with a sharp lancet well charged with lymph, this being introduced obliquely under the cuticle into the cutis, so as to make a valvular aperture; the instrument should be left in for a few seconds, and as it is removed the seat of puncture must be compressed. Several special instruments have been invented for this operation. 2. By making a number of minute superficial punctures, or “tatooning” as it is termed, and then applying the lymph with the flat surface of the lancet. 3. By first rubbing in the lymph, then tearing up the cuticle with the lancet over a surface equal to about the area of a sixpenny piece, and finally rubbing in more lymph. Two such patches are sufficient. 4. By scratching the cuticle, and thus producing superficial scarification, the lymph being then applied. Some employ single long scratches, distant half an inch to an inch from each other; the best plan, however, is to make a number of fine parallel scratches over a small area, and others may be made across these. Scarifiers have been invented for this purpose, but the ordinary lancet answers very well. 5. By abrasion of the cuticle with the edge of the lancet, which is used as an eraser is employed to remove blots from paper. 6. By vesication, liquor ammoniæ being applied, and then the cuticle rubbed off, and the vaccine matter applied.

When the surface over which vaccination is performed is small, as in the case of puncture or limited scarification, it is necessary to inoculate in at least five points in the same arm at distances of about half an inch apart, or in three places on each arm.

Revived lymph may either be inserted directly by means of the lancet; or, if it is on ivory points, these may be introduced into punctures, or rubbed on scarified surfaces.

PHENOMENA FOLLOWING VACCINATION.—At the end of the second or beginning of the third day, little papular elevations are visible over the points of inoculation, with slight redness around. The elevation and redness increase, and by the fifth or sixth day distinct vesicles form. These are round or oval, bluish-white, raised at the margin, and depressed in the centre. At the close of the seventh or beginning of the eighth day a circular inflamed areola forms around each vesicle. The latter continues to enlarge during the eighth day, when it is in its most perfect state, being full, tense, rounded and much raised at the margin, and presenting a pearly colour and lustre, with an appearance of translucency. The contents appear clear and slightly viscid, but minute active particles are visible under high powers of the microscope, which are supposed by Dr. Beale to be particles of bioplasm to which the

lymph owes its active properties, by others to be of the nature of micrococci.

The areola continues to extend for a couple of days, reaching a diameter of from one to three inches, and being accompanied with more or less induration and swelling; sometimes small vesicles form upon it. On the tenth or eleventh day it begins to fade, and at the same time the contents of the vesicle become opaque, while it gradually dries up and becomes brown in the centre, by the fourteenth or fifteenth day a hard reddish-brown scab being formed. This darkens in colour, shrivels, and falls off from the twenty-first to the twenty-fifth day, leaving a permanent scar. A typical cicatrix ought to be circular, white, not less than one-third of an inch in diameter, depressed, with minute pits or foveolæ over its base. Sometimes radiations from the centre are observed.

The appearances and course above described may be modified by certain obvious circumstances, or without any evident cause. After some of the methods of vaccination the vesicles are compound or in crops. In adults they do not usually present thoroughly typical characters, on account of the structure of the skin; while their course is often retarded, and the areola is more diffused. Retardation or acceleration of their progress is sometimes observed without any obvious cause. In some cases an entirely irregular and spurious form of eruption is met with. This irregularity is generally due either to improper lymph having been used, to the child being in an unhealthy condition, or to mechanical irritation at the seat of inoculation; but occasionally it cannot be thus explained.

Many irregularities are observed when the lymph is taken immediately from the cow. "Papulation is deferred till the seventh, eighth, ninth, or tenth day, and the areola is not complete till from the eleventh to the fourteenth or sixteenth day, being also harder, and it is said to revive and decline, continuing to exhibit a brick-red or purplish hue while the hardness remains. The vesicles are usually not more developed than those produced by ordinary lymph. Desiccation is prolonged, and the crust is often retained till the fourth or fifth week."

Certain local and constitutional symptoms usually accompany the development of the vaccine vesicles. Itching, heat, tension, and pain are experienced in the arm at the time of maturation, with a feeling of stiffness and difficulty in movement. Occasionally erythema or erysipelas breaks out; or the vesicles may ulcerate or slough. The glands in the axilla are often enlarged and tender, especially in adults. There is no primary fever, but a symptomatic fever is set up during the process of maturation, and it is said that the temperature may reach as high as  $104^{\circ}$ . At this time the child is fretful and restless, and the alimentary canal is often deranged. In rare instances severe or even dangerous symptoms supervene, especially in weakly children. A general rash is sometimes observed, of a roseolar, lichenous, or vesicular character, which does not usually last beyond a week. These eruptions are more common after vaccination direct from the cow.

RE-VACCINATION.—This is often required in consequence of the primary vaccination having been insufficient or imperfect, as indicated by deficient number or non-typical characters of the cicatrices. But even when the original vaccination has been in every way satisfactory, it is necessary to re-vaccinate after puberty. Some recommend that the operation should be performed every seven years, but this appears quite



unnecessary, and one efficient re-vaccination may be considered as affording absolute and perfect protection. The same precautions and care are required as in the case of primary vaccination. Fainting has been noticed as a frequent occurrence during the performance of re-vaccination.

*Results of Re-vaccination.*—In some cases no effect can be produced by re-vaccination, especially in children. On the other hand, now and then a perfectly typical course is observed, chiefly in adults. Usually the course and characters of the eruption are much modified. It appears earlier, and reaches its height by the fifth or sixth day; being either papular, or in the form of an acuminated vesicle, with an indurated, diffused, and irregular areola. A small scab forms by the eighth day, which soon falls off. There is generally much local irritation; and constitutional symptoms are usually conspicuously more marked than in primary vaccination. Erysipelas is liable to occur, and occasionally fatal septicæmia or pyæmia sets in. I have known a patient sink rapidly after re-vaccination, without any obvious cause.

*REMOTE EFFECTS OF VACCINATION.*—There cannot be the slightest doubt in the mind of any unprejudiced observer, with regard to the powerfully protective influence exercised by vaccination against the ravages of small-pox. In a large proportion of cases, if vaccination has been thoroughly and efficiently performed, and especially after re-vaccination, absolute and complete protection against this malady is established. But even when it is not entirely prevented, the disease manifests itself only in a slight and modified form, is scarcely attended with any danger, and does not leave behind the hideous disfigurement which it often produces if allowed to proceed unchecked. Epidemics of small-pox have been much less frequent and severe since the introduction of the practice of vaccination, and this result has been in direct proportion to the efficiency of the measures which have been put in force to ensure its general and successful performance. This has been observed in every part of the world, and amongst all races. It is very important to bear in mind, that in proportion to the number and typical characters of the vaccination-marks will this immunity from small-pox be the more certain.

It has been stated that certain affections are transmitted to children by vaccination, especially cutaneous diseases, scrofula, and syphilis. There is no reliable evidence that this happens to any serious extent, but cases have been brought forward by Mr. Hutchinson and others which undoubtedly prove that such a result does occasionally occur, and in order to guard against the mere possibility of this untoward event, it is necessary to pay strict attention to the precautions already alluded to, and especially to see that the vaccine-lymph employed is taken from a perfectly healthy child.

*TREATMENT.*—All that is generally required after vaccination is to protect the arm from irritation, and to prevent the vesicles from being scratched. Mr. Purvis recommends that the arm be left entirely out of the sleeve. If there is much subsequent inflammation, wet lint, lead lotion, or cream may be applied; or the part may be covered with finely-powdered starch.

During the pyrexial condition it is well to keep the child indoors; and to give some mild *aperient*, such as a teaspoonful of castor-oil. Unusual complications, such as erysipelas, may call for special treatment, and these are particularly to be borne in mind when re-vaccination has been performed.

## CHAPTER XIII.

## VARICELLA—CHICKEN-POX.

**ÆTIOLOGY.**—Varicella has been supposed to be merely a modified form of variola, but the evidence is conclusive that they are perfectly distinct diseases. It is decidedly an infectious complaint, having a *specific contagium*, and it may be transmitted either with or without direct contact. It is doubtful whether it can be inoculated, but probably not. Occasionally this affection assumes an epidemic form. A second attack is never met with. Varicella is only observed in children as a rule, but now and then it affects young or adult females.

**SYMPTOMS.**—1. **Incubation-stage.**—The ordinary duration of the *period of incubation* in varicella is twelve days, but it may range from ten to sixteen days. There are no symptoms.

2. **Invasion-stage.**—Frequently this stage is absent, the first symptoms being simultaneous with the appearance of the eruption. In other cases this is preceded for twenty-four to thirty-six hours by slight pyrexia, with headache, and sometimes a slight cough.

3. **Eruption-stage.**—The eruption of varicella is rarely delayed beyond twenty-four to thirty-six hours at the latest, there being at first but a few spots, but fresh crops appear during four or five nights, often in considerable numbers, and they may continue to come out for ten or twelve days. The spots are quite distinct as a rule, but occasionally a few become confluent. They are first seen usually on the body, especially about and between the shoulders, and over the chest. Afterwards they extend to the limbs, while the scalp is often much affected, but the face is generally only slightly involved, though the eruption is sometimes abundant here. Some authors have described the eruption as being vesicular from the first. In most cases, however, it begins as bright red spots, slightly papular, not at all hard, and disappearing on pressure. Within a few hours these become vesicular, a clear fluid collecting under the epidermis. The vesicles are of good size, round or oval, ill-defined, and neither umbilicated nor divided into spaces, so that when punctured they collapse completely. There is no inflammatory areola. The appearance is described as being in some instances, “as if the patient had been subjected to a shower of scalding water.” In about twenty-four hours the contents of the vesicles become slightly and uniformly opalescent, and a faint red areola appears. Each vesicle either ruptures or dries up from the third to the fifth day, a small scab extending rapidly from the centre towards the margin. This is generally thin and crumbly, coming away in particles, but occasionally a thick coherent crust is formed. It separates in about four or five days usually. As the skin is not deeply involved or destroyed, only a slight redness is left, which soon disappears, and there is no pitting as a rule; in exceptional instances, however, distinct pits are left, which are round or elliptical, smooth, and shining. Owing to the development of successive crops, vesicles are seen side by side in different stages of their progress. The only subjective sensation which attends the eruption of

chicken-pox is that of itchiness, which may be considerable. A faint peculiar odour is said to be given off, but this is certainly not constant.

The general symptoms are very slight in most cases, there being only a little feverishness, if any. Sometimes there are rather severe exacerbations of fever during the night. Catarrh is frequently present, and may prove dangerous if the bronchi should become extensively implicated. In some cases the patient remains in an unsatisfactory state of health for some time after an attack of varicella.

A number of *varieties* of varicella have been described, but they are of no importance, and most of the supposed varieties of this disease are really modifications of small-pox.

PROGNOSIS.—Death never occurs as the result of varicella, and therefore the prognosis is highly satisfactory.

TREATMENT.—Nothing is required in varicella but to keep the patient quiet; to give a simple and mild diet; and to see that the bowels are freely opened. Children must be prevented from scratching themselves. Catarrh must be attended to if present: and should there be much fever, a saline mixture is useful. Quinine may be administered during convalescence.

---

#### CHAPTER XIV.

#### ERYSIPELAS—THE ROSE—ST. ANTHONY'S FIRE.

ÆTIOLOGY.—Only that form of erysipelas which occurs as an *acute idiopathic* disease is considered in this work, the *traumatic* varieties coming more appropriately within the scope of surgical treatises. Undoubtedly one form of this complaint may be regarded as an *acute specific disease*, and though its contagious nature is denied by some authorities, there is ample evidence to prove that it is an infectious complaint, and that it is capable of being transmitted from one individual to another, especially when a number of persons are collected together, as in hospitals. I have met with several instances corroborative of this statement. Sometimes also the affection assumes an epidemic form. The poison is given off into the atmosphere, and may likewise be conveyed by fomites, as well as by direct contact or inoculation. There is reason to believe further, that erysipelas may by infection originate a group of certain allied diseases, such as puerperal fever or hospital gangrene, and *vice versâ*. What the nature of the contagium is cannot be positively affirmed. It has been supposed to consist in micrococci, which have been found in the inflamed tissues, plugging the lymphatic vessels and spaces, and colonizing the deep tissues and viscera. There seems to be no doubt that micrococci are concerned in conveying infection in surgical cases, and in the puerperal and other forms of erysipelatosus inflammation. Erysipelas has been conveyed by inoculation of the cultivated fungus.

Though capable of being conveyed by infection, on the other hand many cases of erysipelas are met with in which the origin of the attack certainly cannot be thus explained. Indeed, not unfrequently no obvious



*exciting cause* can be discovered, while in some instances the complaint is attributed to local exposure; undue cold or heat; general exposure to cold and wet; very slight injury; irritation of bad teeth; errors in diet, especially the consumption of shell-fish and such articles; and violent mental emotion. When it arises under these circumstances, erysipelas may be regarded as primarily a local disease. Those, however, who regard it as invariably due to organisms, would affirm that the supposed causes mentioned only bring about conditions aiding the action of these organisms upon the system.

Among the most important *predisposing causes* of erysipelas are:—1. Age, the disease being most common in newly-born infants, and in persons from twenty to forty years old. 2. The female sex, especially during menstruation. 3. Individual and family predisposition. 4. The occurrence of previous attacks, erysipelas differing from most other acute specific fevers in this respect. 5. Certain conditions of the system, namely, plethora; the condition induced by intemperance or debilitating diseases, as well as that associated with gout or renal disease; and any low febrile state. 6. Certain local conditions, such as various forms of injury; or the presence of dropsy in a part. 7. A warm season.

**ANATOMICAL CHARACTERS.**—Erysipelas is characterized by a diffuse inflammation of the skin, the subcutaneous cellular tissue being also generally involved, and sometimes the deeper structures. At first there is hyperæmic redness, varying in tint, followed by vesication of the skin, and by serous infiltration of the subjacent areolar tissue, which causes more or less swelling. In severe cases pus may form under the cuticle, in the subcutaneous tissue, or in the deep tissues. There is no tendency to the formation of lymph, so that the inflammatory process is not limited by adhesions; while any pus which is formed is liable to be of a low type. Occasionally erysipelas terminates in ulceration or gangrene, especially when the tissues affected are in a low state of vitality, as, for instance, when they are the seat of dropsy. The neighbouring lymphatic glands and vessels are always affected; while the veins leading from the erysipelatous part are also inflamed in many cases, and may contain pus. Bacteria have been described as being present in abundance in the tissues towards which erysipelatous inflammation is advancing.

In fatal cases the blood is often dark and liquid, and does not coagulate firmly. The various organs are congested, especially the lungs, or they are sometimes the seat of inflammation. The small vessels of the lungs and head frequently contain pus. Minute emboli, composed of white corpuscles or of albuminoid particles, have been found by Bastian and others in the small vessels of the grey matter of the brain.

The mucous and serous tissues may be the seat of erysipelatous inflammation, as well as the cutaneous structures.

**SYMPTOMS.**—After an **incubation-period**, which is said to range generally between ten and fourteen days, but which may extend to three weeks, an attack of erysipelas is in most cases, but not invariably, ushered in by **premonitory symptoms** of a general character. A feeling of illness is experienced, with general uneasiness or muscular pains, disturbance of the digestive organs, sore-throat, headache, restlessness, and other signs of nervous disorder, with a certain degree of pyrexia. Some shivering or chilliness may be felt at the outset, but usually no marked rigors are experienced until the local inflammation is about being, or has been, developed. These symptoms may last from a few hours to four or five days before the characteristic signs of

erysipelas appear, but generally the latter are evident within two or three days. Epistaxis occasionally occurs at the period of invasion.

The **local symptoms** of erysipelas are as follows:—At first there is a feeling of heat, irritation, and tightness in the affected part; the skin is tender to the touch; and a stinging or smarting sensation is felt. Soon the surface becomes red, swollen, firm, tense, and shining; at the same time all the painful sensations being aggravated; and there being increased local heat, as determined by the thermometer. Sometimes the swelling precedes the redness.

Erysipelatous inflammation starts from one spot, and generally extends chiefly in a particular direction, but it sometimes spreads in all directions equally. There is a well-marked boundary-line between the advancing inflammation and the healthy skin, as shown by the difference in colour, and by the abrupt termination of the swelling, but only a gradual transition is observed at that border where the process is subsiding.

The hue of the redness varies, but it tends to become darker as the case progresses. The swelling is much greater where abundant loose cellular tissue exists, and is then often irregular in form and unequal in consistence, while the surface pits on pressure. In structures which are tense, unyielding, and closely attached, such as those of the scalp, the sensations are far more painful than in lax tissues.

In slight cases the inflammation subsides, being followed by desquamation of the cuticle; far more commonly, however, cutaneous vesicles of various sizes form, which contain a yellowish serum, and in serious cases large irregular bullæ or bladders are raised upon the surface. These burst and discharge their contents, often leaving crusts, which on separating may disclose superficial ulceration. The cuticle always peels off extensively. Occasionally signs of more or less suppuration, ulceration, or moist gangrene are observed; and these processes may lead to great destruction of tissues.

The seat and extent of erysipelatous inflammation vary in different cases. Idiopathic erysipelas is most common about the head and face, and it generally begins about the nose, ear, angle of the mouth, lower eyelid, or cheek. Dr. Reynolds has observed that it usually starts at the point where the skin is undergoing transition into a mucous membrane. The erysipelas tends to spread rapidly, so that the whole face, scalp, and neck may become speedily affected, and great swelling is produced, the features being obliterated, the eyelids closed, and the nostrils blocked up, while deafness is often complained of. Not unfrequently abscesses form, especially in the cheeks or eyelids. The inflammation is apt to extend to the mouth and fauces, and may even reach the larynx. There is also a danger of the supervention of meningitis.

In some instances the limbs are affected, especially the legs, and now and then the trunk. I have met with two cases of erysipelas involving the whole of both legs, as a complication of acute rheumatism. Some local irritation may determine the locality of an erysipelatous inflammation.

The time taken by erysipelatous inflammation in running its course varies, but the redness and swelling generally attain their height on the second or third day. Different parts of the surface are seen in different stages of advancement. After it has apparently stopped, the inflammation may again spread, and *relapses* are by no means uncommon. In some cases the complaint is *erratic* or wandering in its progress; or it may assume a *metastatic* character.



Usually the absorbent glands and vessels in the neighbourhood of the affected structures show signs of irritation, being enlarged as well as painful and tender, sometimes very much so, and they may be involved first. Suppuration of the glands takes place in exceptional cases.

The **general symptoms** in erysipelas usually increase with the onset of the local inflammation. Ordinarily they merely indicate more or less pyrexia. The pulse rises to 100 or 120, and is full and strong. The temperature ascends rapidly at the outset, and may attain a height of  $104^{\circ}$  or  $105^{\circ}$  on the first evening of the eruption. Usually the maximum temperature is reached on the third day, but it increases so long as the inflammation advances, and may attain to  $106^{\circ}$  or  $108^{\circ}$ . As a rule there are distinct evening exacerbations, but the evening temperature may be  $2^{\circ}$ ,  $4^{\circ}$ , or even  $5^{\circ}$  lower than that of the morning (Reynolds). Defervescence sets in in favourable cases about the fifth or sixth day of the eruption, and the temperature rapidly falls, becoming normal in from twelve to thirty-six hours. It may, however, remain high for a much longer time, and defervescence is then less critical. These observations apply chiefly to *facial erysipelas*, for great deviations as regards temperature are met with when the disease attacks other parts. Any relapse or extension of inflammation is indicated by a rise in temperature, which may be noticed before any external signs are observed. Complications will also influence the temperature. The urine is febrile, while urea is increased, and chlorides are diminished. Albuminuria is a frequent phenomenon in cases of erysipelas.

In *facial erysipelas* there is considerable restlessness, with, in many instances, mental wandering or actual delirium, especially at night, quite apart from any cerebral complication. In this form the tongue always tends to become dry and brownish; and in all cases of a low type it assumes distinctly adynamic characters, with sordes on the lips and teeth, the pulse also becoming very rapid and feeble, and other typhoid symptoms setting in. Tympanites and hiccup may be prominent symptoms in such cases. This course of events is likewise apt to occur in very feeble or intemperate persons, and in aged subjects.

**COMPLICATIONS.**—The complications to be chiefly feared in connection with erysipelas are cerebral or spinal meningitis; bronchitis; acute intestinal catarrh; and renal congestion or inflammation. As already mentioned, erysipelas may spread to the throat or larynx, or it may involve serous membranes.

**VARIETIES.**—Several varieties of erysipelas are described, founded on the intensity, mode of progress, appearances, and terminations of the local changes. The chief of these are:—1. **Simple** or **cutaneous**. 2. **Miliary**. 3. **Phlyctenous**. The last two are named from the size of the vesicles or blebs. 4. **Oedematous**, where there is much oedema. 5. **Phlegmonous** or **Cellulo cutaneous**, in which the deep tissues are extensively involved, and tend to suppurate. 6. **Gangrenous**. 7. **Erratic** or **Migratory**. 8. **Metastatic**. Varieties are also named according to the part affected, for example, *facial*, *scrotal*, *crural*, and *abdominal*.

The *erratic* form usually presents less hyperæmia and swelling than is ordinarily observed; while the pyrexia is not so severe, considerable and rapid changes in temperature being also observed. This variety tends to run a protracted course, and occurs chiefly in the old, or in those suffering from gout, rheumatism, or kidney disease.



**PROGNOSIS.**—Erysipelas is always a serious malady, and a cautious prognosis should be given in all cases, but especially when it attacks the scalp or face. The principal circumstances which increase the danger of any individual case are as follows:—1. The patient being either very young or of advanced age. 2. A low condition of the system, especially that due to intemperance. 3. The presence of organic disease, particularly renal disease with dropsy. 4. The complaint being epidemic, much depending on the type of the epidemic. 5. Any tendency to typhoid symptoms; or signs of blood-poisoning. 6. Severe cerebral symptoms, particularly if they point to meningitis. 7. Extension of the inflammation to the throat or larynx. 8. A dark colour of the eruption, or the appearance of livid vesicles. 9. Any disposition to involve the deep tissues extensively, or to end in suppuration or gangrene. 10. The sudden disappearance of the external inflammation, with simultaneous occurrence of symptoms indicating that some internal part is attacked.

**TREATMENT.**—1. **General management.**—Unquestionably lowering measures are to be avoided in most cases of erysipelas, and a supporting treatment is that which gives the best results. A nutritious *diet* is necessary from the first, with cooling drinks, and in many instances *alcoholic stimulants* are called for at an early period, not uncommonly considerable quantities being required during the progress of the disease. The patient should, if possible, be isolated, and placed in a comfortable, well-ventilated, but not draughty apartment; and every attention must be paid to hygienic measures.

2. **Therapeutic treatment.**—The bowels should be kept well opened by *saline aperients* in all cases of erysipelas. Ringer highly recommends the administration of tincture of aconite or belladonna, for the purpose of checking the complaint. The most reliable medicinal remedy, as a rule, in my experience, is tincture of perchloride of iron— $\text{m xx-xl}$  every three or four hours. Some practitioners advocate the use of *antiseptics* internally. In dynamic cases quinine or ammonia and bark may be given, along with alcoholic stimulants. It is often necessary to administer opium, chloral, bromide of potassium, or some such agent, at night or more frequently, for the purpose of procuring sleep and relieving pain.

3. **Local treatment.**—Ordinarily it will be found the best plan to cover the erysipelatous part with cotton-wool, having previously powdered it over with flour, or with a mixture of starch and oxide of zinc. When the face is affected, a kind of mask may be made, with apertures corresponding to the mouth, nostrils, and eyes. Among the numerous *local applications* recommended for erysipelas the most important are collodion or flexible collodion; nitrate of silver, either in the form of the solid stick or in solution; extract or liniment of belladonna, or equal parts of extract of belladonna and glycerine; and solution or glycerine of carbolic acid. In some cases great pain is experienced, and then warm fomentations containing opium or belladonna are serviceable, the surface being afterwards dried and covered with cotton-wool. Nitrate of silver has been much used with the object of checking the progress of erysipelas, the stick being rubbed into the skin a little beyond the advancing margin of the inflammation. This measure appears to succeed sometimes. Suppuration calls for free incision; and in the phlegmonous variety scarification is of great value.

4. **Symptomatic treatment.**—Various symptoms may require attention in erysipelas, as well as complications, especially meningitis, and extension of the inflammation to the throat or larynx. The treatment

of these conditions will be considered in their respective chapters, but it may be here mentioned that if there is much œdema about the glottis, it may be requisite to scarify the mucous membrane covering this part, or even to perform laryngotomy or tracheotomy.

5. **Prevention.**—In order to prevent the spread of erysipelas, it is imperative upon those who are attending cases of this complaint, especially medical men and nurses, to exercise every precaution against conveying the disease to others, particularly if they have to come into contact with parturient women, or with persons suffering from wounds or ulcers. Should there be any condition present in any case in which erysipelas is liable to arise, such as dropsy, every precaution must be taken to prevent its occurrence.

---

## CHAPTER XV.

### DIPHTHERIA.

**ÆTIOLOGY.**—Diphtheria is an independent acute specific disease, being ordinarily produced by a *specific poison*, and it is highly infectious, not unfrequently assuming a severe epidemic form. The contagium is believed by some authorities to be associated only or chiefly with the peculiar deposit which forms on the throat and other parts, and it has been asserted that this material must be brought into contact with a mucous membrane, or with an abraded cutaneous surface, before the complaint can be transmitted; there is strong reason to believe, however, that the contagium is also given off in the breath, and is likewise probably contained in the various excretions. Some think that the diphtheritic poison always exerts its first effect upon the throat or other surface with which it comes into contact, and that the general infection only follows the local lesion as a secondary result. Probably, however, the general system is first affected usually. The disease may spread to any of the inmates of a house, but there is always much more danger of infection in the case of those who are brought into contact with a patient suffering from diphtheria, and who therefore inhale the breath, or are liable to have the morbid products coughed out upon them. Infection lasts for an uncertain time after convalescence. The affection is more liable to be propagated if there is much discharge, as from the nose, especially if cleanliness is not attended to. The poison clings tenaciously to houses and rooms for a considerable period, and may be conveyed by fomites. It seems capable of being disseminated some distance, and this has been attributed to the influence of winds. It is a matter of doubt whether diphtheria can be originated by inoculation with the deposit. The same individual may be attacked more than once.

Of the exact nature of the contagium of diphtheria there is no positive knowledge. It has been assumed to consist in certain vegetable organisms, which have been observed in the diphtheritic deposit on microscopic examination; or in micrococci or bacteria, which infiltrate the tissues, and invade the lymphatic system. A specific

bacillus has also been described. Dr. Jacobi of New York strongly maintains that the contagium of diphtheria is of a chemical nature. It has been affirmed that there is a *sporadic* form of this disease, which is not contagious, but which is produced by unfavourable hygienic conditions, especially by drainage emanations. Cases have come under my notice which certainly appeared to bear out this assertion.

The *predisposing causes* of diphtheria are the period of childhood; individual and family susceptibility; bodily fatigue or exhaustion; and nervous excitability. With regard to the influence of hygienic conditions there is some uncertainty. Diphtheria frequently occurs under the most satisfactory sanitary surroundings, but some observers think that the complaint is predisposed to by bad feeding, and unfavourable hygienic conditions; as well as by general debility, or a cachectic state. It has also been stated to be frequent after scarlatina, measles, and whooping-cough, or even after a mere sore-throat. Hot and dry climates and seasons appear to favour the development of the poison-germs of diphtheria.

**ANATOMICAL CHARACTERS.**—A peculiar inflammation of the fauces, attended with the formation of patches of exudation, constitutes the ordinary local manifestation of diphtheria. At first there is redness, which may begin in any part of the throat, being accompanied with swelling and increased secretion of viscid mucus. The redness spreads over the entire mucous surface, and then the exudation makes its appearance. The deposit may commence at any spot, such as on one of the tonsils, on the soft palate, or at the back of the fauces, and may start from one or several points, at first only small specks being observed, which, however, speedily extend and coalesce so as often to form extensive patches, or even to cover uniformly the entire surface. The patches have a variable thickness, and they become thicker by successive layers being formed underneath. The characters of the deposit vary much. The colour is usually grey, white, or slightly yellowish; but it may be brownish or blackish. The consistence ranges from “cream to wash-leather.” The material resembles in some cases wet parchment or damp and dirty wash-leather. On removing it, which is effected with more or less difficulty, a raw bleeding surface is left, or sometimes a distinct ulcer, which is again speedily covered by fresh deposit, the exudation generally involving the substance of, as well as lying upon, the mucous membrane, thus causing its destruction. Usually the epithelium is destroyed, but occasionally traces of its cells are found under diphtheritic deposits. The under surface of a patch may present little spots of blood. If the exudation separates of itself, it is either not renewed at all, or only in thinner films. Occasionally considerable ulceration or sloughing of the soft palate, uvula, or tonsils is set up; or abscesses may form.

The deposit may spread from the throat to the mouth, lips, nose, Eustachian tubes and ears, conjunctivæ, larynx, trachea, or bronchi, even to their finest ramifications; rarely to the œsophagus, stomach, intestines, and gall-bladder. In exceptional instances it starts primarily in certain of these parts. It has also been observed over the vulva or vagina, the anus and rectum, the glans penis and prepuce, the external ear, and other parts. Any raw cutaneous surface is liable to become implicated.

The microscope usually reveals that the diphtheritic material consists chiefly of epithelial and granular cells, with molecular granules of fat and protein. In a specimen examined by Dr. Greenfield, the cells in the



superficial part were found to be chiefly leucocytes, and altered or decomposed catarrhal cells; those in the deeper layers consisted of epithelial cells infiltrated with a highly refractile substance, variously swollen, and with their outlines obscured. He considers that these appearances support Wagner's view of the formation of diphtheritic material by the transformation of epithelial cells. Sometimes fibres and disintegrated tissues are visible. Vegetable growths have also been observed, but they are not always present, and similar organisms are met with in other morbid products. Chemically the substance chiefly resembles fibrin.

The lymphatic glands in the neighbourhood are enlarged and inflamed, especially those near the angles of the jaw. Should there be much ulceration or gangrene of the fauces, general swelling of the neck ensues, owing to infiltration of the tissues with serum or lymph.

In fatal cases of diphtheria the various organs are found to be much congested. The spleen and the absorbent glands generally are enlarged. The lungs are in many cases the seat of acute insufflation, collapse, lobar or lobular pneumonia, or sometimes of apoplexy. The kidneys may show signs of acute parenchymatous inflammation. Fibrinous coagula are frequently observed within the cavities of the heart, and extending thence into the great vessels; and the heart-fibres may have undergone acute degeneration.

**SYMPTOMS.**—Diphtheria is essentially a *general* disease, which is accompanied with *local* lesions. Hence it is usually ushered in by *general* or *constitutional* symptoms; followed by *local* symptoms, referable to the throat or to other parts which happen to become involved. Along with the development of local symptoms, the general disturbance is increased, and may become extremely severe; while the complaint is liable to be followed by a peculiar nervous disorder. In some cases the local symptoms are first observed.

The *period of incubation* in diphtheria ranges usually from two to four days (Squire). It may be limited to thirty hours, or extend to eight days, or perhaps even to a longer period than this. The *invasion* is generally gradual, a feeling of illness, languor, more or less weakness, and depression being often experienced; accompanied with chilliness, anorexia, nausea, diarrhoea, headache, drowsiness, and a certain degree of pyrexia. At the same time some stiffness of the neck, tenderness about the angles of the jaw, or slight sore-throat may be complained of.

**Clinical course.**—The symptoms of the developed disease vary considerably in character and severity, and it is important to notice that the *gravity of the constitutional disorder bears no necessary relation to the throat-symptoms*; while in apparently slight cases grave symptoms may at any time supervene.

In the large majority of instances the *local* symptoms of diphtheria are mainly associated with the throat. The patient complains of more or less soreness or actual pain in this part, especially on swallowing, and this act may become difficult or even impossible in bad cases. There is frequently a constant desire to hawk for the purpose of clearing the throat. On examination the structures of the fauces are seen to be red and swollen, and covered to a variable degree and extent with the diphtheritic deposit. Sometimes the tonsils and uvula are so much enlarged that they seem to block up the passage, and an examination can only be accomplished with difficulty. In bad cases more or less extensive ulceration or sloughing may be observed. The diphtheritic material

is not uncommonly coughed up in fragments of so-called *false membrane*; and offensive matters are likely to be expectorated when the structures ulcerate or become gangrenous. The glands about the angle of the jaw are always enlarged to some degree, and feel tender; in severe cases they become much swollen, and the structures of the neck generally may be considerably tumefied. The implication of these glands is part of the disease, and not merely secondary to the throat-condition; it may be one of the earliest signs. In some instances the nasal cavities are implicated, and symptoms arising therefrom may be the first noticed, a discharge, which is often sanious and offensive, escaping through the nostrils or through the posterior nares. When the larynx is involved, this event is indicated by hoarseness, or complete loss of voice; cough, of a hoarse and croupy character; and obstructive dyspnoea, which often becomes very urgent, the breathing being noisy and stridulous, and subject to paroxysmal exacerbations. If the deposit extends down the respiratory passages to the bronchi, the breathing becomes still more embarrassed. In connection with the morbid conditions of the parts just considered, the breath has often an offensive odour, and may become extremely foetid. Should the diphtheritic deposit form in other parts, such as the stomach or intestines, corresponding local symptoms might be developed; and if it occurs over external parts, it would be at once visible on examination.

The *general* symptoms in diphtheria are usually at first of a febrile character, but their intensity is subject to much variety, and even in severe cases, as judged by the local condition, the temperature need not be high. As a rule, indeed, the temperature in this disease is not much elevated, and it has no characteristic course. The patient not uncommonly experiences a marked sense of illness, weakness, and depression, which may be quite out of proportion to the apparent gravity of the disease; and in some instances I have observed a remarkable foreboding of death on the part of the patient, when there did not appear to be any reason for anticipating such an event. In bad cases of diphtheria the general symptoms assume an asthenic or typhoid character, either from the first, or in the course of the attack, the patient being greatly prostrated, and presenting the usual phenomena characteristic of the typhoid condition. When the respiratory passages are involved the symptoms indicate more or less interference with the aëration of the blood, and may culminate in those of actual asphyxia. The urine generally presents morbid characters in diphtheria, and in addition to being febrile, it frequently contains albumen, as well as in some cases blood and casts.

**VARIETIES.**—Having thus pointed out the general features of the clinical history of diphtheria, it will now be expedient to describe briefly the principal varieties met with, mainly founded on the practical and useful classification of cases laid down by Sir William Jenner.

**I. Mild Form.**—Here there are slight throat-symptoms, and on examination signs of inflammation, with more or less diphtheritic deposit, are visible. The glands about the angles of the jaw are a little swollen and tender. Pyrexia is usually mild and of short duration, but the temperature may rise considerably. The urine is not albuminous. Occasionally extensive deposit on the throat is observed, while the general symptoms are scarcely perceptible. Recovery is rapid and complete; and no sequelæ follow the attack. It must be remembered, however, that in cases which at first seem to be of a very mild character, serious symptoms may subsequently arise.

**2. Inflammatory Form.**—After premonitory symptoms, high pyrexia sets in, the patient at the same time feeling very ill and weak. The pulse soon tends to become feeble and wanting in tone. Throat-symptoms are prominent, while examination reveals signs of marked inflammation, which may be accompanied with much enlargement of the tonsils and uvula, a considerable diphtheritic deposit forming in from twelve to forty-eight hours, which possesses a fair degree of consistence and toughness. This may be coughed up in pieces of *false membrane*, of variable size. Subsequently the throat may ulcerate or slough more or less extensively. The disease is also liable to spread to the larynx or further down the respiratory passages, thus giving rise to dangerous laryngeal symptoms, and causing serious interference with respiration. The glands of the neck are much enlarged. The urine is febrile, but also frequently contains much albumen, as well as some granular casts.

**3. Nasal Form.**—In this variety there is at first a sanious discharge from the nose, accompanied with low fever. Soon the throat is seen to be red and swollen, and fluid escapes through the posterior nares, while the glands about the angles of the jaw swell considerably. The discharge may be very fetid. Afterwards the deposit may form on the pharynx or larynx, the latter being sometimes unexpectedly attacked; or the symptoms may subside, and recovery take place.

**4. Laryngeal and Tracheal Form.**—This is characterized by the exudation starting in the larynx and trachea, but it may subsequently spread to the pharynx. Laryngeal symptoms are therefore prominent from the first. The disease may also extend downwards along the air-passages to the lungs. This variety is now generally regarded as being identical with so-called *true croup* or *croupous laryngitis*, but this question will be considered under the latter disease. Here may be also mentioned certain cases which are grouped together by Sir William Jenner under the term *insidious*, in which, without any particular general disturbance, and only slight sore-throat, laryngeal symptoms suddenly set in with severity, and this course of events may terminate in speedy suffocation.

**5. Asthenic Form.**—In this class of cases the general symptoms assume a low type, either from the outset, or during the progress of the ordinary symptoms of diphtheria. The sense of illness and prostration becomes very great; the complexion is dirty-looking and opaque, and the skin generally may assume a dirty-yellowish tint, having also a peculiar feverish pungency, though the temperature is not remarkably high. The pulse tends to be very frequent, small, weak, and irregular; and the heart's action is greatly enfeebled. The tongue becomes dry and brown, while sordes form on the lips and teeth. Ultimately the ordinary typhoid symptoms set in, with delirium and other low nervous phenomena, and at last the patient sinks.

The symptoms just described may or may not be associated with much diphtheritic deposit over the throat or larynx, but the material is frequently of a soft, pulpy kind, and hence the local symptoms are often not proportionately severe. They are not uncommon in the nasal variety of diphtheria. It is in these cases that extensive ulceration and sloughing are chiefly met with, the asthenic symptoms being then due to septicæmia, resulting from absorption of the decomposing matters into the blood. Under such circumstances the breath becomes very fetid; and there is great swelling about the neck.



6. **Anomalous Form.**—A group of cases may be made to include those in which the diphtheritic deposit first appears in some unusual region, such as about the anus, on the vulva or vagina, over the glans penis or prepuce, in the external auditory meatus, or on cutaneous excoriations or ulcerations. The throat often becomes secondarily involved. The membranous deposit is white, buff, grey, or black, and more or less adherent; it is surrounded by redness, usually vivid and marked. On the skin its extension is preceded by the formation of vesicles, and subsequent excoriation.

**COMPLICATIONS AND SEQUELÆ.**—*Albuminuria* is of frequent occurrence in the course of diphtheria. This condition always demands special attention, and should be looked for daily in all cases from the first. Dr. J. Abercrombie found it in 24 out of 91 cases, but the proportion varies in different epidemics. It has been attributed to various causes, such as the direct action of the diphtheritic poison, leading to waste of tissues and altered blood, or rapid elimination of this poison; the consumption of more food than can be assimilated; interference with respiration; and high fever. These causes may act singly, or in different combinations, but albuminuria does not occur only in cases where there is a high temperature. The most important cause, however of this symptom is acute parenchymatous nephritis, when it may be accompanied with the presence of blood-corpuscles, blood-casts, and epithelial casts in the urine. Dr. Abercrombie never observed this fluid smoky; neither was there any anasarca, nor any symptoms of uræmia. In all the fatal cases there were the ordinary changes of acute parenchymatous nephritis; and in one case the capillary vessels of the cortex of the kidneys were observed by Dr. Samuel West and himself to be crowded with minute shining bodies, which they took for micrococci. Other observers, however, affirm that in cases which have ended fatally within the first few days, with abundant albuminuria, the kidneys have only presented more or less congestion. Albuminuria in diphtheria is an early phenomenon, and nephritis comes on at a much earlier period in this complaint than in scarlatina. Thus albumen may be found in the urine within twenty-four hours of the onset of the disease, and usually appears about the third or fourth day, being exceptional after ten or twelve days. As a rule it does not continue long, being in some instances a mere transitory phenomenon; in none of Dr. Abercrombie's cases did it last more than a fortnight; sometimes, however, it goes on for several weeks, or may even be permanent. The amount of albumen discharged varies considerably, but it may be very large; while it is sometimes intermittent, or more frequently varies in quantity at different periods within the twenty-four hours. Occasionally the urine is much diminished or even suppressed in diphtheria.

Hæmorrhage from the nose, throat, air-passages, and other parts is not uncommon in bad cases of diphtheria, and there may be purpuric spots on the skin. Cutaneous rashes are occasionally observed, transient in duration, and either erythematous, erysipelatous, roseolar, urticarial, or even vesicular in appearance. Pulmonary complications are not infrequent, especially if the respiratory passages are involved, namely:—acute insufflation of the lungs, the vesicles sometimes giving way; pulmonary collapse; lobar or lobular pneumonia; and pulmonary apoplexy. It must be remembered also that the diphtheritic exudation may extend even to the smallest bronchi.

Diphtheria is liable to be followed by important *sequelæ*. Thus, in

some cases the progress towards convalescence is very slow, and a state of marked debility and anæmia remains for some time. Slight albuminuria may also continue for a considerable period; or permanent renal disease may be set up. The most remarkable series of sequelæ, however, are those connected with the nervous system. It will be more satisfactory to discuss these sequelæ in relation with the other diseases of this system, and in the meantime it must suffice to state that they may follow the mildest attack of diphtheria; that they usually supervene after an interval of apparent convalescence; and that the phenomena are those of more or less extensive paralysis, motor and sensory, which usually commences in, and may be limited to, the throat, but generally tends to be more or less progressive, involving ultimately in extreme cases the whole body, and sometimes proving fatal, owing to implication of the respiratory muscles or of the heart, especially in children. Most cases recover, however, but the duration of the symptoms is very variable. Abnormal sensations, such as hyperæsthesia or marked tenderness in various parts, or intense neuralgia, may also follow diphtheria.

**DURATION AND TERMINATIONS.**—The *duration* of diphtheria ranges from two to fourteen days, but complications and sequelæ may prolong its course considerably. *Relapses* also are not very uncommon. Death is a very frequent event, especially in some epidemics. The chief causes of death are:—1. Suffocation, owing to the air-passages being implicated, which is most frequent in children, and generally happens within the first week. 2. Gradual asthenia, most common in persons beyond the age of puberty, death usually occurring after the first week. 3. Septicæmia. 4. Renal disease. 5. Pulmonary complications. 6. Secondary nervous disturbance. This is very fatal in children, but death does not occur from this cause after a lapse of two months. Occasionally patients attacked with diphtheria die within a few hours, apparently killed by the virulence of the poison. Sudden death has also happened in several instances, which has been attributed to syncope; or to the formation of a fibrinous coagulum within the heart, or in one of the great vessels.

**PROGNOSIS.**—Always grave, the prognosis of diphtheria is much worse in children than adults, and especially in infants. The chief signs of danger are:—Implication of the air-passages, with consequent interference with respiration, as well as the development of pulmonary complications; extensive ulceration or sloughing of the throat; great discharge from the nares; epistaxis; repeated vomiting or diarrhœa; very rapid and feeble cardiac action and pulse, or a very infrequent pulse; typhoid symptoms, especially if accompanied with delirium; suppression of urine; signs of uræmia; the presence of abundant albumen, blood, or casts in the urine, particularly if accompanied with laryngeal symptoms; and a sudden rise in temperature. Even mild cases may prove fatal from asthenia; or the nervous sequelæ may set in and cause death. The prognosis is more unfavourable in certain constitutions, and in particular epidemics. The mortality appears to be greater during cold and damp seasons.

**TREATMENT.**—The management of cases of diphtheria must depend very much on their type and intensity, but even the mildest case needs to be carefully watched in its progress, so as to be prepared for any untoward course of events. It cannot be too strongly enforced that there is no specific remedy for this disease, and that the measures to be adopted must be determined by the circumstances of each individual

case. It may be further affirmed that lowering measures are never admissible, a more or less supporting treatment being always indicated for diphtheria.

1. **General management.**—Any patient suffering from diphtheria must remain in bed, well protected from draughts, the room being kept at a good, uniform temperature, and all hygienic conditions being duly observed, especially as regards *cleanliness* and *proper ventilation*. In a severe case it is important that the air of the room should be maintained at a temperature of 65° to 68°, and kept moist with steam, either from a boiling kettle having a long spout, or by boiling water in an open vessel over a spirit lamp. Children should have a tent made over their cribs by means of curtains or blankets, the steam being conducted within this by means of an elastic tube fixed on the spout of the kettle. This matter requires particular attention if there is any tendency to implication of the windpipe. Eucalyptus leaves may be boiled with the water, the steam being thus impregnated with its active principles. Disinfectants should be placed about the apartment; and every precaution must be taken against the spread of the disease.

2. In a mild case of diphtheria it is sufficient to open the bowels; to administer some simple saline mixture; to allow a good quantity of beef-tea and milk, with ice to suck; and to employ soothing local remedies, namely, warm poultices or fomentations over the throat, and some mild gargle, such as milk with warm water, infusion of roses, or a weak solution of chlorate of potash.

3. In cases which are at all severe the **general treatment** demands strict attention. In the first place nutritious *diet* should be given from the outset, including abundance of milk and beef-tea, and it may be desirable to give it in a peptonized form. The patient should also take cool drinks freely, and suck small lumps of ice at frequent intervals. If there are any signs of depression, considerable nutriment is called for. *Alcoholic stimulants* are not required at first in most cases, but they must be given as soon as there is any indication that the powers of the system are failing. They are often needed in large quantities in adynamic cases, and children bear them well. The best stimulant ordinarily is brandy, some of which may be administered beaten up with eggs. Good port wine and iced champagne are also very valuable. If a patient cannot or will not swallow, or if there is severe vomiting, it is highly important to administer food and stimulants, as well as medicines, by means of enemata, and the remarks made with reference to the treatment of children suffering from scarlatina apply equally in the case of diphtheria.

4. As regards **medicinal treatment**, a mild *aperient* may be given daily, if required, in order to keep the bowels open. A *saline* drink, such as a solution of citrate of potash, or, still better, one containing chlorate of potash (3 i ad Oi). is decidedly useful, and should be taken freely. The medicine which I have found most efficacious in cases of diphtheria is tincture of perchloride of iron, which should be administered in full doses—℥ xx-xl every two or three hours. It may be beneficially combined with quinine or with dilute hydrochloric acid. Quinine alone in large doses has been recommended as almost a specific in this disease. Dr. Wade recommends iodide of potassium (gr. ij to iv), with chlorate of potash (gr. v to x), every two or three hours. Amongst other medicines specially advocated may be mentioned pilocarpine or jaborandi, calomel, and cyanide of mercury. Some have great faith



in *antiseptic* medicines, such as carbolic acid, sulpho-carbolates, sulphite of soda, or salicylic acid, but, in my opinion, these agents cannot be solely relied upon. Should adynamic or typhoid symptoms set in, ammonia and bark, camphor, ether, musk, and such remedies are called for; ether may be injected subcutaneously in extreme cases.

**5. Local treatment.**—This is often of essential importance in cases of diphtheria, and in every instance demands careful consideration. At the same time it must be acknowledged that there is by no means any unanimity of opinion as to what local measures answer best, eminent authorities, who have had large experience in the treatment of diphtheria, differing widely on this point; and some, indeed, think that the less there is done the better. The subject was thoroughly discussed at the International Medical Congress, in 1881, but without arriving at any definite conclusions. Of course different cases need to be treated in different ways.

It is almost universally agreed at the present time that under no circumstances should the diphtheritic patches be torn off from the surface of the throat, but in the course of the discussion Dr. Meyer described a plan of treatment advocated by Dr. Nix, of Rude, Denmark, which consists in repeatedly scraping away the membranes, and eventually the subjacent softened infiltrated parts, by means of a scoop, and cauterizing the scraped surfaces energetically with solid nitrate of silver. Dr. Prosser James is also of opinion that mechanical removal of the false membrane is not absolutely contra-indicated, especially from the larynx.

The application of ice externally to the neck is recommended by Dr. Morell-Mackenzie in the early stage, but this is contra-indicated when it causes pain, in young children, in advanced stages, and especially if gangrene be present. Dr. Cohen, of Philadelphia, advocates the local use of iced cloths applied over the neck and up to the ears, as an excitant of the respiratory nerve-centre, if respiration is interfered with by accumulation of false membrane. Poultices or fomentations to the neck may be useful in some cases.

Coming now to the employment of topical applications directly to the throat, these may be made either by means of a solid substance, as a stick of nitrate of silver; gargles; inhalations; injections with a syringe; the index-finger swathed in lint; a large and soft throat-brush, or a firm cotton-wool brush; atomized spray; or by blowing in powders through a straw or quill. The methods of application must be accommodated to the age and other conditions of the patient, and to the purpose intended, but more than one mode may be combined in the same case; for instance, patients who are old enough may often gargle with advantage, while inhalations, the spray, or other modes are employed at stated intervals. It may, however, be desirable to keep the throat-structures at rest, and then gargles should be avoided.

With regard to the nature of the applications which are used, it will be well to discuss them under their several heads.

*a. Caustics and Astringents.*—The objects of the application of *caustics* are to destroy the diphtheritic deposit, and to check its advance or extension. Those which are chiefly employed include nitrate of silver, either in the form of the solid stick, or in strong solution (Di to 3 i of water); and equal parts of hydrochloric acid and water. Powerful *astringents* are also made use of, such as tincture or solution of perchloride of iron mixed with an equal proportion of glycerine. The

liquids are applied by means of a brush. Most practitioners at the present time are decidedly averse to the use of these agents in any form. If they are employed at all, it should only be at a very early period, the application being thoroughly and efficiently made, around as well as over the membranous patches. It is really difficult to determine whether this treatment is positively beneficial, and it certainly may do much harm; while its repetition at more or less frequent intervals, as recommended by some, is decidedly mischievous. Weak astringent gargles, containing tincture of steel and glycerine, or dilute hydrochloric acid with or without chlorate of potash, tannin, alum, or other agents of this class, have been much employed; and tannin, or alum mixed with sugar, have been blown into the throat in the form of powders. Many, however, object to these agents likewise, as being useless or injurious.

*b. Antærics.*—Dr. Morell-Mackenzie lays great stress on the value of agents thus named, which form *varnishes*, and protect the surface of the diphtheritic membrane, excluding it from the air, and possibly interfering with the development of specific organisms. He has found a solution of tolu in ether (1 to 5) answer best; and directs that as soon as any false membrane is seen, both it and the surrounding mucous membranes should be carefully dried with blotting-paper, and immediately afterwards the varnish should be freely applied with a camel's-hair pencil. It must be very thoroughly applied below the deposit to prevent its extension downwards towards the larynx. The application need not be made more than twice a day, and does not interfere with the use of other remedies.

*c. Steam-inhalations.*—These are generally recognized as of great service in the treatment of diphtheria, and some practitioners rely upon them entirely. They may be employed at more or less frequent intervals, or even constantly, according to the severity of the case. They are particularly useful if the windpipe becomes involved. Dr. Morell-Mackenzie states that they are also of service in promoting detachment of the membrane, when this begins to separate after the application of varnish.

*d. Solvents.*—Several agents have been applied locally to the throat in certain ways, intended either to dissolve the diphtheritic membrane, or at any rate so to separate its molecular particles that it easily comes away. The agents chiefly employed for this purpose are lactic acid, lime-water or steam containing small particles of lime evolved in the process of slacking, salt-water, chlorate of potash, phosphate of soda, and borax. There is much difference of opinion as to the efficacy of any of these drugs. Lactic acid seems to be most in favour at present, and Morell-Mackenzie thinks that it fails so often because it is used much too diluted; he uses the American acid of sp. gr. 1.212,  $\text{m xxx}$  in  $\text{ʒi}$  of water. It has even been recommended to apply it undiluted. Dr. Young, of Florence, has employed with much benefit a solution of three drachms of lactic acid in eight ounces of lime-water. These applications are best made by means of some spray-apparatus, but they are also employed directly, and some of the drugs mentioned may be made into gargles, or mixed with glycerine and applied with a brush.

*e. Antiseptics.*—Of the value of these remedies there can be no doubt, especially when putrefactive or gangrenous processes are set up, and it is even thought that they have an immediate effect upon the diphtheritic deposit, destroying the germs in it. The chief agents used are carbolic

acid, Condyl's fluid, hypochlorite of soda, chlorine-water, boracic acid, salicylic acid, chloral hydrate, quinine, and sulphurous acid, either derived from burning sulphur, or in the form of solution. Most of these may be employed as gargles, sprays, or direct local applications. Morell-Mackenzie has seen much benefit from the application of syrup of chloral very freely with a brush every two hours.

In the foregoing remarks it has been impossible to do more than to give an outline of the local measures which have been advocated in the treatment of diphtheria. It must again be urged that every case requires careful consideration, and must be managed on its own merits. It is certainly safer to err on the side of doing too little than too much, especially as regards the employment of strong and irritating remedies. When the nasal cavities are affected, they should be frequently washed out by means of antiseptic injections, and the applications already discussed are also made to the nasal mucous membrane by some practitioners, when this membrane is involved.

6. The treatment which should be adopted when the **main air-tube** is involved has also been a subject of much controversy. If the difficulty of breathing is only moderate, relief may be afforded by giving an *emetic*, by means of which some of the exudation may be got rid of, the most suitable emetics being sulphate of zinc or ipecacuanha; subcutaneous injection of apomorphia has also been recommended. Paroxysmal dyspnoea may sometimes be relieved by inhalations of chloroform or ether. Should there, however, be evidence of considerable obstruction to the breathing, while the exudation continues to increase, the only possible hope lies in the performance of *tracheotomy* or *laryngotomy*, the former being suitable for children, the latter for adults. The trachea should be opened as high up as possible. It may be requisite to remove pieces of membrane through the tracheotomy-tube, but this should not be done by the mouth of the operator, except under urgent circumstances, but by some suitable suction-apparatus. The utmost precautions must be taken after the operation, with the view of preventing inflammation of the respiratory organs. The tracheotomy-tube must also be kept properly cleansed. The operation almost always affords temporary relief and prolongation of life; ultimately the issue is frequently fatal, but still cases do sometimes recover when apparently in almost a hopeless condition. I have had several successful cases under my care. Dr. B. W. Richardson has recorded a case in which the employment of artificial respiration, by means of the double-acting bellows, was efficacious in saving the life of a patient when *in extremis*, after tracheotomy had been performed. If signs are present indicating that the bronchi are extensively involved, no advantage can be derived from opening the windpipe.

7. **Symptoms or complications** may need special attention in diphtheria, such as sickness, or high fever. It is important to look to the urine, and should there be any signs of suppression, poultices and fomentations should be freely applied over the loins, or dry-cupping may be employed.

8. In order to hasten **convalescence**, change of air is most useful, especially to the seaside. Good diet is essential, with *tonics* and cod-liver oil. For the nervous sequelæ after diphtheria the best remedies are quinine, iron, and strychnine, along with a supporting and nutritious diet, which should include a moderate supply of stimulants. Blistering the nape of the neck has sometimes proved beneficial. Galvanism may be employed in connection with paralyzed parts.



## CHAPTER XVI.

## MUMPS—IDIOPATHIC PAROTITIS.

ÆTIOLOGY.—Mumps is an *acute specific disease*, and there can be no doubt that it is infectious. Almost always the complaint assumes an epidemic form, but it may be localized in houses or institutions where a number of young persons are aggregated together. It rarely occurs except in young individuals, being very common about the period of puberty, and also from five to seven years of age. Males are much more frequently attacked than females. Epidemics are most common in spring and autumn.

ANATOMICAL CHARACTERS.—Mumps is chiefly characterized anatomically by inflammation of one or both parotid glands. Some pathologists are of opinion that the process begins in the cellular tissue which pervades the gland-structure; others believe that a catarrh of the gland-ducts first occurs. The affected gland is hyperæmic and enlarged, being infiltrated with a serous fluid. Fibrinous exudation is not often observed, and a most important point to be noticed is that extremely rarely is there any tendency to the formation of pus. The tissues around are more or less infiltrated. As a rule the swelling rapidly subsides, and the gland returns to its normal condition. Occasionally the submaxillary gland is involved; and the testicles or other parts may be the seat of metastatic inflammation.

SYMPTOMS.—The *period of incubation* for mumps varies from fourteen to twenty-two days. In most cases there is some degree of premonitory fever, which generally lasts from one to three days before local symptoms are manifested, but occasionally they come on simultaneously. Pyrexia usually continues throughout the attack, but may subside on the appearance of the local signs, and it is seldom severe, nor does the patient feel particularly ill. A swelling or fulness appears in the region of either parotid gland, commencing just below the external ear, and then extending up to the zygoma, as well as to a variable extent over the face and down the neck, thus giving rise to much disfigurement. It has an elastic feel, being firmer over the centre than at the circumference. The skin may be reddened over the swelling, but is frequently unaltered. More or less pain or uneasiness is felt, with a sense of tension, increased by opening the mouth, by masticating, or by swallowing; there is also tenderness on pressure. Salivation occurs now and then; and occasionally deafness is complained of. In the great majority of cases the swelling subsides about the fifth or sixth day, and has quite disappeared in two or three days more; but in the meantime the gland on the opposite side frequently becomes affected, and goes through a similar course; or both glands may be involved simultaneously. A hardness occasionally remains for some time; and in very exceptional instances abscesses form in the gland, which open externally, or into the external auditory meatus. The submaxillary

gland is sometimes attacked; and the surrounding lymphatic glands, as well as the tonsils, are often enlarged.

An important character of mumps is its liability to *metastasis*, especially in adults. This event may be preceded by some dangerous symptoms. The testicle is most frequently attacked, orchitis setting in as the inflammation subsides in the parotid, with effusion into the tunica vaginalis and œdema of the scrotum. Occasionally the parotid gland and testicle are affected at the same time, or alternately for several times in succession. The orchitis generally runs a favourable course, but it may lead to wasting of the testicle. In females the labia, mammary gland, or ovary may be attacked in the same way. Meningitis is stated to have occurred in very rare instances of mumps.

TREATMENT.—In most cases of mumps but little treatment is required. It is necessary to keep the patient indoors, in a comfortable room, or even in bed if the complaint is at all severe. An *aperient* is useful at the outset, and the bowels should be kept regularly open. *Saline* medicines may be given, so as to promote the action of the skin and kidneys; and during convalescence quinine is serviceable. The *diet* should consist of liquids, especially milk and beef-tea. The only *local* treatment generally needed is to use hot fomentations, and to cover the parotid region with cotton-wool. The application of a leech or two may possibly be required. If an abscess forms it must be opened; and any hardness that is left may be removed by friction with oil, or by painting the surface with tincture of iodine. When metastasis takes place, it is recommended to endeavour to excite the return of inflammation in the parotid, by means of mustard poultices or blisters. Orchitis must be treated by means of rest, fomentations, and other appropriate measures.

---

## CHAPTER XVII.

### WHOOPI-NG- OR HOOPING-COUGH—PERTUSSIS.

ÆTIOLOGY.—Hooping-cough is generally regarded as an infectious disease, depending upon a *specific poison*, which may travel a considerable distance through the atmosphere, or be conveyed by fomites. The contagium is chiefly given off in the breath, and is believed to be present in the secretions from the nasal and respiratory mucous membrane. Letzerich is said to have produced hooping-cough in rabbits by inoculating the trachea with sputa from the human subject. Mr. Dolan, in his Fothergillian Essay, states that in repeating Letzerich's experiments, he found that blood did not produce any effects, but that the sputa and other secretions caused death. Microscopic examination of sputa revealed ordinary bacteroid forms, and Mr. Dolan thought he had observed a special microbe. Bürger has also described a specific micrococcus. Hooping-cough commonly occurs in an epidemic form, but may be sporadic. It may be communicated by infection before the characteristic "hoop" or "whoop" is developed. A second attack is scarcely ever observed.

The chief *predisposing causes* of pertussis are childhood, especially

after the second year; a cold and damp season or climate; and exposure to all causes of "cold."

**ANATOMICAL CHARACTERS AND PATHOLOGY.**—Most authorities regard whooping-cough as a peculiar catarrh of the mucous membrane of the air-passages, attended with hyperæsthesia; others think that it is entirely or partly due to some morbid condition in connection with the vagus nerve. There may be evidence of catarrh in fatal cases, but frequently these are absent. Those who advocate the nervous origin of the disease have described signs of inflammation about the vagus nerves; enlarged bronchial glands pressing upon these nerves; or congestion of the medulla oblongata and its membranes. In most cases, however, none of these appearances are observed. The most important morbid conditions associated with whooping-cough are those which are of the nature of complications, namely, bronchitis; lobular collapse of the lungs; acute insufflation or emphysema; dilatation of the bronchi; and catarrhal pneumonia. Rarely croup or meningitis may be present.

**SYMPTOMS.**—The *period of incubation* for whooping-cough is uncertain, but Dr. Squire has found evidence of the development of the disease as early as from two to four days after exposure to infection. The symptoms are divided into certain stages.

**First or Catarrhal stage.**—At the commencement whooping-cough presents no characteristic signs, there being merely pyrexia, which is often sharp, accompanied with signs of catarrh, namely, running from the nose, sneezing, redness of the eyes, frequent and usually severe paroxysms of cough, at first dry, but soon attended with a peculiar expectoration. This stage may last from two days to two or three weeks or more, and its duration and severity will indicate the probable duration and intensity of the entire attack.

**Second or Spasmodic stage.**—The fully-established disease is characterized by peculiar fits of spasmodic cough. A paroxysm generally sets in abruptly without any obvious cause, being in many cases preceded by a sensation of tickling in the throat, or some other unpleasant feeling. The cough is very severe and distressing, consisting of a number of short, quick, spasmodic or convulsive, and forcible expiratory puffs, followed by a prolonged, clear, shrill inspiratory sound or "hoop," these alternating for a variable number of times; if the fit is of very long duration, the cough at last becomes almost inaudible. It is usually terminated by the expectoration of a considerable quantity of thick, viscid, clear fluid, which may also be discharged through the nose; and not uncommonly vomiting takes place. Breathing being interfered with, the child presents the appearances characteristic of deficient aëration of the blood and venous congestion, and in prolonged attacks may become almost asphyxiated. Usually there is a feeling of much exhaustion, with soreness about the muscles of the chest, after a paroxysm, but these sensations soon pass away. As accidental occurrences may result bleeding from the eyes, nose, mouth, ears, or rectum; involuntary discharge of urine and fæces; hernia or prolapsus ani; or convulsions. *Physical examination* of the chest during a fit reveals that air does not enter the lungs properly. The physical signs of pulmonary complications can often be detected.

The frequency and duration of the paroxysms vary greatly, the one being generally in proportion to the other. As a rule the disease becomes intensified up to a certain point, attaining its height at about the end of the third, fourth, or fifth week, and then it subsides gradually.



During the intervals the patient is usually apparently well, but in severe cases there may be prolonged exhaustion, languor and debility, loss of appetite, headache, sleeplessness, pyrexia, and other symptoms; or various complications may give rise to their special clinical phenomena.

To Dr. T. Morton (*British Medical Journal*, June 10th, 1876) belongs the credit of having independently observed, and drawn attention to the frequent occurrence of ulceration about the frœnum linguæ in cases of hooping-cough. This phenomenon had been long known on the Continent, but in this country it had attracted little notice previous to Dr. Morton's observations; since then, however, it has come into considerable prominence. Prof. Henri Roger presented an elaborate report on the subject, and the following are the main practical conclusions at which he arrived. Sublingual ulceration is not an essential phenomenon of pertussis, and is very variable as to its frequency, depending on the violence of the paroxysms of cough, and on the disposition of the teeth in the first dentition. On the whole it occurs in about half the cases. There is no fixed time at which the ulceration is observed, but it is rarely seen before the third week from the time of infection, and in most cases several days later; it is never observed before the paroxysmal stage of hooping-cough is established, and occurs in direct proportion to its severity. The ulceration is never noticed in infants before dentition, but occurs more readily in those of ten or twelve months than in older children; it is never met with in adults. When the frœnum linguæ is short, no ulcer is produced; while if the arrangement of the teeth is anomalous, other parts of the tongue may be lacerated. There is no preceding vesicle or pustule, but the frœnum often presents a somewhat vivid redness, and then an erosion, or a linear division of the mucous membrane, with an appearance of granulations. At the point of section of the frœnum there is sometimes seen a transverse depression, sometimes a kind of pimple, or a small white and yellow patch, often of a pearly aspect. In other cases a small, median, oval ulcer is observed, with irregular edges, and a pale or reddish-grey base. This may extend some distance on each side of the frœnum, as well as in depth. Generally the ulcer is covered with a whitish or greyish exudation. From the facts observed by him, Roger concludes that the sublingual ulceration in hooping-cough is in no way specific, and that it does not hold any causative relation to the disease, which some have supposed to exist. Its origin is purely mechanical, the lesion being due to the impulsion forwards of the tongue in its hyperæmic state against the lower teeth during the paroxysms of coughing, when the frœnum is easily cut by the sharp lower incisors. The phenomenon may be of use in diagnosis in cases of pertussis, where the nature of the disease is not thoroughly declared, as this is the only complaint in which the cough is violent enough to propel the tongue against the teeth.

**Third or Decline-stage.**—There is no sudden transition to this stage, but a gradual diminution in the frequency and intensity of the paroxysms, while the cough loses its special characters, and expectoration becomes more easy, the sputa assuming an opaque and muco-purulent appearance, resembling the expectoration of ordinary bronchial catarrh; at the same time vomiting ceases. The general health also improves. Finally the cough stops altogether, and the patient is convalescent.

**COMPLICATIONS AND SEQUELÆ.**—Some of these are directly due to the cough; others are accidental. The chief complications and sequelæ

include bronchitis, which may become capillary; lobular collapse; emphysema or acute insufflation; rupture of air-vesicles, followed by subcutaneous emphysema; catarrhal pneumonia; pleurisy; phthisis; acute tuberculosis; croup; convulsions; cerebral apoplexy; meningitis; hernia; gastritis or enteritis, with obstinate vomiting and diarrhoea; and other specific diseases besides hooping-cough. It has been stated that the urine is frequently saccharine in hooping-cough. Mr. Dolan examined the urine of fifty children with confirmed hooping-cough, and only found traces of sugar in thirteen.

**DURATION AND TERMINATIONS.**—The entire *duration* of an attack of hooping-cough is very variable, but from six to eight weeks is stated to be the average. The third stage may continue for an indefinite period; and a *relapse* is not uncommon. Most cases terminate in recovery, but death is not an infrequent event, being occasionally due to the severity of the disease, but usually to complications. Some permanent organic mischief often remains behind; or the chest may become deformed.

**PROGNOSIS.**—Hooping-cough is always a serious disease, and calls for a guarded prognosis. The general circumstances which increase its gravity are that the patient is very young, suffering from dentition, or the subject of constitutional debility; residence in a large town; poverty and its consequences; and epidemic prevalence. The complaint is more dangerous in proportion to the number and severity of the paroxysms; to the degree of pyrexia; and to the gravity of the complications present.

**TREATMENT.**—Numerous *specific remedies* have been brought forward for the treatment of hooping-cough, but they all fail in most cases, the disease running its course unchecked, though it may be mitigated in its severity. The chief indications are:—1. To prevent or subdue the paroxysms of cough, at the same time care being taken that there is no accumulation of secretion in the bronchial tubes. 2. To obviate all complications, and treat them as they arise. 3. To attend to the general health; as well as to the state of the various secretions. 4. To promote convalescence. The means of carrying out these indications will now be considered.

1. It should be a constant rule, in the case of children, to pay immediate attention to any chest-symptoms, and this applies to the early period of hooping-cough. The patient should at the outset be kept in a warm room; be well-clad, with flannel next the skin; and have warm drinks, in order to promote perspiration. An *aperient* may be given; and a mixture containing liquor ammoniæ acetatis with vinum ipecac. should be administered. When the disease is established the most important remedies are *sedatives* and *antispasmodics*, for the purpose of allaying the paroxysms of cough. These must be given in minute doses, and their effects closely watched. The most efficient are belladonna, in the form of tincture, extract, or powdered leaves or root; opium, syrup of poppies, or morphia; hydrocyanic acid; conium; hyoscyamus; tincture of lobelia; cannabis indica; ether; chloroform; valerian; and musk. The alkaline carbonates, especially carbonate of potash, are believed to be useful, and either of these may be combined with one of the above remedies. In my own experience I have found most benefit from a combination of vinum ipecac. with hydrocyanic acid ( $m \frac{1}{4}$  to  $\frac{1}{2}$ ) or with tincture of belladonna. Some advocate the employment of inhalations of chloroform or ether.

2. Of the various **specific remedies** advocated for hooping-cough the chief are alum (which is in some cases decidedly valuable); dilute mineral acids, especially nitric; cochineal; arsenic; nux vomica or strychnine; bromide of potassium or ammonium; infusion of clover; quinine in small doses often repeated; tincture of myrrh; and repeated *emetics*. The agents last-mentioned are useful if there is any tendency to accumulation of secretion in the bronchi. *Metallic salts*, namely, those of copper, zinc, iron, and silver, have been recommended by various authorities, and may be useful in cases which tend to assume a chronic form. *Inhalations* of carbolic acid have also been advocated, and their use seems to have been attended with marked success in some instances.

3. **Local applications** have been tried, namely, touching the larynx with a strong solution of nitrate of silver; counter-irritation over the chest or along the vagus nerve; friction over the chest with opium, belladonna, and other liniments; and the application of a belladonna-plaster. These measures are of doubtful benefit.

4. The **general management** of patients suffering from hooping-cough is important. In bad weather they should be confined to the house altogether, or even to one room, maintained at a uniform temperature; but in favourable seasons it is decidedly beneficial for them to be out in the fresh air during the warmer part of the day. The clothing must be sufficiently warm. It is important to attend to the diet, and to the state of the alimentary canal; should dentition be proceeding, the teeth must be looked to. Children who are sufficiently intelligent should be taught to suppress unnecessary cough as much as possible.

5. **Complications** must be watched for, and treated as soon as they arise. Inflammatory affections do not bear lowering measures well in hooping-cough, and supporting treatment is indicated in the majority of cases.

6. During **convalescence**, *tonic* remedies, especially iron and quinine, are very useful. Change of air is also found to be highly beneficial in prolonged cases, or a sea-voyage. Good diet is needed, and a little wine is desirable sometimes. There is no protection against hooping-cough, except in keeping away from the source of infection.

---

## CHAPTER XVIII.

### INFLUENZA—EPIDEMIC CATARRH.

**ÆTIOLOGY.**—Influenza is essentially an *epidemic* disease, and usually attacks a large number of persons, either simultaneously or in rapid succession. It often breaks out in several parts of a district at the same time. The epidemic generally progresses in a certain direction, and is said to have a cyclical course; frequently, however, it prevails over a very large area. The inhabitants of large towns are chiefly affected, especially of those parts which are low, damp, overcrowded, and in other unfavourable hygienic conditions. Sometimes the disease breaks



out even at sea. It is very prone to modify the characters of other affections.

The *exciting cause* of influenza is believed to be a *specific poison*, which is conveyed only by the atmosphere, through which it is widely diffusible. The nature of this poison is quite unknown. Most authorities regard the complaint as being infectious; others consider it to be of malarial origin, and non-infectious. Inoculation cannot be effected in any way. Various hypotheses have been advanced to explain the occurrence of epidemics of influenza, but none of them are at all satisfactory. They break out at all seasons, but sudden changes of temperature are said to favour the development of the disease.

The chief individual *predisposing causes* of influenza are the female sex slightly; adult and advanced age; a low condition of the system; exposure to cold; and, it is said, the existence of chronic lung and heart diseases. The presence of any acute disease is believed to afford protection against influenza. One attack does not prevent another, and it has even been stated to render the individual more susceptible.

**ANATOMICAL CHARACTERS.**—The usual morbid appearances in influenza are those of catarrh of the mucous membrane lining the nose and its communicating sinuses, the mouth, throat, and respiratory tract; and of the conjunctivæ. In severe cases capillary bronchitis, pulmonary congestion and cedema, or pneumonia may supervene, the inflammation often involving both lungs. Sometimes the lining membrane of the entire alimentary canal, and that of the genito-urinary apparatus, are affected. Occasionally pleurisy or pericarditis supervenes; or very rarely meningitis. There is no splenic enlargement.

**SYMPTOMS.**—Influenza is a disease running a specific and definite course; and characterized by pyrexia, with much constitutional disturbance, and local symptoms due to the implication of the mucous membranes just indicated. The *period of incubation* generally lasts from a few hours to five or six days, but may extend to two or three weeks.

The *general symptoms* usually precede the *local*, but not always. The *invasion* is often markedly sudden, but in other cases it is gradual. The early symptoms are chilliness, lassitude, pains in the limbs, and, in some cases, intense headache, or nausea and vomiting; followed by fever, which is usually high, the skin being very hot and dry, though sometimes there is much sour perspiration. At the same time the patient complains of a feeling of great prostration and debility, apathy, lowness of spirits, and mental inaptitude; with severe aching and shooting pains about the chest, back, limbs, and neck; headache, giddiness, and general restlessness. The pulse is at first frequent, full, and bounding, but soon tends to become soft, weak, and slow. The urine is febrile. The pyrexia generally presents evening exacerbations, and it is said to be in some districts intermittent. In uncomplicated cases the duration of the fever usually varies from four to eight days, being frequently terminated by *crisis*, accompanied with copious perspiration, a free flow of urine depositing lithates, or diarrhœa; but in other cases the pyrexia subsides gradually.

The *local symptoms* vary according to the seat and extent of the catarrh. Usually this begins in the nose and conjunctivæ, and spreads downwards. The nasal cavities feel hot and dry at first, and the eyelids smart. Soon a watery acrid discharge flows abundantly, and there is much sneezing, the sense of smell being impaired or lost; occasionally profuse epistaxis occurs. The mouth, tongue, and throat feel sore, and

taste is defective. Severe pain is experienced across the forehead, owing to implication of the frontal sinuses. There may be pain along the Eustachian tube, with noises in the ears, and some degree of deafness. Examination reveals redness of those membranes which are visible; while herpes is often seen about the lips. The symptoms indicating implication of the air-passages are hoarseness; soreness and a tickling sensation along the larynx and trachea; more or less dyspnœa; oppression and stuffiness across the chest; paroxysmal cough, at first dry, but afterwards attended with bronchitic expectoration. These catarrhal symptoms usually subside from the fifth to the seventh day, the materials discharged undergoing the ordinary changes observed in the course of a catarrh. The tongue is furred, and there is much thirst, with loss of appetite. Gastro-enteric catarrh is evidenced by epigastric pain and tenderness; redness of the tongue; nausea or vomiting; and diarrhœa.

Cases of influenza differ much in their severity, and not unfrequently dangerous pulmonary complications arise, especially capillary bronchitis and pneumonia. The latter is apt to come on very insidiously, without any prominent symptoms. In these and other cases there is sometimes a tendency to adynamia, the tongue becoming brown and dry. Nervous symptoms are also occasionally prominent, namely, delirium, stupor, and convulsions.

**DURATION AND TERMINATIONS.**—Uncomplicated cases of influenza generally begin to convalesce from the fifth to the tenth day, but the duration may be much prolonged by complications. The great majority of cases end in recovery, but convalescence is often very tedious, and sequelæ are apt to remain, namely, great debility with nervous depression; neuralgic and rheumatic pains, which are common about the head and neck; or persistent cough. Occasionally chronic bronchitis, emphysema, chronic laryngitis, or phthisis is set up. Death is usually the result of lung-complications, but is sometimes preceded by adynamic symptoms.

**PROGNOSIS.**—The circumstances which render an attack of influenza grave are very early or advanced age; a feeble constitution; the presence of chronic pulmonary or cardiac disease; serious lung-complications, with great dyspnœa, inability to expectorate, and signs of imperfect blood-aëration; nervous disturbance; evidences of weak circulation; or adynamic symptoms. Some epidemics are much more fatal than others.

**TREATMENT.**—It has been satisfactorily proved that lowering treatment is injurious in influenza. In all cases it is advisable to keep the patient in-doors, in a cool, well-ventilated room, but protected from draughts. At the outset an *aperient* is useful, and in adults a dose of calomel seems to be beneficial, but repeated purgation is decidedly to be deprecated. Some recommend an *emetic* at the commencement, but such treatment is only indicated if there is much nausea. The *diet* must depend on the severity of the case; if it is slight, a moderate quantity of beef tea and milk may be allowed; but in severe cases, attended with much depression, a considerable amount of liquid nourishment is required. It is found preferable to give things cool, and cold or iced drinks are very grateful, and may be freely allowed. Dr. Parkes recommended a highly dilute solution of nitrate of potash with lemon-juice and sugar. *Alcoholic stimulants* are not required at first, unless there is much debility, except in old persons, who generally need them early; in some instances large quantities of wine or brandy are called for, but they must be used cautiously. Quinine is a remedy which is usually well borne, and does

much good; it is most valuable towards the decline of the disease, but may be given from the commencement.

The catarrhal symptoms are best relieved by inhalations of steam, to which some add ether, chloroform, or conium. Dr. Parkes suggested that direct *local applications* to the nasal mucous membrane and throat might be useful. For the bronchial catarrh ipecacuanha wine in full doses answers best, and it may be combined with some *sedative*, such as henbane or conium, care being taken that there is no accumulation of secretion. Opium should only be employed with particular caution. Poultices, sinapisms, and warm or anodyne fomentations to the chest are often valuable. Should capillary bronchitis or pneumonia supervene, a *stimulant* treatment is decidedly indicated, ammonia with decoction of bark and chloric ether, camphor, or other remedies of this class being administered, as well as alcoholic stimulants. Free dry-cupping is often valuable in these cases. The patient must be encouraged to cough should there be extensive bronchitis, in order to get rid of the secretion, and if this accumulates an *emetic* must be given.

If the general pains are severe, iodide of potassium with quinine often gives relief. It may be necessary to administer opium, or to have recourse to subcutaneous injection of morphia. Some practitioners recommend the administration of colchicum. Pyrexia may be moderated by cold sponging. Cold to the head, or the application of two or three leeches might be necessary, should dangerous nervous symptoms arise.

During convalescence *tonics* are needed, especially quinine and iron, with nourishing food, and wine or beer. Change of air is highly beneficial, and the patient must wear flannel, and guard against taking cold. *Expectorant* remedies are often required at this time, and excessive cough must be allayed by opiates.

---

## CHAPTER XIX.

### EPIDEMIC, ASIATIC, ALGIDE, OR MALIGNANT CHOLERA— CHOLERA MORBUS.

**ÆTIOLOGY.**—Cholera is an acute specific disease, which chiefly prevails as a virulent epidemic, but in certain regions is endemic. The *exciting cause* is undoubtedly a *specific poison*. This has been by many observers attributed to an organism, but all previous observations have been eclipsed by those recently made by Koch, who affirms that he has discovered a *specific bacillus*, which is more or less curved, and actively mobile. These are found only in the intestinal contents and evacuations. The real nature of this bacillus, and its relation to cholera, are questions at present undergoing thorough investigation, and no definite opinion can be given on the subject. The evidence is strongly in favour of cholera being an infectious disease, and that it is capable of being transmitted from one human being to another, but there is abundant proof that the stools constitute the main, if not the only channel of contagion, and that the great cause of the propagation of cholera is the contamina-



tion of water used for drinking purposes with the excreta of persons suffering from this complaint. It has been stated that the specific poison is innocuous when first discharged, and only acquires virulent properties after the lapse of four or five days. The admixture of an extremely minute quantity of cholera-stools will impart to great quantities of water the power of originating the disease, if taken into the alimentary canal. Probably the contagium becomes rapidly multiplied in the water, especially if this is exposed to the heat of the sun. Milk is also liable to convey the cholera poison, as in the case of typhoid fever, and possibly other articles of food may constitute the vehicle for its transmission. There does not seem to be any danger from merely being in the presence of those suffering from cholera, but emanations from the excreta into the atmosphere may generate the disease, being afterwards swallowed or inhaled, especially in places which are ill-ventilated.

The poison of cholera is regarded by some weighty authorities as having a malarial origin, and they deny that the malady is infectious. Pettenkofer believes that the germs of the disease, after leaving the human body, develop and multiply in the subsoil moisture under the influence of heat, and then rise as a miasm into the atmosphere.

The immediate cause of a *cholera-epidemic* is often obscure and difficult to detect, but a more correct knowledge of the ætiology of the disease will probably in future clear up much of the uncertainty on this matter. Many authorities maintain that cholera has been imported into Europe and other parts of the world from India, and Macnamara affirms that every outburst of the disease can be traced back through a series of cases to that country, it having been propagated thence by human agency, and always having followed the principal paths of human intercourse.

Certain conditions tend to promote the spread of cholera, and to aggravate its intensity, namely, a high temperature, with a moist, heavy, and stagnant atmosphere, cholera being therefore most prevalent in certain hot climates, and during hot seasons; a low position of a district; and unhealthy sanitary conditions, especially overcrowding, want of proper ventilation, accumulation of decomposing organic matter from imperfect drainage or any other cause, and impure and unhealthy food or water. The effects of these causes have been but too evident in the recent epidemic on the continent. Many other conditions have been supposed to affect the prevalence of cholera, such as the nature of the soil, the electrical state of the air, or the amount of the ozone present in the atmosphere; but the statements on these matters are extremely contradictory. It is found that most cases of cholera break out early in the morning.

Certain individual *predisposing causes* have been mentioned, but about many of these there is also much contradiction. Among the chief are fatigue, as after marching a long distance; destitution; errors in diet; abuse of purgatives; depressing mental influences, especially grief and fright; rather advanced age; race; intemperate habits; a bad state of health; certain occupations; and recent arrival in an infected district. One attack of cholera does not afford protection against another.

ANATOMICAL CHARACTERS.—The morbid conditions met with in the majority of cases of death from cholera may be thus summarized:—The temperature generally rises after death, and the body remains warm

for some time. Rigor mortis sets in very speedily, there being often powerful muscular contractions, displacing and distorting the limbs. The skin is mottled, more or less livid or blue, especially in dependent parts, and the limbs are shrunken, but these appearances are less marked than before death. Some striking peculiarities as regards the distribution of the blood are usually observed. The left cavities of the heart are contracted and rigid, and almost or quite empty, as well as the arterial system generally; the right cavities are distended with blood, as are likewise the pulmonary artery and its divisions, and the systemic veins. The pulmonary capillaries and veins, however, contain little or no blood, while the lungs are more or less collapsed, in some cases being almost completely airless and bloodless. Occasionally there is some degree of hypostatic congestion. The condition just described is regarded by some observers as being highly important in a pathological point of view, but Macnamara affirms that it is frequently due to *post-mortem* change, the blood being forced by post-mortem rigidity out of the left ventricle and arteries into the capillary and venous systems; and that if the examination is made immediately after death, the left side of the heart will be found as full of blood as the right. Most of the organs of the body are not congested, but, on the other hand, they are shrunken and pale, their capillaries being empty, but the alimentary canal and kidneys are commonly more or less injected. The blood is frequently much altered in its physical and chemical characters, being thick, dark, and tarry-looking, becoming lighter, however, on exposure. Most observers affirm that it is deficient in coagulability. Ecchymoses are sometimes seen under mucous and serous membranes; while the endocardium, and the fluid contained in serous cavities, are often stained with hæmatine.

The stomach and small intestines generally present more or less injection of their mucous lining, that of the intestines being also somewhat thickened and œdematous. The bowels are distended, and contain a quantity of materials in the main similar to those discharged during life. They differ, however, in having an abundant admixture of detached epithelium, which is believed therefore to be shed after death. Sometimes there are masses of gelatinous or fibrinous matter, or much grumous blood. The glandular structures are commonly enlarged and prominent, especially Peyer's and the solitary glands, the latter in rare instances presenting ulceration. In exceptional cases a diphtheritic deposit has been observed. The large intestines are usually contracted, but do not exhibit any special characters. The bladder is contracted, sometimes extremely so, and its epithelium, as well as that of the urinary passages and vagina, may be shed profusely.

In cases of cholera which survive into the *reaction-stage*, more marked *post-mortem* appearances are visible, indicating gastric and intestinal inflammation; acute Bright's disease; extreme congestion, low inflammation, or gangrene of the lungs; serous inflammations of a low type; or other complications to be hereafter mentioned; whilst those characteristic of cholera disappear more or less.

**SYMPTOMS.**—Cholera affords in typical cases a well-defined clinical history, which it is customary to divide into certain stages. The *period of incubation* is of uncertain duration, but it may range, according to different observers, from one to eighteen days. From two to four days is a common incubation-period (Squire).

**1. Invasion-stage.**—In many instances this stage is not apparent, the disease manifesting itself suddenly in all its virulence. Diarrhœa is the

most important premonitory symptom observed, which may or may not be attended with griping. Nervous disturbances have also been noticed sometimes, though many doubt their reality, such as a sense of languor, debility, exhaustion, or marked depression, trembling, altered expression of countenance, unaccountable lowness of spirits, headache, giddiness, noises in the ears, epigastric uneasiness and oppression, and various other symptoms. This stage is of short duration.

**2. Evacuation-stage. Stage of Development.**—At this time the prominent symptoms are severe purging and vomiting, the materials discharged having special characters; constant thirst; painful cramps; and signs of marked general disturbance, in the direction of prostration and collapse, combined with great restlessness. The purging is the first symptom, and it often sets in early in the morning, becoming speedily very frequent or almost constant, being followed by a sense of much exhaustion and of sinking at the epigastrium. The stools are very profuse, watery, at first coloured by the previous intestinal contents, but soon presenting peculiar characters, and being named “rice-water” stools, from their resemblance to water in which rice has been boiled. At this time they are perfectly liquid, exceedingly pale, somewhat opalescent or occasionally whitish or milky, having but little odour. When this liquid is allowed to stand, more or less sediment falls, resembling flakes of boiled rice, leaving a whey-like fluid above, which has a specific gravity of from 1005 to 1010, and a neutral or slightly alkaline reaction. The quantity of deposit is actually very small, Dr. Parkes having found the amount deposited from a pint not to weigh when dried more than 4 grains. Chemically the evacuations consist mainly of water, holding in solution a considerable proportion of salts of soda and potash, especially chloride of sodium, with but very little albumen or other organic matter. The sediment has been supposed to be modified fibrin or mucus. Microscopically the objects which have been described are abundant granules; active amœbiform particles of bioplasm; nuclei; round, nucleated, and granular cells, resembling pus or exudation-cells; peculiar hyaline cells; a few epithelium particles; fungi, bacilli, vibrios; and occasionally triple phosphates. In exceptional cases blood or its colouring matter is discharged. Often the diarrhoea is painless, but there may be griping, and a burning sensation at the pit of the stomach is frequently experienced. Vomiting comes on later, and is less severe and profuse, occurring chiefly after anything is taken. The vomited matters, which are often expelled with much force, at first consist of the previous stomach-contents, but soon assume the characters of a clear, colourless or yellow, thin fluid, mixed with mucus and disintegrated epithelium. The cramps usually set in at the same time as the rice-water stools appear, affecting mainly the muscles moving the fingers and toes, the calves of the legs, and the thighs, but sometimes the abdominal muscles also suffer. Thirst soon becomes a distressing symptom.

In proportion to the severity of the purging and vomiting a sense of exhaustion is felt, and signs of depression and collapse appear, culminating, if the symptoms do not subside, in those characteristic of the next stage, under which they may be more conveniently described.

**3. Stage of Collapse. Algide stage.**—There is no abrupt commencement of this stage, but a more or less rapid transition from the former. The aspect of the patient becomes highly characteristic. The features are pinched and shrunken, assuming a leaden or livid hue,



especially about the lips; the eyeballs sink in their sockets, while the lower eyelids fall, and the eyes are half-closed; the nose becomes sharp and pointed; and the cheeks are hollowed. The entire surface of the body is more or less cyanotic, but especially that of the extremities, while the skin presents a peculiar wrinkled and shrivelled aspect, being often at the same time bathed in cold sweats, the hands appearing sodden like those of a washerwoman. When the skin is pinched up the folds disappear slowly. The *temperature* rapidly falls, and the surface soon assumes a death-like coldness, particularly over exposed parts, though it is stated that the temperature within the body is usually increased. In the mouth it ranges from  $79^{\circ}$  to  $88^{\circ}$ , in the axilla from  $96^{\circ}$  to  $97^{\circ}$  (Goodeve); in the vagina and rectum it is considerably higher. The circulatory organs and blood afford evidences of grave disturbance. The radial pulse is exceeding feeble and thready, or even extinct, and in bad cases no pulsation can be felt in the brachial, or even in the carotid arteries, while the cardiac impulse and sounds become extremely weak or almost imperceptible. The general capillary circulation is seriously embarrassed. When a vein is opened, little or no blood escapes, this fluid being thick, viscid, and tar-like. The respiratory functions are also impeded. There is paroxysmal dyspnoea, accompanied with gasping for breath, and a sense of oppression and craving for air, at last becoming almost continuous. The expired air is cold, and very deficient in carbonic anhydride. The voice is extremely weak, and often becomes ultimately a mere whisper or even entirely inaudible. The nervous system necessarily suffers severely. As a rule muscular prostration is marked, but the strength is now and then wonderfully maintained. There is great restlessness and jactitation, with wakefulness, the patient tossing about and throwing off the bed-clothes. At first much anxiety is felt, but this soon changes into apathy and indifference. Occasionally headache, giddiness, tinnitus aurium, muscæ volitantes, or cloudiness of vision are complained of. The mind is for a time clear though inactive, but in cases ending fatally stupor sets in, followed by coma. Reflex excitability is markedly impaired. Cramps continue from time to time.

A prominent feature of this stage is the *impairment or complete cessation of the functions of absorption and secretion*. No saliva is formed; while the urine is almost entirely or quite suppressed. At this time the purging and vomiting diminish in amount and frequency, though there may be much retching; the stools are less liquid usually, they contain mucus or gelatinous masses, and are often passed in bed. Ultimately they may become extremely offensive, the smell resembling that of decomposed fish. Intense thirst is experienced, with a sense of heat in the epigastrium, the patient constantly craving for cold drinks, which are swallowed with spasmodic avidity, probably to be immediately rejected. The tongue feels cold to the touch.

The intensity of the symptoms just described varies much. When they are developed in their full severity recovery seldom takes place, death occurring more or less speedily, being preceded by signs of more and more complete interference with the respiratory functions, increased capillary stagnation, and coma. In most cases the temperature rises with the approach of death. In the less marked cases, however, recovery follows not unfrequently, and there is no condition which is utterly hopeless. The phenomena attending restoration will now be considered.

4. **Stage of Reaction.**—The prominent signs indicating restoration after an attack of cholera are a gradual change in the expression, general aspect, and colour; improvement in the pulse and cardiac action, with diminution in the capillary stasis; and return of heat to the surface. Breathing becomes at the same time more regular and calm; while the restlessness, thirst, and other symptoms abate; and the secretions are re-established. The patient often falls into a calm doze; vomiting ceases, but a little purging may continue, the stools, however, containing bile. There is said to be no actual rise of temperature at the beginning of reaction, but a cooling of the interior parts of the body while the outer parts warm up (Jüterbogk). This stage may terminate in speedy convalescence, but such is often not the case, certain *complications* or *sequelæ* being very liable to supervene, or now and then a *relapse* taking place, which may prove fatal. Occasionally also the reaction is imperfect, and the symptoms continue to a greater or less degree, there being no pyrexia, and the patient dying in a few days, or sinking into a typhoid state, or ultimately making slow progress towards recovery. A most important matter during the progress of convalescence is to look for the re-establishment of the secretory functions, especially as evidenced by an increase in the quantity of urine. The temperature not unfrequently rises above the normal without any obvious cause.

**COMPLICATIONS AND SEQUELÆ.**—Among the less important complications and sequelæ mentioned by Dr. Goodeve are mild consecutive fever, with general disturbance, which may assume a remittent or intermittent type, usually ending in recovery in a few days; obstinate vomiting, often associated with more or less gastritis, which may become very serious; frequent hiccup, with gaseous eructations, and loss of appetite; and want of sleep. The more grave complications usually met with are acute desquamative nephritis with signs of uræmia, the renal disease sometimes becoming chronic; “cholera-typhoid”; severe enteritis, occasionally of a diphtheritic character; chronic diarrhœa or dysentery; and low pneumonia or pleurisy. The urine is usually albuminous, and may contain some hyaline casts during convalescence, but in favourable cases it soon becomes normal. In some instances, however, it assumes the characters indicative of acute renal disease, while other symptoms of this condition appear, along with signs of uræmia. The term *cholera-typhoid* has been used vaguely; the symptoms are merely those pertaining to the *typhoid state* generally, and they may be associated with uræmia, or with any adynamic inflammation, or they are sometimes independent of obvious morbid changes, being then probably due to blood-poisoning. The temperature rises should inflammatory complications set in.

A *cholera-eruption* or *exanthem* has been described, but though erythematous, maculated, papular, urticarial, or even purpuric eruptions appear in some instances, there is none characteristic of cholera.

As occasional sequelæ are mentioned inflammation of the genitals; parotid bubo; ulceration of the cornea and its consequences; gangrene of various parts; and the formation of bed-sores, boils, or ulcers. In many cases, especially if the illness has been prolonged, a condition of marked debility and anæmia remains behind.

**VARIETIES.**—In some cases of cholera the *collapse-stage* sets in after little or no previous purging or vomiting, death ensuing very speedily. On the other hand this stage may be imperfectly developed. During an epidemic of cholera numerous cases of diarrhœa are met with, lasting

several days, and generally unattended with pain, to which the terms *choleraic diarrhœa* or *cholerine*\* have been applied. The stools are usually pale, liquid, and copious; there may be vomiting and cramps; while the patient feels much exhausted and seriously ill. These cases have been regarded as the result of a milder dose of the cholera poison, and they may pass into true cholera, though sometimes they prove fatal without becoming distinctly of this nature. Towards the end of some epidemics the choleraic diarrhœa passes into a kind of low fever.

Here also may be mentioned the so-called *sporadic, bilious, or English cholera*, or *summer diarrhœa*, the symptoms of which sometimes closely resemble those of true cholera. Ordinarily they are less severe; the stools and vomited matters contain bile; there is more griping; urine is not entirely suppressed; the duration is longer; while the mortality is much less (Goodeve). Some cause, such as an error in diet, can generally be found for the attack of English cholera.

**PATHOLOGY.**—All authorities seem agreed that cholera is primarily due to the action of some *specific poison* upon the system, the nature of which is at present doubtful. Beyond this point there are wide divergencies of opinion. Dr. George Johnson and others consider that all the phenomena of cholera are directly due to this poison, which acts first on the blood, in which it is enormously multiplied, and then affects certain portions of the nervous system, especially the sympathetic and the nerve-centres influencing the respiratory and circulatory organs, thus leading to paralysis of the coats of the intestinal smaller arteries and capillaries, with consequent distension and free transudation, while the small vessels of the lungs are spasmodically contracted, and will not allow the blood to pass through these organs. According to this view the purging and vomiting are regarded as *eliminatory* of a morbid poison. Another class of pathologists believe that the cholera-poison acts primarily and immediately on the alimentary canal, and that the subsequent phenomena of collapse are the consequence of the intestinal disease, and of the violent purging and vomiting accompanying it, being due to the physical changes in the blood, and to the disturbance of the sympathetic nervous system thus induced. The blood does unquestionably undergo some very marked alterations in cholera. As already stated, it becomes very thick and dark. Water is rapidly withdrawn, both from the liquor sanguinis and from the corpuscles, and hence a serious disturbance arises in the relation of these constituents to each other. Soon also the proportion of saline ingredients is much diminished, while that of the organic elements is relatively increased, especially of the corpuscles and albumen. The specific gravity is considerably raised. Occasionally the blood is acid. During the *collapse-stage* it may contain urea and other products of decomposition, some of which originate in changes in the stagnant blood itself. In the *reaction-stage* these materials are often very abundant. Drs. Lewis and Cunningham have described peculiar microscopic changes observed in choleraic blood removed during life, as well as in that obtained after death, namely, the rapid development and multiplication of active bioplastic bodies, ultimately forming cells; and they consider that these changes may go on in the body, and that this may account for the abundant bioplasts and cells found in the cholera-evacuations. The alterations in the blood will explain the thirst, and the drying-up and shrivelling of the tissues;

\* The term *cholerine* has also been applied by the late Dr. W. Farr to the poison which originates cholera.



as well as to a great degree the capillary stagnation, the disturbance of the respiratory and circulatory functions, and the suppression of secretions. At the same time these phenomena are partly to be accounted for by the influence exerted upon the heart through the sympathetic nerve, the feeble action of this organ aiding in producing many of the symptoms, because it cannot drive the blood through the vessels. The dyspnoea is also to some extent due to pulmonary collapse. The cyanotic appearance is partly the result of stagnation of blood; partly of its concentrated and venous character.

It is important to note that the fact of purging ceasing during the collapse-period does not always imply that fluid has ceased to transude, for at this time the intestines are often paralyzed, and may contain a large quantity of fluid which they are unable to expel.

The phenomena which characterize the *reaction-stage* are due chiefly to the deleterious products which accumulate in the blood, and they are more liable to arise in proportion to the duration of the collapse-stage, and to the time which elapses before secretion is properly established after reaction has commenced. These phenomena are probably promoted in some cases by improper employment of stimulants and drugs.

PROGNOSIS—MORTALITY—DURATION.—It need scarcely be remarked that the prognosis in cholera is always very grave. The *mortality* varies in different epidemics, ranging from 20 or 30 to 70 or 80 per cent.; it is highest in the early part of an epidemic. On an average more than half the cases recover. The chief general circumstances rendering the prognosis worse are infirmity and old age; unfavourable hygienic conditions; previous intemperance; debility from any cause; or the existence of renal disease. During the actual attack the prognosis, both immediate and remote, is more grave in proportion to the rapidity with which signs of collapse set in; and to their intensity and duration. Rapid cessation of pulsation in the larger arteries; great disturbance of the respiratory functions; a striking fall in temperature; marked cyanosis; or a tendency to coma are all very bad signs. The cessation of purging is sometimes unfavourable, indicating paralysis of the intestines. When reaction sets in, there are many dangers to be feared, but a more favourable progress towards convalescence is to be expected in proportion to the rapidity with which the functions of secretion and absorption are re-established, and to the continuous and regular improvement in the symptoms. Most of the subsequent complications or sequelæ are exceedingly serious.

The *duration* of cases of cholera may range from a couple of hours to some weeks, reckoning in its sequelæ. The average duration of fatal cases is from two to three days. The length of each stage varies considerably.

TREATMENT.—1. The **preventive treatment** of cholera is extremely important, and calls first for consideration. During an epidemic of this disease all the rules laid down in a former chapter, relating to the management of contagious diseases and epidemics, must be rigidly carried out, under the personal superintendence of competent individuals. *Cleanliness* and *free ventilation* are highly important. Particular attention is demanded with regard to the *choleraic stools*, which should be immediately disinfected, and so disposed of that there shall be no danger of their becoming mixed with drinking water, care being taken that the sewers and drains are kept in good order, and that they are well flushed with disinfectants from time to time. On no account must the excreta be

recklessly thrown out on the ground, and if there is no proper place to receive them, they should be buried a considerable depth in the earth, away from all habitations. Most important is it to attend to the *water-supply*, and to see that the water used is abundant and pure. It should always be filtered. Food must also be looked to, and especially milk. Persons exposed to infection should be warned against errors in diet, intemperance, and other injurious influences; while everything must be done to calm the minds of those inhabiting an infected district, and to prevent needless fear and depression. During an epidemic of cholera it is very properly the custom to organize staffs of medical men and their assistants, in order to carry out thoroughly all the necessary preventive measures, and to treat cases as soon as they arise, house-to-house visitation being practised daily. It is highly desirable that all persons who are able to do so should remove from infected districts.

With regard to the disposal of the dead, the bodies should be buried as soon as possible, each being surrounded in its coffin with some disinfectant, such as a mixture of charcoal, lime, and carbolic acid. An apartment which has been occupied by a cholera-patient must be thoroughly disinfected and cleansed, and it is often necessary to destroy clothing and bedding.

2. The **curative treatment** of cholera is unfortunately in many cases quite hopeless, but often much may be done, particularly at an early period of the complaint. It is a great mistake to follow any routine plan in all cases, but the practitioner should be guided as to the measures to be employed by the actual condition of the patient, and the stage of the disease. Personal supervision on the part of the medical attendant is desirable, so far as this is practicable, in order to see that the treatment is properly carried out. The earlier this treatment is commenced the more likely is it to be successful, and patients should take to their bed at once. During a cholera-epidemic, the slightest case of diarrhœa ought to receive the most prompt attention, and the public should be instructed on this point, places being established where they may at once obtain the necessary medicines.

In the *evacuation-stage* two directly contrary plans of treatment have been employed, most practitioners using measures for checking the diarrhœa; a few encouraging it, acting on the principle that it is eliminatory of a poison. For this purpose the use of castor-oil at frequent intervals, calomel, sulphate of magnesia, and other *purgatives* has been advocated. Undoubtedly in some cases of early choleraic diarrhœa a dose of castor-oil is beneficial, with the view of getting rid of irritant matters; but, apart from all theoretical considerations, experience has proved that the systematic employment of the castor-oil treatment is by no means attended with favourable results. Most decidedly the evacuations ought to be checked as soon as possible, in my opinion. Opium is the great remedy for this purpose, the best preparations being the compound soap pill, tincture of opium, liquor opii sedativus, or Dover's powder, the liquid preparations answering best if there is much vomiting, or if speedy absorption is required. This drug, however, requires much care in its employment in cholera. Should there be signs that the collapse-stage is approaching, particular caution is necessary in administering opium, while it is inadmissible if this stage has become established. If the indications are favourable, it seems best to give a full dose at once, and subsequently to repeat it in small quantities as it is needed. Should the first dose be vomited, it must be repeated after



a short interval. Various *astringents* are also useful, especially acetate of lead (gr. ij-iii); tannin or gallic acid (gr. x-xxx); and dilute sulphuric acid. Some prefer giving opium by itself, and administering the remedies just mentioned between times; others combine them with opium. The experience of some cases seems to indicate that the encouragement of very free sweating at the commencement of an attack of cholera may prove beneficial.

In the *collapse-stage* opium must on no account be given, but if purging continues, one of the astringents just mentioned may be employed. At this time most reliance is to be placed on the judicious use of *stimulants*. Before any indications of collapse appear, stimulants are not called for, but as soon as any weakness of the pulse is observed, or other signs of sinking, their administration should be commenced. The practice of pouring in large quantities of alcoholic stimulants is to be highly deprecated, and their employment needs the most careful regulation. The most serviceable are brandy with iced water, and champagne. They must be given in small quantities; at more or less frequent intervals according to circumstances; and their administration must be mainly guided by their influence upon the pulse. If the purging has ceased, brandy may be given in enemata with beef-tea. Diffusible stimulants are also of service in this stage, such as aromatic spirit, solution, or carbonate of ammonia; the various ethers; camphor, which has been vaunted as a specific; musk, and similar remedies. These may be combined with essential oil of peppermint, cinnamon, or cajeput. Niemeyer found a few cups of hot strong coffee useful in many cases.

With regard to *diet*, it is useless to give any nourishment at the commencement of an attack of cholera, as this is only rejected immediately. A little beef-tea, chicken-broth, or arrowroot and milk may be tried at a later period, if the vomiting ceases. The patient should be allowed an unlimited supply of ice to suck throughout, which Macnamara considers invaluable in the treatment of cholera, but he lays great stress on prohibiting every kind of drink until the collapse-stage sets in, when iced water in moderation may be permitted. Enemata of iced water, or, on the other hand, those of warm milk have been recommended.

*Local measures* are often of much service for the relief of symptoms. A large mustard poultice should be applied at once over the abdomen, and repeated as occasion requires. For the relief of the cramps, hot-bottles, sinapisms, and friction, either with the hand alone, or with turpentine or chloroform liniment, may be employed. If they are very severe, inhalation of chloroform is admissible. In the collapse-stage Niemeyer recommended the application of cold compresses over the abdomen.

Should *reaction* set in, the utmost care must be exercised, and the natural progress towards convalescence must not be interfered with by needless medication. The diet requires particular attention at this time, only the blandest liquid food being allowed in moderate quantities, and this regulation of food is demanded until the patient has been entirely restored, it being improved gradually as the stools become natural. Water may be freely allowed during this stage, and it has been recommended to dissolve some chloride of sodium and carbonate of soda in it, in order to replace the loss of these salts. It is highly important to watch for the re-establishment of the secretions, and, if necessary, measures may be adopted to encourage this result. *Complications and sequelæ* must be treated as they arise. It need only be mentioned here that it is



not always desirable to check diarrhoea at this time, should the stools be very offensive; and that inflammatory affections require a supporting treatment. *Tonics* and iron are often serviceable during convalescence.

It is important to pay strict attention to the *cleanliness* and *ventilation* of the sick-room, and to see to the *immediate removal* and *disinfection* of wet and soiled bed-clothes, it being advisable also to have a mackintosh placed under the patient. Due precautions must be taken against bed-sores, frequent examination of parts pressed upon being made. Should the urine be retained, the bladder must be emptied by means of the catheter; if it is suppressed, hot applications over the loins, and dry-cupping are indicated.

For such a disease as cholera it is not to be wondered at that innumerable *specific* modes of treatment have been advocated, but all have proved equally inefficient. Without making any comment, I merely mention some of the most prominent, namely:—the administration of saline salts, chiefly carbonates and chlorides, either freely by the stomach, by enemata, or by injection of a warm solution into the veins; the use of warm, vapour, or hot-air baths, or of the wet sheet; application of ice to the spine; the employment of *antiseptics*, such as carbolic acid, sulpho-carbolates, or chloralum; inhalation of oxygen; inhalation of nitrite of amyl; the administration of calomel, gr. i-ij, at frequent intervals, or of bisulphide of mercury. Salicine and salicylic acid have also been specially recommended. The various cholera-drops and pills, which are held in repute in different countries, are made up of stimulants, generally combined with some preparation of opium.

---

## CHAPTER XX.

### GLANDERS AND FARCY—EQUINIA.

**ÆTIOLOGY.**—These affections, which are occasionally observed in man, are transmitted from the horse, ass, or mule. It is doubtful whether they are distinct diseases, or varieties of the same malady. They are produced by the inoculation or contact of a *specific poison*, which is contained chiefly in certain discharges from the nasal cavities, but also in the excretions, secretions, and blood; and they are rarely met with except amongst those whose occupation brings them much into contact with horses, such as ostlers and grooms.

Glanders may be propagated in consequence of a horse snorting, and thus expelling a quantity of the discharge from the nostrils on to an individual, or into the surrounding air, from which it is afterwards inhaled. The poison may also be conveyed by fomites, to which the discharges have become attached. It is even said that glanders may arise from breathing the atmosphere of a stable inhabited by glandered horses. These diseases can be re-transmitted to the horse and ass, as has been proved experimentally, and there is reason to believe that they are capable of being communicated from one human being to another. They have been attributed to a *specific bacillus*.

**ANATOMICAL CHARACTERS.**—Glanders and farcy are characterized by certain peculiar morbid changes. In the former, hard nodules form in

connection with the mucous membrane of the nose, mouth, throat, and respiratory passages; as well as in the lymphatic glands, skin, subcutaneous tissue, muscles, lungs, and other organs. These are made up of cells, the result of proliferation, which soon degenerate, the nodules rapidly becoming soft and breaking down, forming abscesses and ulcers. The Schneiderian membrane is inflamed, and presents little pustular elevations or patches, which finally break out into excavated ulcers, and these may lead to necrosis of the cartilages and bones, with perforation of the septum. The frontal sinuses may contain a puriform fluid. The larynx, trachea, and bronchi are also frequently affected. The lungs present pneumonic patches or abscesses. Small yellow elevations may be seen on the pleuræ, with fibrinous deposits underneath. The lymphatic glands are enlarged, softened, and reddish. Superficial petechiæ, ecchymoses, or gangrene are sometimes observed; or there may be suppuration or gangrene of the subcutaneous tissues, or in the substance of the muscles. A peculiar eruption appears on the skin.

Farcy differs from glanders in that the nasal mucous membrane is not affected, but so-called *tubercles*, *buds*, or *tumours* form in connection with the skin, which break down into deep, unhealthy ulcers; or the lymphatic system may be chiefly involved. In acute farcy an extensive fatty embolism of the pulmonary vessels is supposed to occur.

**SYMPTOMS.—1. Acute Glanders.**—The *period of incubation* is usually from three to eight days. After inoculation local signs of inflammation appear first as a rule, the neighbouring lymphatics being also affected. Ordinarily the disease is ushered in by such general symptoms as rigors, languor, pains in the limbs and joints, headache, often vomiting and diarrhoea, with a certain degree of pyrexia. Soon subcutaneous formations are noticed, especially on the face and near the joints, which quickly change into abscesses containing unhealthy and foetid pus. Over these the skin becomes red or violet, and sometimes limited gangrene sets in. Usually a peculiar cutaneous eruption appears, especially on the cheeks, arms, and thighs. It begins in the form of small, intensely red spots, which soon become papular, and afterwards pustular. This eruption is not due to any exudation under the cuticle, but to circumscribed destruction of the true skin. Dark bullæ often appear also on the face, trunk, fingers or toes, and organs of generation, followed by gangrene. Erysipelatous inflammation of the nose, eyes, and surrounding parts is not uncommonly observed, which may extend to the scalp. The eruption is preceded and accompanied by profuse foetid sweats.

A prominent symptom of glanders is a discharge from the nose, at first thin and scanty, but soon becoming abundant, purulent, viscid, extremely foetid, and often sanious. It clogs the nostrils and obstructs breathing, while it escapes through the posterior nares, causing much distress and irritation. A thick matter also comes from between the eyelids, and sometimes from the mouth, which is then the seat of ulceration and pustules. The submaxillary lymphatic glands may be enlarged.

The constitutional symptoms increase in severity as the case progresses, being more or less of a typhoid character, and indicative of septicæmia. Thus there is marked prostration, with a brown and dry tongue, and a very rapid, feeble, and irregular pulse. Diarrhoea and

tympanites are frequent symptoms, the stools being exceedingly fœtid, and sometimes bloody. There is also dyspnœa, with hurried breathing, hard cough with but little expectoration, weak voice, and foul breath. Delirium and coma ultimately set in, and death terminates the scene, in most cases at the end of the second or beginning of the third week, but it may happen much earlier or later than this.

2. **Chronic Glanders.**—This variety is very rarely met with, and is usually a sequel of farcy. Its chief symptoms are lassitude and articular pains; sore-throat; disagreeable or painful sensations in the nose, with more or less puriform and bloody discharge; cough with expectoration, dyspnœa, and altered voice. After a time ulceration may be observed on the mucous membrane of the nose, followed by caries or perforation. The pharynx may also be ulcerated. There is no eruption. The general symptoms are less marked than in the acute form of glanders, but the latter may supervene. The duration of chronic glanders is very variable.

3. **Acute Farcy.**—The great difference between this affection and glanders lies in the want of implication of the nares. In one class of cases the eruption is present; in another there is no eruption, but merely inflammation of the lymphatic glands and vessels, with soft tumours under the skin, named *farcy buttons* and *farcy buds*. The former group are by far the more serious.

4. **Chronic Farcy.**—After constitutional symptoms, subcutaneous tumours form, which become abscesses, and these discharge their contents, leaving foul, deep, and indolent ulcers. As a consequence there is wasting, with great debility. The complaint may end in true glanders; or death may result from exhaustion or pyæmia. Occasionally recovery takes place. The duration is very variable.

5. **Equinia Mitis.**—An affection is thus named which is derived by contagion from horses suffering from the "*grease*." The symptoms are fever, depression, and shivering; with a pustular eruption, which dries up into scabs, and these fall off, leaving distinct scars.

PROGNOSIS in all these affections is very grave, for they almost always prove fatal. Chronic farcy may terminate in recovery.

TREATMENT.—The most important matter is to exercise due precautions for the prevention of these diseases. If inoculation should happen, the affected spot should be immediately destroyed by some escharotic. A supporting, stimulating, and tonic plan of treatment is the only one which offers any chances of success in dealing with either of these affections; at the same time strict attention being paid to cleanliness and other *hygienic conditions*. Abscesses should be opened as they form. *Antiseptics* internally might, perhaps, be of service; and these agents would also be useful as local applications.

---



## CHAPTER XXI.

## MALIGNANT PUSTULE--ANTHRAX--CHARBON.

THIS malady has attracted considerable attention of late years, and has been particularly discussed in this country under the name "Wool-sorter's disease." Therefore, although it is a comparatively rare complaint, it needs to be considered in some detail.

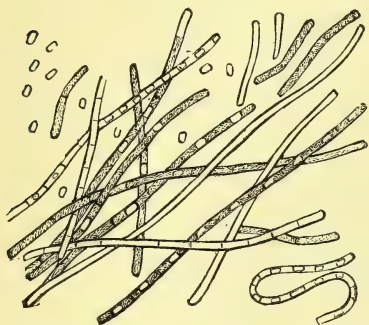
**ÆTIOLOGY AND PATHOLOGY.**—Malignant pustule is a *specific disease*, conveyed by contagion, mainly from sheep and oxen, but also from horses, and possibly from other animals which it affects, such as elephants, camels, etc. In these animals it is known under the name "splenic fever," in connection with the mitigation of which the remarkable experiments of Pasteur, alluded to in the chapter on CONTAGION, were made. The poison is transmitted either directly or indirectly, but almost invariably from the dead carcase, rarely, if ever, from the living animal. All parts of the dead bodies are capable of conveying the disease. The contagium is usually transmitted by direct inoculation, the matter being brought in some way into contact with an abraded or wounded surface, but there is reason to believe that it may be absorbed by the unbroken skin in parts where this structure is very thin. Rarely malignant pustule arises from eating the flesh of cattle that have died from splenic fever, but the poison is destroyed by proper cooking. Flies and other insects have also been supposed to be instrumental in carrying it to human beings. The disease has further been attributed to the consumption of milk or butter containing the poison. The wool and hair of animals that have died from splenic fever are now generally recognized as important sources of infection. Anthrax may thus arise either by direct inoculation, or by inhaling the dust containing the poison. It is also believed that it may be communicated by means of drinking-water; or by wool-waste and bone-dust used as manure.

Considering its mode of origin, it will be readily understood that malignant pustule is met with almost invariably in those who follow certain occupations, which bring them into contact with the carcasses or skins of infected animals, or with their wool or hair. Thus it occurs among butchers, slaughterers, tanners, wharf-labourers who handle foreign hides and fleeces, wool-packers and sorters, furriers, horsehair cleaners, workers in felt manufactories, &c. The patients are almost always males, and chiefly young adults, but the complaint has been met with at eleven years of age (Davies-Colley).

Malignant pustule has been proved to be due to a specific organism, originally discovered by Davaine, and named by him *bacteridium*, but now generally known as the *bacillus anthracis*. This is found in the blood, in the fluid of serous cavities, and in the tissues. It is either diffused, or aggregated into masses in the vessels and lymphatics. Bacilli are present in the eruption and in the surrounding skin. Usually they appear in the form of minute rods or filaments, motionless, apparently homogeneous, but the longer ones are

really made up of segments. They range from  $\frac{1}{2500}$  inch upwards, and are either straight, curved, bent, or looped. They may be cultivated, producing spores, which are highly tenacious of life, and can reproduce the bacillus, and thus originate the disease. By cultivation also filaments of great length may be developed, forming loops, curves, or spirals, and becoming matted together inextricably. As previously

FIG. 11.



Spores, rods, and cultivated filaments of *bacillus anthracis*.  $\times 500$ .

mentioned, these bacilli have by cultivation been so modified by Pasteur, as to produce a mitigated and benignant form of splenic fever in animals. His researches, moreover, show that they may be cultivated and increased in the earth around buried carcases, and are thence supposed to be carried to the surface by earthworms, thus propagating the disease.

**ANATOMICAL CHARACTERS.**—The following is a general summary of the *post-mortem* appearances likely to be met with in fatal cases of anthrax:

—Early *rigor mortis*, of short duration; venous congestion and hypo-

stasis; superficial petechiæ; œdema of the face; rapid decomposition, with subcutaneous emphysema of the neck and face; a tarry condition, and sometimes a peculiar viscosity of the blood, which is indisposed to coagulate; ecchymoses or large extravasations in internal parts; congestion of, or hæmorrhages in connection with organs. The spleen is usually much softened, and the liver may be also somewhat soft. Œdema and partial collapse of the lungs are common. When there is an external malignant pustule, hæmorrhagic patches are found radiating into the surrounding tissues, which are extensively infiltrated with a blood-stained semi-gelatinous fluid.

Certain special forms of anthrax are described, with corresponding anatomical characters. In the *pulmonary* form the thoracic structures and those of the neck are mainly or solely implicated—cellular tissue, lymphatic glands, air-tubes, lungs, and pleuræ—indicated by much congestion, swelling of glands, serous effusion, gelatinous œdema, and hæmorrhages. Professor Greenfield thinks that the lesions frequently present in the larger bronchi correspond with the external malignant pustule, and that the virus, having gained an entrance by local infection of the mucous membrane, is conveyed to the bronchial glands, and thence into the blood.

In another form—*gastro-intestinal*—the peritoneum, stomach, and intestines are mainly involved, and it is said that pustules and carbuncles have been found in the intestines, similar to those on the skin.

**SYMPTOMS.**—The symptoms of anthrax present much variety, but a primary division of cases may be made into (1) those in which there are specific external manifestations; and (2) those in which no such manifestations occur.

1. In the former class, or those of true *malignant pustule* or *carbuncle*, the external local lesion is first perceived. This usually results from direct inoculation, but it is said that it may arise from general infection. Almost always some exposed part is affected, such as the lip, cheek, eyelid, or some other part of the face, the neck, or the hand or

arm. The malignant pustule appears at the seat of inoculation in a few hours, or within two or three days. At first slight redness is observed here, like that produced by the bite of a gnat; then a small papule forms, which vesicates at the top; the vesicle bursts, discharging a watery fluid, either clear or turbid, and often blood-stained. A deep-red surface remains, which dries up into a central dark-brown or black eschar or slough, on an angry and hard base. The tissues around rapidly swell, and become œdematous, or hard and brawny. The eschar extends; and around it appears an erysipelatous livid-red areola, upon which usually a circle of secondary vesicles forms. The absorbent vessels and glands in the neighbourhood become inflamed, the glands being often much enlarged. When the face is affected, the swelling of the parts may be very great, extending also to the neck; and if the lip is involved, saliva escapes in abundance, and the breath is extremely foetid. The patient often feels itching, stinging, or burning sensations, causing him to scratch the part. Should recovery take place, the eschar separates, or more or less extensive sloughing ensues, and the ulcerated surface left heals by granulation. Numerous carbuncles may develop in cases of malignant pustule from general infection, but these seem to be merely of the ordinary kind. In exceptional cases *malignant anthrax œdema* is met with, without an actual pustule, especially affecting the eyelids.

The general symptoms accompanying the local phenomena are usually those of fever, and the temperature may be very high. Such symptoms may, however, be absent or slight, and even in severe cases there may be little or no constitutional disturbance. In cases tending towards a fatal issue symptoms of blood-poisoning arise, namely, great prostration and exhaustion, mental depression, cold clammy sweats, a very weak and rapid pulse, hurried respiration, sometimes diarrhœa, low delirium and other nervous phenomena, ending in coma. Not uncommonly, however, the mind is clear to the last, but a cyanotic condition supervenes before the fatal issue.

2. In the cases which have no external manifestation—*internal anthrax*, the symptoms are by no means always well-defined, and they are very variable. There may be premonitory symptoms indicating nervous disturbance, before those of an acute character appear, but they are often absent, the onset being sudden, with vomiting, rigors, headache, or other symptoms. The chief symptoms are marked general prostration, great mental depression and anxiety, hurried and laboured breathing, coldness of the extremities, and delirium and other nervous symptoms, though in some cases the mental faculties are clear to the last. Usually the temperature is not high, and the external surface may be cold; but sometimes there is a tendency to hyperpyrexia. Symptoms of collapse often precede a speedily fatal termination, which results from the general disease—*anthracæmia*. In some cases pulmonary lesions are prominent, as revealed by difficult and laboured breathing, and signs of cyanosis, these phenomena being out of proportion to the physical signs. Cough is rarely a conspicuous symptom. In other instances symptoms connected with the alimentary canal are marked, such as dysphagia, sometimes hæmorrhage from the mouth and throat, vomiting, abdominal uneasiness or pain, and diarrhœa, the stools being sometimes bloody. Sore-throat and swelling of the glands and other tissues of the neck are observed in some cases.



**DURATION AND TERMINATIONS.**—In fatal cases of malignant pustule death may take place in a few hours, or not for five or six days. The duration varies somewhat in the different forms. Although if the disease is left to itself the termination is almost always fatal, many cases of the external form have recovered under suitable treatment.

**PROGNOSIS.**—This is necessarily very grave, but if energetic local treatment can be carried out at an early period, the prognosis is much more hopeful. Cases of a mild character also occur sometimes, in which recovery takes place.

**TREATMENT.**—In *external malignant pustule* local treatment at as early a period as possible is of essential importance, and even if the changes have advanced considerably, it may be of great service. The local measures are free excision or incision; and efficient cauterization, which is best carried out by means of strong carbolic acid, nitric acid, or the actual cautery. Afterwards carbolic dressings must be used. General treatment also demands particular attention, and this alone can be carried out when there is no external manifestation of the disease. The patient should be kept in a well-ventilated room, and have abundant nourishment, with more or less alcoholic stimulants. Quinine, tincture of iron, and mineral acids may be given internally; and carbolic acid or other *antiseptics* might also be administered with advantage. Diffusible stimulants are frequently indicated, such as ammonia and ether. Symptoms must be treated as they arise. In cases where the respiratory organs are specially involved, it has been recommended to use inhalations of carbolic acid; and should there be abundant pleuritic effusion, the fluid ought to be removed.

The *prevention* of malignant pustule is of great importance in relation to those who, on account of their occupation, are exposed to infection. This, however, can only be efficiently carried out by the destruction of all infected carcasses and hides; and the thorough disinfection of all materials, especially those coming from foreign parts, which may be the means of propagating the disease. It has been suggested that the consumption of a large quantity of animal food may diminish the danger of those exposed to contagion.

---

## CHAPTER XXII.

### HYDROPHOBIA—RABIES.

**ÆTIOLOGY AND PATHOLOGY.**—Hydrophobia is unquestionably a specific contagious disease, resulting from the action of a *specific poison*, supposed to be of the nature of an organism, which in the human being is almost invariably introduced into the system through the bite of a dog suffering from rabies, inflicted upon some exposed part of the skin, but is in rare instances transmitted from other allied animals, as the cat, wolf, or fox. Only a small proportion, however, of persons bitten by dogs actually suffering from rabies take the disease. Pasteur has recently been able to prevent or mitigate the disease in dogs by inoculating the poison attenuated by cultivation. It may be conveyed in consequence of a dog merely licking an exposed surface; and it has even been supposed that

the poison may be absorbed by the entire skin, where this is thin. The contagium is contained mainly in the saliva or buccal secretion, and at any rate it is only through the agency of this fluid that it can enter the human system. It has been affirmed that the disease can be transmitted from man to man. Mental anxiety has been presumed to predispose to hydrophobia, and, indeed, some go so far as to maintain that this complaint is merely the result of mental terror, produced in a person who has been bitten by a mad dog, and that it is entirely independent of contagion; this, however, is a mere assumption, against which there is the strongest evidence.

It is supposed that the poison of rabies undergoes a kind of fermentation, and becomes increased both at the wound and in the system. It then acts upon the blood, and subsequently is presumed to affect the eighth pair of nerves and the nerve-centres, especially the medulla oblongata, which will account for the phenomena observed.

**ANATOMICAL CHARACTERS.**—The morbid changes which are associated with rabies in the dog, and hydrophobia in man, have of late years received considerable attention, and have been investigated by several eminent pathologists. The most striking alterations are observed in connection with the nerve-centres, and they are mainly revealed by microscopic examination. The more obvious appearances which have been described are marked congestion of the meninges of the brain and spinal cord; excess of serum in the ventricles; and effusion of blood or serum about the upper part of the cord, with small extravasations into its substance.

The microscopic changes in the nerve-centres have been studied by Dr. Gowers, and it will suffice to give here a summary of his observations, which correspond in the main with those of other workers in the same field of pathology. In specimens removed from the human subject he noticed more or less of the following alterations. There was great distension of the minute vessels of the grey matter of the cord and medulla, most marked in the neighbourhood of the grey nuclei in the floor of the fourth ventricle. Many of the medium-sized vessels, especially the veins, were distended with blood, and contained ante-mortem clots, some portions of which had undergone a granular change, while in others a peculiar change was observed, the outline of the corpuscles having disappeared, and given place to a spongy structure, which seemed formed by the swelling and fusion of the corpuscles. Sometimes these clots by their position and aspect resembled emboli, but Dr. Gowers believes that embolism plays no part in the changes observed. The medium and larger veins of the medulla presented aggregations of small cells within the peri-vascular sheaths, sometimes in a single layer, sometimes in many layers, and so densely packed as to compress the vessel they surrounded. In exceptional cases the cells had extended beyond the peri-vascular sheaths, and had infiltrated the adjacent tissue. Here and there a diffuse local infiltration of similar cells into a small area of the tissue could be seen, and in the centre of the infiltrated area a small dilated vein, without a lymphatic sheath. In other places spots of larger size were thus infiltrated, especially in the neighbourhood of the hypoglossal nucleus. These areas corresponded to the condition termed "miliary abscess." Similar small cells were scattered through the tissue much more abundantly than in health. They are considered by Dr. Gowers to be of the nature of migrated leucocytes. In every case examined many vessels were observed to be surrounded by spaces,

either empty or containing more or less granular debris. The nerve-cells presented comparatively little change. Many had a granular aspect; while others had a somewhat swollen appearance. Adjacent to, or around many cells were spaces, in some cases apparently empty, in others containing granules.

With regard to the position of these lesions, they were found to be comparatively slight in the spinal cord, but were much more marked above the decussation of the pyramids, and still more above the point of the calamus scriptorius. They were most intense in the neighbourhood of the hypoglossal, pneumogastric, and glosso-pharyngeal nuclei, and slighter in the auditory, facial, and fifth nuclei. The higher part of the pons was much less affected. The region most affected corresponds to what is regarded as the "respiratory centre" of the medulla.

The morbid appearances observed in the dog were similar to those in man, only more intense, amounting to a local disseminated myelitis. Changes in the convolutions have also been described by Benedikt in dogs which had died from rabies.

As to the relation of the vascular changes to the disease, it is doubtful whether they are primary or secondary to irritation of the nerve-elements. Dr. Gowers believes that "the lesions are *characteristic* of hydrophobia, not in themselves, but in their character and distribution, being intense about the respiratory centre, taken in conjunction with the fact that they are due to an acute disease."

Among other *post-mortem* appearances which have been described in cases of hydrophobia are congestion of dependent parts, often very marked; increased vascularity of the fauces, with follicular enlargement, and occasionally a deposit of lymph; and acute desquamative nephritis.

**SYMPTOMS.**—A very indefinite *period of incubation* intervenes between the introduction of the poison, and the development of the symptoms of hydrophobia. About forty days is said to be the average, but it may vary from fifteen days to many months, or, it is said, even years. In some cases unusual objective appearances or subjective sensations are developed in connection with the cicatrix of the bite, before the actual symptoms of hydrophobia break out, such as redness, itching, numbness, or curious and ill-defined feelings.

When the disease begins to declare itself, the patient feels uncomfortable, low-spirited, despondent, and restless; has an undefined feeling of anxiety or dread; and complains of giddiness, or of alternate chills and heats. Then follows a sense of oppression in the chest, with involuntary deep sighing inspirations from time to time; or a sudden catch in the breathing may first occur, attended with severe pains in the epigastrium, due to spasm of the diaphragm. The subsequent characteristic symptoms are grouped by Mr. Erichsen as:—1. Spasmodic affection of the muscles of deglutition and respiration. 2. Extreme sensibility of the surface and of the special senses. 3. Excessive mental terror and agitation. The nature of the malady is generally revealed to the patient by a fit of choking brought on by an attempt to drink, and by finding that swallowing has become difficult or impossible. This condition becomes rapidly worse and worse, each attempt at drinking bringing on a spasm of the muscles of deglutition and respiration, which is attended with a feeling of intense oppression and impending suffocation, causing great distress. Not unfrequently solids can be swallowed at first without producing any disturbance. Soon the sight or sound of any liquid, or anything that



even suggests the idea of drinking, brings on the spasmodic attacks, while the patient spits out the viscid secretion which forms in abundance in the mouth as fast as it is produced, so as not to be tempted to swallow it. The skin and special senses also become extremely sensitive, so that the least touch or a sudden sound or light will bring on the spasms, which ultimately extend to other muscles, assuming more or less the characters of general convulsions. The patient is in a state of great terror, anxiety, and depression, combined with restlessness. Often fits of furious mania subsequently occur, in which the patient is extremely dangerous and utters strange sounds, which has given rise to the idea of barking being a symptom of hydrophobia. In the intervals the intellect is generally quite clear. Sometimes there are curious persistent delusions. As the case progresses towards a fatal termination, which may be said to be the invariable result, the special symptoms diminish, or may even disappear altogether, and the patient gradually sinks from exhaustion and collapse. Albuminuria has sometimes been observed. In rare instances death occurs suddenly, from suffocation during a fit of spasm. The duration of hydrophobia is from three to five or six days.

TREATMENT.—The most important indication is to *prevent* hydrophobia, by immediately cauterizing the part bitten by means of nitrate of silver, the hot iron, or potassa fusa; or by complete excision. Other modes of preventive treatment are quite useless. The patient's mind should be calmed as much as possible, and he should be prevented from brooding over his danger. There is no remedy at present known which is of any real service for the cure of hydrophobia, once the disease has become developed. Relief might be afforded by subcutaneous injection of morphia, atropine, or curare; by the application of the ice-bag to the spine; or by the inhalation of chloroform. It has been affirmed that the disease has been cured by the wet-pack and cold baths, but more positive evidence is needed before such a statement can be regarded as established.

---

## CHAPTER XXIII.

### DENGUE—DANDY FEVER—BREAKBONE FEVER.

ÆTIOLOGY.—Dengue is a peculiar epidemic fever which attacks a great number of people in rapid succession. It is not met with in this country, but prevails in America, the East and West Indies, and other parts of the world. It is not known what the actual cause of the complaint is; some believe it to be contagious, but the evidence on this point is insufficient and unsatisfactory.

SYMPTOMS.—After an *incubation-period* of from twenty-four hours to ten days, the invasion is usually somewhat sudden, the symptoms being chilliness, a sense of debility and general uneasiness, with violent pains in the muscles of the limbs and back, as well as in a variable number of joints, which are somewhat swollen. There is also headache, and the alimentary canal is often disturbed, but the tongue remains clean. At first the symptoms very much resemble those of acute rheumatism.

In a short time fever sets in, and sometimes a rash or papular eruption appears. The lymphatic glands and testicles may be painful and enlarged. In from twelve hours to three or four days the symptoms subside, but debility remains, with some painful sensations. After an interval of two, three, or four days the fever and pains return, or severe headache sets in. The tongue becomes much furred, and there is considerable epigastric uneasiness, accompanied with nausea. An eruption appears on the fifth, sixth, or seventh day, especially on the upper part of the body, which presents very variable characters, either resembling that of scarlatina or measles, or being papular, vesicular, pustular, furunculous, erysipelatous, or petechial. As it disappears, scaly desquamation sometimes takes place. The eruption is attended with itching and tingling.

The symptoms present very different degrees of intensity, and they may assume an asthenic character. Almost all cases of dengue recover, the average duration being about eight days, but stiffness and soreness of various parts, with debility, may remain for some time, and sometimes several *relapses* occur.

TREATMENT.—*Emetics* are recommended at the outset of an attack of dengue, with free purgation. The patient must remain in bed. A *saline diaphoretic* mixture may be given; and opium is to be freely administered for the relief of pain. The surface of the skin may be sponged in order to subdue pyrexia; and cold applied to the head for the relief of headache. The *diet* must be regulated, and if there are any signs of depression, stimulants and nourishing food should be given. Quinine and mineral acids are recommended during the remission, as well as during the period of convalescence.

## CHAPTER XXIV.

### PESTIS—PLAGUE.

ÆTIOLOGY.—This disease is of a specific nature, and generally prevails as an epidemic, but may be sporadic. Formerly it was met with in Europe, but at the present time its chief seats are Egypt, Syria, Asia Minor, and the coast of Barbary. It is undoubtedly contagious, and can be conveyed in various ways. The chief *predisposing causes* are overcrowding and bad ventilation; want of cleanliness, with accumulation of filth; insufficient and unwholesome food; debility from any cause; a warm and moist condition of the air; and residence on marshy soil, or in the neighbourhood of certain rivers. Epidemics often follow famine, and are generally preceded by a sickly, oppressive, warm, and moist season.

ANATOMICAL CHARACTERS.—The blood is dark, and remains fluid or coagulates imperfectly, while it rapidly putrefies. All the organs are greatly congested and softened, especially the spleen; also the mucous and serous membranes, which may present petechiæ and ecchymoses. The serous cavities contain more or less effusion. The absorbent

glands generally are swollen, dark, softened, or disintegrated. Buboës and carbuncles are usually present.

**SYMPTOMS.**—Plague is characterized by fever, generally of a low type, with the local development of buboës, carbuncles, and petechiæ. The *period of incubation* is very short, and symptoms may set in almost immediately after exposure to infection. After inoculation, the distinctive glandular swellings are developed in four days. The *invasion* is sudden in most cases, and the severity of the constitutional symptoms varies from a slight fever to one of the most virulent type. The early symptoms are rigors, restlessness, a feeling of debility and languor, headache and giddiness, nausea or vomiting, and precordial oppression or uneasiness. The expression is heavy and stupid, and the eyes look muddy or suffused. Soon the temperature rises, while prostration increases, with a tendency to syncope. The pulse is frequent, but weak or irregular. The tongue is thickly furred, and tends to become dry and brown or black, with sordes on the teeth. The other prominent symptoms are vomiting, sometimes of black matters; great thirst; diarrhœa with offensive stools; hurried respiration; and very foul breath. The urine is much diminished in quantity, and may contain blood. Hæmorrhages from mucous membranes are not uncommon. Nervous symptoms are generally present, such as delirium, stupor, coma, or convulsions. Death may take place before the appearance of any local signs.

Buboës are formed chiefly in connection with the glands of the groin, but are also seen in the axilla, and about the angles of the jaws. They appear at different periods, being preceded by darting pains. Generally they form abscesses, which discharge and heal slowly, leaving permanent scars. Carbuncles are observed mostly on the limbs, but may affect any part of the body. They vary in number and size, and are liable to end in gangrene, sometimes thus causing great destruction of tissues. Petechiæ, vibices, and livid patches are seen only in bad cases, and there may also be extravasations of blood into the subcutaneous tissue in such cases.

**PROGNOSIS** is very grave.

**TREATMENT.**—Attention to all *hygienic measures* is essential in the treatment of plague. The bowels should be freely opened. An *emetic* at the outset is recommended. Nutritious food, stimulants, mineral acids, and *tonics* are the remedies which afford the best results. Probably those who advocate the use of *antiseptics* would give them largely in this disease. Cold affusion or sponging may also be employed. Poultrices are indicated for the buboës and carbuncles, followed by antiseptic dressings when they are discharging.

---



## CHAPTER XXV.

## YELLOW FEVER.

**ÆTIOLOGY.**—There is much discussion amongst those who have had opportunities for the observation of cases of yellow fever, as to whether it is *malarial* in its origin, or of the nature of a *specific contagious disease*. Most authorities maintain the latter view, holding that true yellow fever is of the continued type, but that it may be simulated by malarial remittent fevers. There appears to be strong evidence proving that the disease can be conveyed by infection, and it has thus originated in seaport towns in this and other countries, owing to the arrival of vessels with cases of yellow fever on board. Fomites may be the means of propagating it.

The late Dr. Hamilton, in an excellent Thesis on Yellow Fever, founded on much personal observation, sums up his conclusions as follows:—

“1. That yellow fever is the highest development of a group of diseases which depend on some unknown cause, but which appears to be in some way connected with, or dependent on, organic decomposition.

2. That the various individual diseases of this group may change their type and pass one into the other, according to the intensity of the cause, or the more or less favourable conditions under which such cause acts.

3. That the general laws of zymotic diseases, as observed in this country, hold good for this group also.

4. That the same causes which augment or develop contagious properties in the zymotic diseases of this climate, will develop contagious properties in yellow fever.

5. And that consequently yellow fever may under such circumstances become contagious and spread.”

In certain regions yellow fever is endemic, and it occurs also in severe epidemics. Its principal seats are the West Indian Islands, the seaports of North and South America, the South Coast of Spain, Mexico, and the West Coast of Africa. It requires a temperature of at least 72° F.; and is rarely met with at an altitude of more than from 2,000 to 3,000 feet above the level of the sea.

Among the chief external *predisposing causes* are mentioned:—long-continued high temperature; a swampy or low-lying and crowded district; filthiness, and other anti-hygienic conditions. The disease is more liable to attack children, males, the white races, and those who have recently come into an infected district. Intemperance and other excesses, fatigue, and exposure to night air and dews also increase the liability to the complaint; while individual predisposition seems to exist in some instances.

**ANATOMICAL CHARACTERS.**—The body may or may not be emaciated. The skin is of a deep yellow colour, as well as much congested in depen-

dent parts, and in those distant from the centre of circulation. The tissues are generally soft and flabby. More or less congestion of organs is observed, sometimes with extravasations of blood and effusions into serous cavities. Softening of the heart, with molecular degeneration of its fibres, is usually met with; and the blood coagulates imperfectly, though soft clots are generally found in the cardiac cavities. The stomach is the most frequent seat of morbid changes. It often contains more or less "black vomit," or blood undergoing alterations. Sometimes a black or bloody mucus sticks to the lining membrane. Signs of congestion or inflammation are present in most cases. An inflammatory condition has been described in connection with the membranes of the cord; and in the sympathetic system.

**SYMPTOMS.**—The prominent symptoms of yellow fever vary in different epidemics, and cases exhibit all grades of intensity, from a very mild form of the disease to one of the most malignant type. Usually three stages may be recognized, following a *period of incubation*, the duration of which is usually from two to four days, but it is said that it may range from one to fifteen days.

1. **Invasion-stage.**—The attack may be preceded by premonitory symptoms, or may come on quite suddenly. Chills generally occur at the outset, but are not always observed in tropical climates. These alternate with a sense of heat, and soon there is marked pyrexia, its degree being in proportion to the previous chills, the temperature presenting a morning remission. The pulse is frequent, and in most cases full and strong. The face is flushed, the eyes are red and suffused, and the expression is anxious and distressed. The skin feels hot, dry, and harsh. The tongue is covered with a white fur, moist, red at the tip and edges, with enlarged papillæ. Sore-throat may be complained of; and there is a constant desire for cool drinks or ice. Gastric symptoms may be present from the first, but as a rule they only become prominent in from twelve to twenty-four hours. These symptoms include a sense of oppression, uneasiness, weight, or burning pain in the epigastrium, with considerable tenderness; nausea, violent vomiting, and retching, the vomited matters being of a bilious character, or containing streaks of blood or chocolate-coloured flocculi. After a while the stomach rejects everything without any effort. There is usually obstinate constipation, with unhealthy stools, which are deficient in bile; and there may be much flatulence. The urine is deficient in quantity, of dark colour, and generally contains albumen.

Nervous symptoms are most distressing in the majority of cases. Severe frontal headache is complained of from the first, with shooting pains in the temples and eyes. One of the earliest and most prominent symptoms in most instances, however, is pain in the lumbar region and limbs, which often becomes so intense as to elicit screams and groans, and to make the patient writhe in agony. As this stage advances the patient becomes very restless, the mind is confused, or wild and violent delirium may set in, attended with hallucinations. Occasionally there is more or less stupor.

The invasion-stage lasts from a few hours to two or three days usually, but it may extend to four or five days. It is longer in the milder cases.

2. **Stage of Remission.**—A marked improvement is observed at the close of the first stage, which in most cases is only temporary, but sometimes is permanent, convalescence setting in, preceded by critical dis-

charges. The symptoms subside more or less completely, the patient feeling comparatively comfortable and often hopeful, occasionally appearing to be quite well. But at this time there are frequently some unfavourable signs, namely, more marked tenderness in the epigastrium; a yellowish tinge of the skin and urine; a slow pulse; and sometimes heaviness or stupor. The duration of this remission is usually but a few hours, but may be prolonged to twenty-four hours.

**3. Stage of Collapse or Secondary Fever.**—In most cases signs of collapse appear, with great prostration and debility. The skin generally assumes a yellow, orange, or bronzed hue, but not invariably. This spreads from the forehead downwards, and is dependent upon the colouring matter of the blood. The circulation is impeded, the pulse becoming very rapid, weak, and irregular, while capillary congestion or stagnation is observed in dependent and distant parts, sometimes accompanied with petechiæ and vibices; at the same time the heart may be beating violently. In bad cases hæmorrhages are common, especially from the mucous surfaces. The tongue tends to become dry, brown, or black; or it is smooth, red, and fissured; sordes may also form on the lips and teeth. The gastric symptoms return and become very intense. The so-called *black vomit* sets in by degrees, the black colour being probably due to altered blood. It is often preceded by *white vomit*. Black vomit is not a constant symptom, and the exact characters of the vomited matters vary, pure blood being sometimes discharged. Similar materials may pass away in the stools. The urine is often more or less deficient, and contains albumen; it is sometimes entirely suppressed or retained. The patient frequently lies in a state of apathy and gloomy indifference. Ultimately collapse becomes extreme, with a cold, clammy skin; slow, sighing respiration; and hiccup. Consciousness may be retained to the last; or low delirium or coma may set in, with convulsions at the close.

In some cases the symptoms of this stage of yellow fever are those of more or less intense secondary fever, instead of collapse. This course of events may terminate in convalescence; or the fever assumes a typhoid type, ending fatally.

**VARIETIES.**—As already stated, great differences are observed in the intensity of the symptoms of yellow fever; and also in the nature of the phenomena which are most prominent in different cases. Some patients are prostrated at once, and die very speedily. The named varieties are:—1. **Algid.** 2. **Sthenic.** 3. **Hæmorrhagic.** 4. **Petechial.** 5. **Typhous.** These several terms indicate the prominent characters which are peculiar to each variety.

**PROGNOSIS.**—Yellow fever is always a terrible disease, but the mortality varies much in different epidemics. Death usually takes place from the fourth to the sixth day, but may be delayed to the ninth or eleventh day, or even to a much later period than this. It has been observed that many apparently hopeless cases recover, while others which seem to be mild prove speedily fatal; hence the prognosis is very uncertain. A concise list of favourable and unfavourable signs is given by Dr. Macdonald in Reynolds' *System of Medicine*, Vol. i., p. 492.

**TREATMENT.**—Attention to all *hygienic measures* and rules of health is of prime importance in the treatment of yellow fever. At the outset hot drinks and warm foot-baths have been recommended, with *emetics* and *purgatives*. Large doses of calomel or of quinine used to be given, but they have been proved to be injurious.



It is important to excite free action of the excreting organs as soon as possible. Copious enemata containing turpentine are serviceable. Saline drinks may be given abundantly. The skin should be sponged; or wet-packing may be resorted to if the patient is very hot. In an epidemic of yellow fever which occurred in America, it is affirmed that a patient was restored when apparently in a hopeless condition, by being placed in a net under which an india-rubber sheet was hung, and constantly syringed with iced water. Liquid food should be given in small quantities, with cool drinks, and plenty of ice. Alcoholic stimulants well diluted are also valuable. Champagne is most beneficial, if it can be obtained.

Various symptoms require attention in yellow fever, but especially vomiting. For its relief lime-water and milk, hydrocyanic acid, creosote, chlorodyne, and chloroform have been found most useful. Great care must be exercised in the administration of opium or morphia, particularly if there is any tendency to suppression of urine. Chlorodyne is suggested as a substitute, in order to procure sleep and to relieve pain; hot applications or mustard poultices being also applied externally over painful parts. Hæmorrhages, collapse, and typhoid symptoms must be treated by the ordinary remedies. During convalescence quinine may be given, if recovery should take place.

---

## CHAPTER XXVI.

### CEREBRO-SPINAL FEVER—EPIDEMIC CEREBRO-SPINAL MENINGITIS.

**ÆTIOLOGY.**—This complaint prevails as an epidemic, and presents the characters of an acute specific disease, but its *exciting cause* is by no means definitely made out, although micrococci have been described in the exudations. There is no reliable evidence that cerebro-spinal fever is at all contagious. It has been attributed to the action of a malarial poison; to excessive fatigue; to the use of unwholesome food, especially diseased grain; or to exposure to cold. Among the chief *predisposing causes* are early age, especially from fifteen to thirty, the complaint being uncommon after thirty-five, and very rare after forty, but not infrequent in children; the male sex markedly; and a cold season, most cases occurring during winter or early spring. Unfavourable hygienic conditions do not seem to have much influence. Cerebro-spinal fever is met with as an epidemic principally where young people are collected together, and it is particularly common among young recruits in barracks.

**ANATOMICAL CHARACTERS.**—The most important morbid changes observed in cases of cerebro-spinal fever are those indicative of inflammation of the membranes of the brain and spinal cord. There is hyperæmia of the scalp and of the structures within the cranium, the sinuses of the dura mater containing much dark fluid or softly-coagulated blood. There may be small hæmorrhagic effusions in connection

with the dura mater. The sub-arachnoid space contains excess of serum, but the most striking appearance is due to the presence of more or less exudation in this space, both over the convexity and at the base of the brain, though it is much more abundant in the latter situation, the cranial nerves being often imbedded in it. The amount and characters of this material vary with the duration of the disease; in cases of very short duration it is generally in small quantity, whitish, and soft; later on it becomes more abundant, yellowish or greenish, and somewhat purulent-looking; while in prolonged cases it is again whiter and firmer, there being also more serum under these circumstances. The brain-substance is unduly vascular, and may be softened, especially in the neighbourhood of the ventricles, which contain in many cases a small quantity of purulent-looking fluid, or occasionally much serum.

The membranes of the cord present similar changes, there being a purulent fluid under the arachnoid, with exudation, which is almost entirely confined to the posterior surface.

Rigor mortis is well-marked. Post-mortem congestion rapidly sets in; and purpuric patches are usually visible. The blood is dark and tarry-looking, and the muscles are of a deep colour. The spleen, liver, and lungs are often much congested, and there may be signs of inflammatory complications. Purulent infiltration of the eyeball has been occasionally met with; and also effusion into the joints.

**SYMPTOMS.**—In the great majority of cases cerebro-spinal fever is not ushered in by any premonitory symptoms, and the *invasion* is quite sudden, being indicated by a rigor or chill; faintness; intense headache, causing the patient to cry out, and being usually more or less general, but sometimes localized, especially at the back of the head; vertigo; frequently epigastric pain and cerebral vomiting, the vomited matters being generally of a bilious character; great restlessness; and pyrexia. The pupils are contracted. In a day or two the pain extends to the back of the neck, and then down the spine, being increased in this locality by movement and pressure. The head is drawn back, partly voluntarily in order to relieve pain, partly from spasm of the muscles. In three or four days distinct tetanic spasms set in, there being often well-marked opisthotonos, and occasionally trismus, risus sardonicus, or strabismus. The respiratory muscles may become involved, causing serious interference with breathing. The skin generally becomes extremely sensitive, and severe pains are felt in the limbs, these being aggravated or sometimes only produced by movements of the spine. At first the mind is clear, but soon mental confusion is observed, followed by muttering delirium, and afterwards by stupor, which in fatal cases ends in deep coma. In rare instances epileptiform convulsions occur; or there may be hemiplegia, paraplegia, or signs of paralysis of some of the cranial nerves. Amaurosis is occasionally noticed; and deafness is not uncommon.

Early in the course of the disease herpes usually appears, chiefly about the lips and face, but it may be observed on the limbs or trunk. Other forms of eruption are sometimes seen, and the body becomes more or less thickly scattered over with purpuric spots in bad cases, in some instances considerable patches being involved, which may become quite black or even gangrenous; in such cases mucous hæmorrhages are also liable to occur. The degree of pyrexia varies much in different cases, but the temperature usually ranges from  $100^{\circ}$  to  $103^{\circ}$ , in some instances rising to  $105^{\circ}$  or higher. It has no regular course, and often

presents sudden variations, but there is generally a slight evening exacerbation. The pulse rises to 100 or 120, but is liable to considerable changes in frequency; it is sharp, weak, and wanting in tone. The respirations are hurried. The bowels are constipated, and the abdomen is retracted. The urine may contain a large quantity of albumen in severe cases; and when stupor sets in, it is retained or passed involuntarily.

A favourable termination of the complaint is indicated by a gradual subsidence of the nervous phenomena; restoration of the mental faculties; and a steady fall in temperature. Convalescence is very slow, and headache usually continues for some time. There may only be partial restoration, the mind being permanently impaired, or some form of paralysis remaining; or death may take place after many weeks, from general marasmus.

Cases of cerebro-spinal fever present much variety in their severity, and in their combinations of symptoms, and different writers have arranged them into corresponding groups or *varieties*.

COMPLICATIONS AND SEQUELÆ.—These are not uncommon in cerebro-spinal meningitis, the chief being inflammatory affections of the eyeball, especially the right one, which may end in suppuration and total destruction of its tissues; inflammation of joints, ending in accumulation of pus; bronchitis, pleurisy, or pneumonia; pericarditis; and parotitis.

PROGNOSIS.—Cerebro-spinal fever is a grave malady, but the mortality varies in different epidemics from 20 or 30 to 80 per cent., the average being about 60 per cent. The early appearance of purpuric spots, and the occurrence of hæmorrhages, are very unfavourable signs. Death is most to be feared during the first few days, but it may take place after many weeks. The mortality is highest at the commencement of an epidemic; and amongst young children, or persons over 30 years of age.

TREATMENT.—If there is much depression at the outset, it is desirable to give small quantities of *stimulants*, and to apply heat externally. Subsequently the indications are to promote absorption of the exudation; to relieve the pain and muscular spasms; to support the strength of the patient; and to treat symptoms and complications as they arise. Mercury and iodide of potassium have been given with the view of aiding absorption, but the former drug seems to be quite inadmissible in the treatment of cases of cerebro-spinal fever. The application of a few leeches to the temples or behind the ears often relieves the headache. Ice should be applied constantly to the head and along the spine. In prolonged cases it has been recommended to blister the nape of the neck and the spinal region. The most reliable remedies for relieving the symptoms are opium internally, or hypodermic injection of morphia; hydrate of chloral; bromide of potassium in full doses; and belladonna. The patient must have nutritious and sustaining food throughout; and considerable quantities of *alcoholic stimulants* are frequently required. Enemata are often of much service for their administration, as well as for the purpose of acting upon the bowels. It is requisite to pay due attention to hygienic conditions. During convalescence *tonics* and good diet are necessary.

---



## CHAPTER XXVII.

## MALARIAL OR PALUDAL FEVERS.

## I. ON MALARIA OR MARSH-MIASM.

THERE are certain affections which have been almost universally recognized as being the result of the action of a *malarial* or *telluric* poison upon the system. Amongst these the most striking are fevers of an *intermittent* or *remittent* type, before considering which it is therefore requisite to indicate the main facts relating to malaria. Of the existence of such a poison there can be no doubt, though this has been denied by a few observers, some of whom have attributed the effects noticed to general chilling of the body; others to some electrical condition of the atmosphere.

1. **Origin and propagation.**—The malarial poison is ordinarily an emanation from soils more or less rich in organic matter, and which are not devoted to the maintenance of healthy vegetation. The essential conditions for the production of this poison are decomposition of vegetable organic matter; a certain temperature; and a certain degree of moisture. Without the first of these it cannot possibly originate. Very rarely are malarial diseases generated under a temperature of 60° F., and the heat must be of some duration. As this rises, they become more prevalent and more severe, and hence they are very virulent in certain tropical climates. There must not be too much moisture, else the poison is absorbed by it; while it is not formed if the atmosphere is dry.

The necessary conditions, as regards vegetable decomposition and moisture, are met with under the following circumstances:—1. In marshes and swamps, unless the soil is peaty or constantly overflowed with water, conditions which materially interfere with the development of malaria. 2. Where there is much vegetable matter in the soils of valleys and ravines, at the bases of mountain ranges in tropical climates, in alluvial deposits, along the banks of tropical rivers, in old estuaries, the deltas of rivers, &c. 3. Where surfaces covered with much vegetation have been temporarily overflowed, so as to be left moist. 4. During the draining of lakes, ponds, &c. 5. In sandy plains containing organic matter, if there is a subsoil of clay or marl, conditions often existing simultaneously in old river courses. Also in the lower chalk-formations, with a subsoil of clay or marl. 6. In certain hard granitic or trap rocks containing organic matter, such as fungi, especially if they are disintegrating. 7. In turning up the soil in the early cultivation of land, digging canals, making railways, &c. 8. Where copious vegetation has been cleared away in dense jungles for purposes of cultivation, sufficient being left behind to decompose. It frequently happens that the first result of attempts at cultivation of a new district is the production of malarial diseases, which ultimately

disappear. 9. When tracts of land are from any cause allowed to fall out of cultivation, especially if the soil is rich in organic matters. 10. On board ship, where decomposing vegetable matter is mixed with bilge-water; or where malarial mud has in any way been accumulated.

The following conditions influence the development and propagation of malarial affections:—1. *Season*. Usually these complaints are most prevalent towards the latter part of summer and in the autumn, and many malarial districts may be visited without any danger in the winter, which cannot be approached in the warm season. They are particularly liable to occur after long-continued dry and hot weather, followed by warm rains. In climates where the summer is short, even though very hot, they are not prevalent. 2. *Water*. Abundance of water constitutes a protection against malarial affections, because it absorbs the poison. Hence they are temporarily diminished by long and heavy rains and floods. Any deep sheet of water, especially running water, affords some degree of protection, and thus the intervention of a river may prevent the poison from passing from one of its banks to the opposite one. A ship at a little distance from shore is in comparative safety. Some believe that sea-water is peculiarly protective, though it is said that the occasional admixture of salt water increases the emanations from marshes. 3. *Winds*. These frequently convey the malaria for a considerable distance along plains, and may thus be the means of originating malarial affections in places remote from a malarial district, also counteracting the good effects of the intervention of water, &c. On the other hand, a storm may drive away the poison altogether. 4. *Low districts* are more dangerous than those which are elevated, the malaria tending to cling to the earth. By the aid of ravines and hot air, however, it may be carried up mountains to a great height, it is stated even as much as 2,000 or 3,000 feet. The lower rooms of houses are more dangerous than the upper. 5. *Trees*. When in large numbers these afford decided protection, both by interfering with the propagation of the poison, and by keeping off the sun's rays from the soil; in some cases, however, they seem to be injurious. Certain trees are said to exert a specially protective influence, particularly the *Eucalyptus globulus*, but this is more than doubtful. 6. *Mountains and hills* interfere considerably with the dissemination of malaria. 7. *Time of the day*. Morning and evening dews augment the danger from malaria materially, probably from condensing the poison. It is highly dangerous to sleep in tents at night in malarial districts. 8. The *air of cities* in some way renders the poison innocuous, for though a malarial disease may be raging in the surrounding districts, it does not penetrate far into their interior. 9. *Artificial heat* destroys malaria, if sufficiently intense. 10. *Individual susceptibility* is increased by certain circumstances, namely, recent arrival in a malarial district; fatigue and exhaustion from any cause; exposure to the full heat of the sun; sudden changes in temperature, and chills of all kinds; intemperance; exposure on an empty stomach; over-feeding; mental exhaustion or nervous depression; and over-crowding. Some persons are far more susceptible than others. Young children and old persons are least subject to malarial affections; and males are said to be more predisposed than females. White races suffer more than blacks. It is stated that the use of drinking water from a malarial district may produce malarial diseases.

2. **Nature.**—Many believe that the malarial poison is a gas, resulting from vegetable decomposition. The observations and experiments which have been made within recent years appear, however, to have demonstrated in a striking manner the precise nature of the malarial poison, and to have shown that it is a vegetable organism, which has been named by its discoverers—Tommasi Crudeli and Klebs—the *bacillus malariae*. In their original experiments these observers obtained from the soil and water of the *Agro Romano*, and from the lower strata of the atmosphere, a microscopic fungus, consisting of numerous movable shining spores, of a longish oval shape. They cultivated this fungus, and introduced it beneath the skin of healthy dogs. All the animals experimented upon had typical intermittent fever; while the spleen became enlarged, and a large quantity of the characteristic fungus was found in it. Since these experiments were performed, other observers have affirmed that they have detected the bacilli in human beings, in a more advanced stage; and Crudeli states that they are always present in the blood during the period of invasion of the fever, but during the acme only spores can be seen. They have also been found in the human spleen; and in the marrow of bones in animals experimented upon. Sternberg failed to recognize the bacillus, and thinks that Klebs produced a septicæmia, not an intermittent fever.

It has been suggested that there are different kinds of paludal poison, but of this there is no proof. When animal matters are mixed with the decomposing vegetable material, the poison seems to be more virulent.

3. **Mode of entrance into the system, and the effects produced.**—The malarial poison is chiefly inhaled, and then absorbed by the pulmonary membrane; it may also be taken up by the stomach, which it often seriously disorders; and possibly by the skin. Malaria acts on the nervous system, and gives rise to fevers of an intermittent or remittent type, followed after a time by permanent organic changes, especially in the liver and spleen; while it also originates neuralgic affections. Certain other disorders are attributed to its influence, such as diarrhoea, gastric derangements, palpitation of the heart, pains in the limbs and joints, and amenorrhoea; as well as a general state of ill-health, and a peculiar malarial cachexia, with ultimate degeneration of the race. A large number of males in some malarial districts are said to be impotent. Dysentery and hepatic abscess are very prevalent in certain tropical malarial regions. Malaria imparts a peculiar periodicity to the affections which it originates, and once they have been developed they are liable to recur on subsequent occasions independently of the action of the original exciting cause, sometimes, indeed, apparently arising spontaneously. Various complaints tend to assume a periodic character in malarial districts.

4. **Prophylaxis.**—The precautions to be taken by those who are obliged to reside in malarial districts may be gathered from what has been already stated. Everything must be avoided which increases the individual tendency to malarial diseases; and every means of protection employed, so far as this is possible, in arranging a place of residence and other matters. It is a useful practice to give cinchona bark or quinine daily to those who are unavoidably exposed to malaria. Garlic and the eucalyptus globulus have also been used as preventives. It has been proposed to plant the eucalyptus extensively in malarial regions, as a preventive measure.



## II. INTERMITTENT FEVER—AGUE.

**ÆTIOLOGY.**—Ague is the malarial fever which prevails in this country, occurring mainly in low marshy districts. Cases are also met with here in which the complaint has originated in foreign climates. Once the disease has been excited, subsequent attacks may arise without any exposure to malarial influence.

**ANATOMICAL CHARACTERS.**—The spleen presents the most notable changes in ague, being at an early period much enlarged from congestion, softened, and sometimes pulpy. After a while it becomes permanently hypertrophied and firm, constituting the so-called *ague-cake*. The liver is also congested and soft, and ultimately hypertrophied; it has been stated that albuminoid disease of this organ is occasionally produced in cases of prolonged ague. The stomach and duodenum are often congested, their mucous membrane being also softened; in some cases ulcers have been observed. In rapidly-fatal cases the heart is softened, and its tissue undergoes degeneration. Chronic Bright's disease is believed to be set up sometimes by ague. In persons who have resided for a long period in malarial districts, black pigment is often found in the spleen, liver, and kidneys. The blood is unhealthy, and may also contain black pigment.

**SYMPTOMS.**—Ague is characterized clinically by paroxysms of fever, running through certain definite stages, and occurring at more or less regular intervals, with intermediate periods of complete apyrexia.

1. The **invasion** is generally indicated by the ordinary symptoms premonitory of fever, which are noticed for some days before the disease declares itself, the pyrexia presenting well-marked remissions, with a periodic tendency. Sometimes the attack is sudden.

2. A **paroxysm** or **fit** of ague consists of three successive stages, named respectively the *cold*; the *hot*; and the *sweating stage*.

*a. Cold stage.*—Ordinarily a fit of ague is preceded by general uneasiness and languor, inaptitude for any exertion, headache, and loss of appetite. Soon the patient feels cold, first in the limbs, then along the back and over the body. The teeth begin to chatter, and finally the entire frame shivers. At the same time the general surface of the skin appears pale and shrunken, especially that of the face, the features being pinched and sharp. The tips of the fingers and the lips are blue, while in severe cases the whole surface assumes a purplish hue. Cutis anserina is frequently observed. The patient often complains of pains in the back and limbs, as well as of headache. The tongue is usually pale, moist, clean, and cool; appetite is lost, but thirst is a frequent symptom. Nausea and vomiting are not uncommon, with uneasiness and a sense of weight in the epigastrium. There is a feeling of dyspnoea, with hurried breathing, and often a dry cough, the expired air being cool. The pulse is usually frequent and small, and may be irregular.

The intensity of this stage varies greatly. There may be signs of serious depression or collapse, with a tendency to stupor or coma. Its duration ranges from a few minutes to three, four, or five hours.

*b. Hot stage.*—The transition to this stage may be sudden, but is generally gradual, being indicated by alternate flushings and chilliness, or by parts of the body becoming warm. When it becomes fully

developed, the skin feels burningly hot and dry, is red and tumid, and sometimes a patchy rash appears. The face is flushed, and the eyes are injected and sparkling. There is intense thirst, with dryness and heat of mouth, total anorexia, a white tongue, and sometimes nausea or vomiting. The heart and great arteries throb, and the pulse is generally strong and full. Respiration is more quiet than in the first stage. Headache is always present, with a sense of throbbing, and sometimes more or less delirium occurs, which may be very violent, or convulsions may set in.

This stage lasts usually from three to eight hours, the extremes being from two to eighteen hours.

*c. Sweating stage.*—Perspiration breaks out first about the forehead, and then by degrees extends over the body. Its amount varies, but it is generally considerable or even profuse, so that the bed-clothes become saturated, and sometimes even the bedding. In cases originating in certain malarial districts, the perspiration is said to have a peculiar sickly and most disagreeable odour. It continues to flow for some time, during which the pyrexia becomes reduced, and the symptoms rapidly abate; the patient usually soon falls asleep, and awakes feeling well or comparatively well. Along with the sweating there is a critical urinary discharge, and not unfrequently diarrhoea. Anasarca has been observed when sweating is deficient.

**3. Intermission.**—At first a person suffering from ague may feel quite convalescent during the periods intervening between the paroxysms, but soon more or less languor and depression is experienced, with neuralgic pains, and loss of appetite, while the patient becomes pale and anæmic. After a while permanent organic mischief is established, especially in connection with the spleen, attended with more serious symptoms, which are described in the chapter on DISEASES OF THE SPLEEN.

**Temperature.**—The course of temperature in ague is quite characteristic, the paroxysm being marked by a rapid *ascent*; short and intense *stationary period*; and critical *defervescence*; the temperature in the intervals being perfectly normal. A rise is observed as soon as, or even before, the cold stage begins; at first it is only slight and gradual, but soon becomes rapid, continuing during the hot stage, and sometimes into the commencement of the sweating stage. The temperature generally runs up to 105°, but may reach 107°, 108°, 110° or even 112° in hot climates.

When sweating begins, there is generally a slight alternate rise and fall at first, but soon a steady fall sets in, of 2° or more every five to fifteen minutes, until the temperature becomes normal. It is important to notice that even before the paroxysms are experienced, and after they have apparently ceased, the temperature has been observed to rise at the usual periods.

*Urine.*—During the cold and hot stages water is increased, but it diminishes at the close of the latter stage, and is very deficient while sweating is going on, so that the urine becomes concentrated, and its specific gravity rises. Urea suddenly increases in amount as soon as the rise in temperature begins, and this continues until the sweating stage sets in, when it rapidly or gradually diminishes, often falling below the normal. A relation is said to exist between the amount of urea discharged and the temperature. Uric acid is also considerably in excess, and urates are generally deposited at the close of a fit of ague. Chloride

of sodium is greatly increased; while phosphates are much diminished or even disappear after the height of the paroxysm. Albumen, blood, or casts are not unfrequently present in the urine. In the intervals the state of the urine varies much. It is not uncommonly alkaline in reaction. Urea is deficient as a rule during the intermissions.

**TYPES AND VARIETIES.**—The chief types of intermittent fever are those founded upon the length of the interval between the paroxysms, namely:—1. **Quotidian**, in which there is a daily paroxysm, with an interval of twenty-four hours. 2. **Tertian**, where a fit occurs every other day, the interval being forty-eight hours. 3. **Quartan**, a paroxysm taking place every third day, the interval being seventy-two hours. These are the usual types, but exceptionally the following are met with:—4. **Double quotidian**. 5. **Double tertian**, a seizure occurring every day, but at different hours, or presenting different characters. 6. **Double quartan**, out of three days two having each a paroxysm, the third none. 7. **Duplicated tertian**, there being two paroxysms one day, none the next. 8. **Erratic or irregular**. Other very rare types are described.

The *quotidian*, which is the most common, has the longest paroxysm; this is said to occur earlier in the day, and to have the shortest cold stage, but the longest hot stage. In the *quartan* variety, which is the least common, the conditions are just the opposite; the *tertian* being intermediate. Sometimes the fits tend to begin earlier or later each time, and thus ultimately one type may be converted into another; or the change may take place suddenly.

A paroxysm may present certain peculiarities. Occasionally one or more of the stages may be wanting. The phenomena are in rare instances limited to certain parts of the body; thus in paralyzed patients they may be confined to the non-paralyzed parts.

Certain forms of ague are also described which depend upon the character of the symptoms present, namely:—1. **Sthenic**. 2. **Asthenic**. 3. **Pernicious or malignant**, the last approaching the *remittent* type, and only occurring in hot climates, being attended with delirium, coma, or an algide or collapsed condition.

**COMPLICATIONS AND SEQUELÆ.**—Persons suffering under the influence of *malaria* are very liable to pneumonia, which comes on rapidly, often involves both lungs, and is of a very dangerous character. This is particularly observed in patients returning from hot malarial climates to cold climates. The blood is also prone to undergo acute changes of a serious nature, ending in marked anæmia, with excessive formation of white corpuscles, this condition being accompanied with dropsy. Various forms of neuralgia are apt to follow ague.

**PROGNOSIS.**—Intermittent fever is not often directly fatal in this country, and can usually be cured. Some of its varieties are exceedingly grave, and especially those cases classed as *pernicious*. The complications just mentioned are also very serious. If treatment has been long delayed, so that the malarial cachexia has become established, it is difficult to bring about a complete cure. The *quartan* type of ague is the most obstinate form to get rid of. It must be remembered that those who have suffered from this disease are liable to future attacks, apart from any exposure to malarial influence.

**TREATMENT.**—1. **During the paroxysm.** In the *cold stage* of an ague-fit the patient should remain in bed, being well covered with blankets, some form of dry heat being applied externally, and hot drinks



administered. In this country nothing further is necessary as a rule. When there is much depression, *diffusible stimulants* are required, and a little opium may be given to relieve great restlessness. Persistent vomiting is best checked by giving an emetic of sulphate of zinc, with plenty of warm water. If this stage is greatly prolonged a hot-air bath may be employed. In the *hot stage* the skin should be sponged freely; and cooling *effervescent* or *saline* drinks be given. During the *sweating stage* nothing is necessary but to keep the patient covered, so as to prevent a chill.

2. **During the intervals.** The great remedy at this time is quinine, and it rarely fails to bring about a speedy cure. There is much difference of opinion as to the mode in which this drug should be administered. By some it is recommended to give one large dose—gr. xx-xxx, either before, or at the close of, the paroxysm. In a large number of cases which came under my treatment at the Liverpool Northern Hospital, I obtained most satisfactory results from the administration of gr. iii-iv every four or six hours during the intermission, and therefore am disposed to adhere to this practice. In some cases it is said that the stomach rejects quinine, and then it may be combined with a little opium, or be administered by *enema*. The *subcutaneous injection* of the neutral sulphate of quinine has also been advocated. It is important to notice that the remedy must be continued for some time after the paroxysms have apparently ceased, that is, until the temperature has become quite normal. Various substances have been employed as substitutes for quinine. Of these the most reliable are cinchona bark, cinchonine, quinidine, cinchonidine, salicine or salicylic acid, and arsenic. The last is decidedly beneficial, and has the advantage of being cheap. It is best given in the form of Fowler's solution, beginning with four or five minims three times a day, after meals. Amongst other remedies recommended for ague are apiol, chinoline, Warburg's tincture, narcotine, sodium hyposulphite, and the alkaline sulphites. Dr. Mossmann of Greenville, U.S., informs me that full doses of chloral, given just before the expected paroxysm of intermittent fever, will prevent its occurrence. Nitrate of pilocarpine is also said to cut short the chill, and to avoid the hot stage, producing sweating.

3. **Symptoms and complications** may arise in the course of ague, requiring special attention. They must on no account be permitted to interfere with the use of quinine. Possibly venesection may be indicated, but I have never met with a case of ague necessitating it in this country. Adynamic symptoms must be treated by external and internal stimulation.

For the cachexia induced by ague, as well as for the different neuralgic affections, quinine, iron, and arsenic constitute the most reliable remedies, and they may be advantageously combined. Phosphorus may also be found serviceable. The tincture of eucalyptus globulus has been highly extolled. Patients should immediately be sent from a malarial region to some suitable climate, care being taken to attend to all hygienic conditions; to give good food; and to see that the clothing is warm, especially if they have to undergo a change from a hot to a cold climate. Various mineral waters and baths may be useful, such as Carlsbad and Friedrichschall waters, and warm baths or Turkish baths.

4. The **preventive** treatment of ague is that indicated for malarial diseases in general; and, if possible, residence in a malarial district should be immediately discontinued.

### III. REMITTENT FEVER.

**ÆTIOLOGY.**—The malarial fevers of hot climates often assume a *remittent* type, presenting irregular exacerbations and remissions, the latter being less distinct if the fever is very intense. They vary much in their severity, and have received many local names.

There is no distinct limit between this class of fevers and those of an intermittent type, both being due to the same cause, but this is aided by a high temperature in originating remittent fever. One type sometimes changes into the other.

**SYMPTOMS.**—There are generally *premonitory* signs, but the attack may be sudden. Gastric irritation is usually first noticed, there being a sense of uneasiness or oppression at the epigastrium, nausea, and anorexia; with headache, general pains, and a feeling of languor. Some chilliness or rigors may be experienced, but there is no *cold stage* of any duration, and the temperature rises immediately. The *hot stage* becomes very intense, the skin being burning and dry, the face flushed, the eyes injected, with extreme headache, giddiness, restlessless, sleeplessness, and often delirium, which is sometimes violent. Vomiting and nausea are commonly present, the vomited matters consisting first of food, then of a watery fluid, and finally of biliary matters; they may become brown or black. A sense of great oppression and weight is felt in the epigastrium; the tongue is furred and tends to dryness, the lips are parched, and there is intense thirst. The pulse is frequent, and either full, or small and compressible.

The symptoms abate generally in from six to twelve hours, but may continue for twenty-four, thirty-six, or forty-eight hours, or even longer. Some perspiration usually breaks out as improvement takes place.

The *remission* is of variable duration, and this is followed by an exacerbation, the symptoms being of greater intensity than during the first paroxysm. The time at which remissions take place, as well as their number, differ in different cases. When the disease is established, there is almost invariably a morning remission. The exacerbation may begin at noon, declining towards midnight; or it may begin at midnight and last till morning. In severe cases there may be a double exacerbation, namely, at noon and midnight.

As the case progresses, signs of intense adynamia may set in. Yellowness of the skin is common; and hæmorrhages sometimes occur. These symptoms, associated with black vomit, often cause remittent fever to resemble specific yellow fever. Occasionally marked jaundice is observed. The spleen and liver are usually enlarged and tender. The urine is generally stated to be scanty, dark, and of high specific gravity. In India Mr. Maclean has noticed just the opposite characters. It is always acid, and rarely albuminous. Urea is increased, and uric acid diminished, until convalescence is established.

The entire *duration* of remittent fever ranges from five to fourteen days usually. The *terminations* are in death, from blood-poisoning or exhaustion; in recovery, usually ushered in by free perspiration, but sometimes taking place gradually; or by transmission into an intermittent fever.

**TREATMENT.**—It is important to attend to *hygienic conditions*, and especially to have good *ventilation*. During the hot paroxysm cool drinks should be given freely, and, if necessary, cold may be applied to

the head. *External application of cold*, by one of the methods recommended for hyperpyrexia, is most valuable in remittent fever. Vomiting must be checked by the usual remedies. As soon as the remission occurs, quinine must be given in 10, 15, or 20 grain doses every two hours; if the stomach rejects this remedy, it must be administered by enemata. Quinine is to be given until the system has become saturated with it, and signs of cinchonism are evident. This drug is also to be used should any complications arise. Warburg's tincture has gained much repute in the treatment of remittent fevers.

It is generally stated that all antiphlogistic remedies are to be deprecated in remittent fever, as well as the use of calomel, except as an aperient. The administration of tincture of aconite has, however, been well spoken of in the treatment of this disease. It is desirable to keep the bowels well opened. Bland nourishing *diet* is necessary; and *stimulants* are often required in considerable quantities.

---



## CHAPTER XXVIII.

## ON THE DIAGNOSIS OF ACUTE SPECIFIC DISEASES.

For several reasons it has seemed to me advantageous to discuss the diagnosis of *acute specific diseases* in a connected manner, and to devote a special chapter to the consideration of this subject. Obviously it is of great moment in ordinary practice that an accurate diagnosis of either of these affections should be arrived at as speedily and promptly as possible, and, in the large majority of cases, any one possessing the requisite knowledge of their characters may come to a positive conclusion on this matter without hesitation or delay. At the same time it is always important to avoid carelessness or undue haste in forming an opinion. Not a few cases come under observation, more especially those of a febrile character, in which it is impossible at the outset to arrive at any certain conclusion as to their nature, and under such circumstances it is better to postpone the diagnosis for a shorter or longer period, watching the progress of events, and observing the phenomena which present themselves. In some doubtful cases it may be allowable, or even desirable, to give an opinion as to the probable nature of the disease, but it is a great mistake to venture upon any definite and dogmatic diagnosis on insufficient data.

Several of the acute specific disorders present such characteristic symptoms, that once these are fully pronounced, there is no difficulty in recognizing their nature. Others, however, are not so clearly marked off, and careful consideration may be necessary in order to discriminate between them. I proceed therefore to point out briefly the chief facts which have to be known and taken into account, in order to form an opinion as to the diagnosis of these complaints.

1. The diagnosis is materially assisted by a correct acquaintance with the **ætiology** of each acute specific disease, and by the information elicited in relation thereto in any individual case. It is important to remember which of these complaints are infectious, and which are frequently, or may be exceptionally, originated in other ways; the modes in which the contagium is conveyed in each disease, and the channels by which it enters the system; the conditions, both intrinsic to the individual and extrinsic, which affect its propagation; the period of incubation; and the effect upon the patient of previous attacks, whether these afford probable security against future attacks, or increase the predisposition to the disease.

The early recognition of a particular acute specific disease may be much aided by the fact that an epidemic is prevalent at the time; or by direct evidence that the patient has been exposed to infection in some way, or has been under the influence of some other well-known cause of fever, such as malaria. It not uncommonly happens, however, that very careful inquiry is necessary in order to trace infection, as, for instance, in many cases of typhoid fever and scarlatina; the contagium too may be so obscurely conveyed by fomites, that it may be most difficult or even impossible to track it to its original source; while it must be borne in mind that some diseases which are ordinarily transmitted by infection, such as typhus or relapsing fever, may possibly be generated *de novo* under certain circumstances. Hence, even if a source of contagion cannot be discovered, this does not necessarily exclude a specific disease which may be suspected to be present. As an illustration of the value of remembering the conditions which influence the occurrence of acute specific diseases, may be mentioned the effects of age and hygienic conditions with respect to typhoid and typhus fever respectively. Most of these complaints are, as a rule, only observed once in the same individual, and hence, in the case of any suspected fever, it is well at the outset to find out what affections of this class the patient has previously suffered from, so that these may be excluded by probability. At the same time the fact must be recognized that second and even third attacks of the eruptive fevers do sometimes occur; while certain of the acute specifics, such as erysipelas, are predisposed to by previous attacks, and this may help in fixing upon, or at least in suspecting one of these affections, before the clinical phenomena are at all significant.

2. Obviously it is essential to have a complete and correct acquaintance with the **clinical history** of each of the affections now under consideration, before a diagnosis can be made with any degree of certainty. This includes:—

a. Its *mode of invasion*.

b. Its more *characteristic symptoms*, particular attention being paid to those which are present at an early period.

c. The chief facts connected with the *skin-eruption*, in the case of those diseases which are attended with such a phenomenon.

d. The *degree of pyrexia*, and *course of temperature*, as indicated by the thermometer, when this is at all significant.

e. Its *ordinary course*, *duration*, *modes of termination*, and *mortality*.

f. The *varieties* it may assume, so as to be prepared for any deviation from the usual type.

g. Its *ordinary complications* and *sequelæ*. A knowledge of these is useful in several ways in a diagnostic point of view. Thus it may guard the practitioner against mistaking some local mischief which he may have discovered, such as pneumonia, for the entire disease, when it is but a complication of some fever; it enables him further to be on the look-out for such morbid conditions as are liable to arise in the course

of, or after any particular fever; while the development of some special and peculiar complication or sequela may throw much light upon the nature of an otherwise obscure case.

3. The diagnosis of the specific fevers more especially, is greatly aided and facilitated by a definite knowledge of the complaints which they severally resemble, and with which they are likely to be confounded. In the first place it is requisite to bear in mind, with regard to each of them, the diseases of its own class which approach it in its characters, and are liable to be mistaken for it; and, secondly, other febrile diseases which are more or less like it, whether dependent upon local mischief, or due to some deleterious agent within the system, such as rheumatic fever or pyæmia.

It would only involve unnecessary repetition to recapitulate the points just indicated with reference to each acute specific disease, as they have been already considered in detail, and my main object in this chapter has been to sketch the kind of information required, and to suggest the line of reasoning to be adopted, in order to arrive at a diagnosis of individual cases of these complaints. I propose, however, first to make a few remarks respecting FEBRICULA; then to present in the form of a table the chief clinical features of the principal fevers prevalent in this country, and the diseases for which they are liable to be mistaken; and, lastly, to notice briefly each of the more peculiar, or less common specific affections.

FEBRICULA.—The occurrence of a sharp febrile attack, unattended with phenomena characteristic of any other fever, and not dependent upon any discoverable local lesion, may be looked upon as indicative of *febricula* or *synocha*. The rapid rise of temperature is important, especially in distinguishing this complaint from typhoid fever. At the outset of course it is often impossible to say what may be the nature of any particular case; and there can be no doubt but that many cases of so-called febricula are really examples of pyrexia associated with some local morbid condition which has not been detected. The opinion has been advanced that within the group of cases classed as febricula are included more than one form of specific fever, perhaps several, but the present state of knowledge certainly does not warrant any such assumption.

#### DIAGNOSTIC TABLE OF THE PRINCIPAL FEVERS.

In the following table, the main clinical characters of the diseases recognized as *specific fevers*, which are ordinarily met with in this country, are arranged according to the plan indicated in the preceding remarks; after which the affections which they severally resemble, and are likely to be mistaken for, are pointed out; and lastly, any special facts which demand attention, as bearing upon the diagnosis of either of these complaints, are noticed.



	Typhoid Fever.	Typhus Fever.	Relapsing Fever.	Scarlatina.
MODE OF INVASION.	Often very gradual and ill - defined. Patient cannot fix date. No marked rigors. May be only diarrhoea for some time.	Generally well-marked, and may be very sudden. Usually marked rigors. Speedy prostration and high fever.	Generally remarkably sudden. Severe rigor. Great weakness.	Usually distinct. Chilliness, but not severe rigors. Rapid pyrexia. May be nervous phenomena, such as convulsions or coma.
CHARACTERISTIC SYMPTOMS.	Marked frontal headache, but other head symptoms not prominent, or only come on at a late period. Abdominal symptoms, viz., pain and tenderness, especially in right iliac fossa; tympanites; gurgling in right iliac region; diarrhoea, with peculiar stools; not uncommonly intestinal hæmorrhage. Enlarged spleen. No striking prostration. Epistaxis not uncommon. Tongue peculiar at first. Pulse liable to great variations.	Great depression and prostration. Dying and muddy aspect of countenance, with dusky flush on cheeks, and dull heavy expression. Early and marked head symptoms, and low nervous phenomena. Rapid tendency to typhoid condition. Pupils often much contracted.	Sharp frontal headache. Severe pains in back and limbs. Pains in epigastrium and hypochondriac regions. Bilious vomiting with retching. Enlargement of liver and spleen. Peculiar aspect of countenance. No marked nervous symptoms usually. Much debility, but not prostration. Pulse very frequent at an early period. Epistaxis and other hæmorrhages common.	High pyrexia. Flushed face. Sore-throat of variable intensity, but always marked. Vomiting. "Strawberry" tongue. No prominent head symptoms usually. Pulse very frequent, generally strong and full.
SKIN-ERUPTION.	7th to 12th day. Generally confined to abdomen, chest, and back. Comes out in successive crops, each lasting 2 to 5 days. Consists of minute rose-coloured spots, slightly raised, disappearing on pressure. Only a few visible at the same time. Continue to appear until 28th or 30th day, or even later. Sudamina not uncommon.	4th or 5th day usually. First about back of wrists, axillæ, or epigastrium; spreads rapidly over body and limbs, but rarely seen on neck or face. Entire eruption out in from 1 to 3 days. Consists of: - <i>a</i> , irregular, dusky-red subcuticular mottling; <i>b</i> , maculæ or mulberry spots, deepening in colour, and soon not fading on pressure. Disappears from 14th to 21st day. Skin gives off peculiar odour. Sudamina less common than in typhoid.	No specific eruption. Sudamina may appear at crisis. Often fine desquamation of cuticle after crisis.	2nd day usually. First on neck and upper part of chest; spreads rapidly to face and over trunk and limbs. Consists of a rash, beginning as minute points, coalescing to form patches or to cover entire surface; colour more or less bright-red; no elevation generally. Reaches height 4th to 5th day, then gradually fades to 9th or 10th day. Followed by marked desquamation. Often puffiness of face, &c.; much itching and tingling, with burning sensation. Sudamina common.
DEGREE OF PYREXIA, AND COURSE OF TEMPERATURE.	Peculiar ascent; rise of 2° each evening with morning remission of 1°, therefore daily rise of 1°; temp. finally reaches to from 104° to 106° in evening usually, with slight morning remission. Decline gradual, indicated by more distinct morning remission, followed by slight evening fall, with very considerable morning remission; some time before evening temp. normal.	Regular ascent without any remission until 4th or 5th evening, up to 104°, 105°, or higher; then usually slight morning remission, and marked fall on 7th morning; subsequent rise, but not to former maximum usually; then continuous, with morning remission varying from $\frac{1}{2}$ ° to 1½°. Rapid decline, temperature becoming normal in from 12 to 48 hours.	Continuous ascent for 4 or 5 days; temp. at last reaches 104° to 106°; slight morning remissions. Rapid fall at crisis to below normal, with subsequent speedy rise to former temp. or even higher; sudden deferescence again at second crisis.	Continuous ascent until rash attains height; generally reaches 104° to 106°, may be higher; slight morning remission. Deferescence may be by crisis; or gradual when eruption begins to fade.

Measles.	Small-pox.	Varicella.	Erysipelas.
<p>Rather sudden, and generally marked. Chilliness or rigors. Occasionally convulsions.</p>	<p>Generally sudden. Often repeated and strong rigors. Rapid pyrexia.</p>	<p>Not marked.</p>	<p>Often gradual, with indefinite febrile symptoms.</p>
<p>Moderate pyrexia. Catarrhal phenomena, viz., coryza, &amp;c. Only slight sore-throat, if any. More or less catarrh of respiratory passages, extending to bronchi, and increasing during eruptive stage. May be much diarrhoea and vomiting.</p>	<p>High pyrexia. Uneasiness or pain in epigastrium. Nausea and much vomiting. Severe pain in back. Much debility and sense of illness. Fever rapidly abates when eruption appears, followed by secondary fever. Often symptoms pointing to implication of mucous surfaces. Prodromal rashes may appear, especially in certain parts.</p>	<p>Slight pyrexia, without special symptoms; or may be no symptoms at all.</p>	<p>No special symptoms at early period, but premonitory symptoms of other exanthemata absent. May be much fever. Often marked rigor before local phenomena appear. Objective signs may be preceded by subjective sensations, as heat, irritation; or by enlargement of lymphatic glands in the neighbourhood. In facial erysipelas may have epistaxis at outset. Severe symptoms during eruption, with tendency to typhoid condition.</p>
<p>4th day generally. First on face, especially forehead, then spreads to trunk and limbs, often in three distinct crops on successive days. Consists of minute red points at outset, soon enlarging to distinct papules, which tend to form crescentic or semilunar patches; colour darker than that of scarlatina. Declines in same order in about 12 hours. Followed by slight desquamation; reddish or coppery discoloration may remain for some time. Much itching.</p>	<p>3rd or beginning of 4th day. First on face, especially on forehead; spreads over body and limbs in a day or two, often in successive crops. Consists of "pocks" going through stages of:—<i>a.</i> small bright-red spot; <i>b.</i> hard, shot-like pimple; <i>c.</i> vesicle, becoming umbilicated; <i>d.</i> pustule, reaching maturation about 8th day; <i>e.</i> scab, which leaves reddish-brown stain on separation, or a pit. Hard inflamed areola forms. Very variable mode of arrangement and number. Swelling of face, &amp;c. Intense itching. Disagreeable odour given off. Eruption may be on mucous surfaces.</p>	<p>Within 24 or 36 hours. First about shoulders and chest; extends over body and limbs; may be many on scalp; but generally few on face; comes out in successive crops for 4 or 5 nights. First bright-red, slightly papular spots, not hard; in few hours vesicular, large and ill-defined, superficial, not umbilicated, no inflamed areola; do not become pustular, but gradually opalescent, and dry up or rupture 3rd to 5th day. Usually few in number and scattered. As a rule no pitting left.</p>	<p>Usually within 2 or 3 days. Generally on face and head. Signs of rapidly spreading inflammation of skin from a point, usually in some one direction, sometimes equally in all. Much heat, redness, swelling, and tension. Followed by formation of vesicles or bullae, which rupture or dry up. Subsequent extensive peeling of cuticle. May end in suppuration, ulceration, or gangrene. Inflammation liable to extend after apparently stopping. May assume erratic or metastatic character; or extend to mouth, throat, larynx, &amp;c. May affect limbs or other parts.</p>
<p>Continuous ascent up to height of rash; temp. not usually above 103°; morning remissions slight, marked, or absent. Defervescence by rapid crisis, from 4th to 10th day. Temp. liable to be much influenced by complications.</p>	<p>Rapid rise of temperature to 104°–106°. Speedy fall nearly or quite to normal when eruption appears; a second rise as the eruption matures, varying with the amount of this; temp. reaches 104° or 105° in a typical case. Gradual defervescence. May be another elevation of temperature when desiccation occurs.</p>	<p>No special course. Pyrexia usually slight; may be marked rise of temp. in the evenings.</p>	<p>Very variable course according to part affected. In facial erysipelas rapid rise of temp. usually. When local inflammation appears may reach 104° or 105° on first evening; increases so long as inflammation extends, but usually reaches maximum on 3rd day; may be 106° or more; generally evening exacerbations, but may be distinct fall. Defervescence usually about 5th or 6th day of inflammation; as a rule by rapid crisis, temp. becoming normal in 12 to 36 hours; may be more gradual. Temp. will rise with relapse or extension of inflammation, and is much influenced by complications.</p>

	Typhoid Fever.	Typhus Fever.	Relapsing Fever.	Scarlatina.
COURSE, DURATION, TERMINATIONS.	Continuous. Duration usually from 3 to 4 weeks; rarely beyond 30 days. Most cases end in recovery. No crisis, but defervescence gradual. Convalescence slow, and health liable to remain permanently impaired. Relapse not uncommon.	Continuous. Duration usually from 14 to 21 days. Most cases recover, but mortality varies in different epidemics. Generally marked crisis, followed by deep sleep, and rapid improvement in symptoms. Convalescence usually comparatively speedy. Relapse exceedingly rare.	Peculiar course, viz., sudden crisis usually from 5th to 7th day, attended with profuse sweating and other phenomena, followed by complete or partial cessation of symptoms; from 12th to 17th day sudden relapse, with same symptoms as at first; second crisis generally in from 3 to 5 days; may be a series of relapses. Duration variable. Almost always recovery, but convalescence often very slow, with debility, anemia, muscular and arthritic pains, &c., as sequelæ.	Continuous. Duration often prolonged, on account of desquamation. Mortality very variable, but often high. Convalescence liable to be retarded by various sequelæ, especially renal disease, with albuminuria and dropsy. Very rarely relapse.
DISEASES RESEMBLING.	<p>Fevers.</p> <ul style="list-style-type: none"> <li>Typhus fever,</li> <li>Relapsing fever,</li> <li>Febricula.</li> </ul> <p>Other diseases.</p> <ul style="list-style-type: none"> <li>Acute tuberculosis.</li> <li>Tubercular meningitis.</li> <li>Pneumonia.</li> <li>Pyæmia.</li> <li>Renal disease with uræmia.</li> <li>Gastro-enteritis.</li> <li>Chronic peritonitis, with ulceration of the bowels.</li> <li>Perityphlitis.</li> </ul>	<p>Typhoid fever. Relapsing fever. Measles at early period. Erysipelas. Febricula.</p> <p>Asthenic pneumonia, especially in old, feeble, or intemperate persons. Cerebral or meningeal inflammations. Certain cases of delirium tremens. Blood-poisoning from uræmia, pyæmia, or septicæmia.</p>	<p>Typhus fever. Typhoid fever.</p> <p>Rheumatic fever. Gastric or hepatic affections.</p>	<p>Measles. Rôtheln. Diphtheria. Small-pox in the invasion-stage.</p> <p>Acute throat-inflammations. Rosæola. Urticaria. Erythema.</p>
REMARKS.	Cases of typhoid fever are liable to be very obscure, and present considerable differences in their clinical history; care is therefore necessary in diagnosis. When there is <i>persistent diarrhœa</i> , always bear in mind typhoid fever, and use the thermometer. Patients may walk about during the entire attack. The most important complications are <i>perforation of the bowels</i> and <i>peritonitis</i> . More or less bronchial catarrh is always present.	Typhus fever can be generally recognized without much difficulty. It differs from typhoid not only in the points indicated in the table, but also in frequently attacking persons beyond middle life, and being much influenced by unfavourable hygienic conditions, which probably may even originate the disease <i>de novo</i> . There is a great liability to <i>hypostatic congestion</i> .	Relapsing fever is most liable to be mistaken for typhus at first, but the differences indicated above ought to enable the diagnosis to be made. The two diseases are promoted by the same anti-hygienic conditions. Among the most distinctive complications are <i>abortion</i> , and a peculiar form of <i>ophthalmia</i> .	Scarlatina presents several important varieties. There may be no rash at all, and cases may present very slight symptoms; on the other hand these may be of a very malignant character, ending in speedy death, making the diagnosis very difficult. The occurrence of scarlatina may only be known by desquamation taking place, or renal disease setting in.



Measles.	Small-pox.	Varicella.	Erysipelas.
<p>Continuous. Duration usually within a fortnight. Most cases recover. Termination generally by rapid crisis. Very rarely relapse.</p>	<p>Course and duration very variable. Termination fatal in large proportion of cases, about 1 in 3. Recovery is gradual, and convalescence often much delayed.</p>	<p>Short duration. Never fatal. Generally speedy recovery.</p>	<p>Variable duration. Termination not uncommonly fatal. When favourable, usually marked crisis. Convalescence established at variable time.</p>
<p>Scarlatina. Rötheln. Small-pox (at early stage of eruption). Typhus fever. Influenza (in the invasion-stage).</p>	<p>Febricula. Varicella. Measles (in early stage of eruption).</p>	<p>Small-pox.</p>	<p>Typhus fever.</p>
<p>Roseola. Syphilitic exanthem. Flea-bites.</p>	<p>Lichen (at early period). Pustular syphilitic eruption.</p>		<p>Acute Eczema. Erythema.</p>
<p>Measles may occur without its eruption, or without its catarrhal symptoms. There are also malignant varieties, difficult to recognize. The diagnosis of <i>Rötheln</i> has been sufficiently indicated in the account of that disease, and it has not been thought necessary to consider it in this table.</p>	<p>Small-pox presents many varieties, especially as regards the eruption, and this is greatly modified by vaccination; it is even believed that there may be no eruption; malignant forms of the disease are also met with. Hence diagnosis may be difficult. The greatest difficulty lies in distinguishing mild cases from those of chicken-pox. The occurrence of the prodromal rashes is important in the diagnosis of early cases of small-pox.</p>		<p>Cases of erysipelas are often very difficult to recognize at the outset. The complaint might be suspected if there were febrile symptoms, without any of the premonitory signs of other fevers, or any symptoms pointing to local disease, especially if accompanied with any unusual subjective sensations about the face or other parts; or if lymphatic glands seem to be inflamed. The fact of a patient having had previous attacks of erysipelas may help in foretelling a coming one.</p>

**DIPHTHERIA.**—The character of the general symptoms; the local throat-symptoms; with satisfactory examination of the throat, will in most cases enable this disease to be recognized. It may at first be mistaken for any form of acute sore-throat, or for scarlatina, extensive thrush, or herpes on the pharynx. In the last-mentioned complaint vesicles may be seen on the fauces, which cannot be removed; the pain is much more severe, but limited; and herpes is also visible on the lips. Laryngeal diphtheria cannot be distinguished from croupous laryngitis, and these two conditions may be regarded as identical. Some asthenic cases of diphtheria may be difficult to recognize at first. A history of exposure to infection may aid the diagnosis, but it must be remembered that diphtheria may probably arise without such exposure.

**INFLUENZA** is sufficiently characterized by its epidemic distribution; marked febrile symptoms, with great depression: and catarrhal phenomena. It might be confounded with simple catarrh.

**HOOPING-COUGH.**—In the early stage this complaint cannot be diagnosed with certainty, but it might be suspected if the disease is epidemic: if a child suffers from cough of a violent and spasmodic character; and if there is much pyrexia. Subsequently the peculiar fits of cough, with characteristic expectoration, are usually absolutely distinctive of whooping-cough. In doubtful cases ulceration about the frænum linguæ might help the diagnosis.

**MUMPS.**—This affection is also very easily recognized as soon as the peculiar inflammation of the parotid gland sets in, running its special course. The local condition might be mistaken for inflamed lymphatic glands in the neighbourhood. The metastatic inflammations which are liable to arise must be borne in mind.

**GLANDERS—FARCY.**—When these diseases are fully declared, their diagnosis presents no difficulty. At the onset glanders may be mistaken for acute or subacute rheumatism, but the occupation of the patient might lead to the suspicion of glanders; while in this affection there is more prostration from the first, and the joints are not swollen or red.

**MALIGNANT PUSTULE.**—The occupation of the patient is an important element in the diagnosis of early or obscure cases of malignant pustule, and this complaint should always be borne in mind under such circumstances. The external manifestation has been mistaken at first for a mosquito-bite, and absorption of arsenic through an abrasion; and later for malignant facial carbuncle, a poisoned wound, and primary chancre of the face. When the pustule presents well-marked characters, there ought to be no difficulty in diagnosis. In doubtful cases the finding of bacilli in the fluid of the vesicle may verify the diagnosis. This may also assist the diagnosis in cases where there is mere œdema. Internal anthrax is very difficult to diagnose positively, except by taking into consideration the occupation of the patient, and is liable to be mistaken for many other affections.

**HYDROPHOBIA** is another quite peculiar disease, the symptoms of which are highly characteristic. The chief liability to error lies in the fact that a patient who has been bitten by a supposed mad dog may be imagining all kinds of symptoms simulating those of hydrophobia, especially if of a nervous or hysterical temperament.

**CHOLERA.**—During an epidemic of cholera, it is the safest plan to treat any case presenting suspicious symptoms as being of this nature. The painless purging and vomiting, with "rice-water" stools; cramps; intense thirst; great restlessness; suppression of secretions; rapid collapse;

and peculiar appearance of the face, are but too significant of the malady. A sporadic case often presents much difficulty, and it may be impossible to determine whether it is one of Asiatic cholera or choleraic diarrhoea. In choleraic diarrhoea usually some cause can be found for the attack; it is less severe; the stools and vomited matters contain bile; more or less griping is felt; urine is not entirely suppressed; the duration is longer; and the mortality is much less. Cholera may set in so violently and under such circumstances as to simulate irritant poisoning. On the other hand intense gastro-enteritis, excited by poison or some other irritant, has been mistaken for cholera; and so likewise has the collapsed condition resulting from the rupture of a gastric or duodenal ulcer.

CEREBRO-SPINAL FEVER is another very well-marked disease, characterized by the circumstances under which it arises; its sudden onset; high fever; and symptoms indicative of grave cerebro-spinal meningitis. The appearance of herpes, or of purpuric spots and hæmorrhages, may also assist the diagnosis. The complaint might be mistaken at first for typhus, typhoid, or relapsing fever; and subsequently for tetanus; but there is more danger of its being confounded with other forms of meningitis.

DENGUE—PLAGUE—YELLOW FEVER—REMITTENT FEVER.—The diagnosis of these diseases, which prevail in foreign climates, needs but little special notice. The symptoms of dengue and plague have already been sufficiently described. Yellow fever and malarial remittent fever may resemble each other, both being frequently attended with a yellow skin, and with black vomit. The points of distinction are said to be as follows:—Yellow fever is contagious, has only one paroxysm, and is not periodic; a second attack is very rare; the disease cannot prevail at a temperature at which malarial fevers are often met with: hæmorrhages and albuminuria are very common, the latter being almost invariable; while quinine has not the influence over the disease which it exercises in the case of malarial fever. Relapsing fever might possibly be mistaken for yellow fever, but it differs in its course; in attacking chiefly the poor and destitute; in the rarity of black vomit, and comparative infrequency of jaundice; and in being scarcely ever fatal.

INTERMITTENT FEVER in this country is almost always readily recognized by its peculiar paroxysmal febrile attacks, and course of temperature. Some difficulty might be experienced at first in distinguishing a case of ague, where there was no history of malarial exposure, which is not always easily traced, but all doubt ought to be removed by watching the patient for a day or two. Cases of malarial cachexia might afford some difficulty in diagnosis.

---



## B. CONSTITUTIONAL DISEASES.

## CHAPTER XXIX.

## RHEUMATISM.

THIS term is applied to very different diseases, some of which are entirely of a local nature. For convenience' sake, however, it will be expedient to consider all the varieties of rheumatism in the present chapter.

## I. ACUTE ARTICULAR RHEUMATISM—RHEUMATIC FEVER.

**ÆTIOLOGY.**—It is generally believed that the immediate *pathological cause* of rheumatic fever consists in the presence in the blood of a morbid material, generated within the system in consequence of some derangement of the nutritive and eliminatory processes, which acts as a direct poison upon certain tissues. This agent is, moreover, presumed to be a normal ingredient of one of the ordinary excretions, only accumulated in excess, the common opinion being that it consists of *lactic acid*. The results of experiments seem to favour this view, the usual phenomena of acute rheumatism having been produced by injecting this acid into serous cavities, as well as during its internal administration. Another view maintained as to the origin of rheumatic fever is that it depends upon some disturbance of the nervous system. Mr. Hutchinson calls rheumatism a "catarrhal arthritis," and is of opinion "that it is, in the main, a liability to joint-disease, brought about by exposure to cold and wet, through reflex nervous influence." Others think that the nervous centres are affected by the lactic acid, and that they react upon the joints; while still another view supposes that a primary disturbance of the nervous system leads to disorder of nutrition, and the production of lactic or other acids, which then cause the symptoms of acute rheumatism.

The germ-theory has been advanced to account for rheumatic fever. Hueter has extended his view of the origin of inflammation generally to this disease. He supposes that micrococci enter the system and set up endocarditis, the joint-affection being secondary to this, and due to embolism. Dr. Harkin of Belfast has advanced a different endocardial theory of the origin of acute rheumatism. He believes "that it is essentially a specific form of endocarditis of neuropathic origin, generally allied with myocarditis; that in its unchecked progress, it speedily modifies the composition of the blood, the innervation and calorification of the body; in its ordinary course giving rise to lesions in the textures,

the joints, pericardium, pleura, the neurilemma, the meninges of the brain; in fine, in any organ accessible to nervous or arterial influence." He considers that the exciting cause is a chill, and that the effect is produced through the medium of the nervous system.

Dr. Dyce Duckworth, following Charcot, believes that there is a basic arthritic stock or diathesis, from which arise as branches two main classes of disorder, commonly recognized as rheumatism and gout.

Rheumatism has been attributed by Dr. MacLagan to a miasm entering the system from without, generically allied to, but specifically distinct from, the miasm of malarial fever.

*Exciting causes.*—The ordinary exciting cause of acute rheumatism is a sudden chill, induced by exposure to cold and wet; sitting in a draught when heated or perspiring; neglecting to change wet clothes; or in various other ways. In not a few instances no definite cause can be fixed upon. Errors in diet, digestive and hepatic derangements, suppression of menses, chronic uterine diseases, excessive lactation, and numerous other disturbances have been ranked as causes of acute rheumatism. Scarlatina seems to lead to this complaint in some instances, probably by interfering with the excretory functions of the skin.

*Predisposing causes.*—Acute rheumatism is distinctly a hereditary disease, and it tends to run in families. It chiefly attacks for the first time persons from 15 to 35 years old, being especially frequent between 16 and 20, but no age is exempt, and I have met with a good many cases in young children. Previous attacks decidedly increase the predisposition to the disease. More cases are met with among males, and in the lower classes, on account of their greater exposure to the ordinary exciting causes. Climate and season have considerable influence, the affection occurring mainly in temperate but very moist climates, and where sudden changes of temperature are experienced. It is far less common in tropical and very cold countries. A large number of cases are met with in the eastern counties of England. The same conditions influence the prevalence of the complaint at different seasons. A state of ill-health from any cause is said to predispose to rheumatic fever, as well as mental depression or anxiety; but many individuals are attacked when in apparently perfect health and spirits. Joints which are much used, or which have been injured, are particularly liable to become affected in the course of the disease.

*ANATOMICAL CHARACTERS.*—The morbid changes associated with acute rheumatism are chiefly evident in connection with fibrous, fibro-serous, and synovial structures. A variable number of the joints of the limbs present signs of acute inflammation. The synovial membrane is very vascular, thickened, and relaxed; there may be a deposit of more or less lymph; and the joint contains a moderate quantity of fluid effusion, chiefly serous, but having flakes of fibrin and abundant cells floating in it, the latter often resembling pus-cells. The tissues around the joints are much infiltrated with fluid. In cases of long duration actual pus may form, and the cartilages sometimes become eroded. The sheaths of tendons may also be inflamed, and occasionally they contain a purulent fluid. The muscles are often dark, soft, and infiltrated in fatal cases.

In the majority of cases where death occurs from rheumatic fever, the morbid appearances characteristic of pericarditis, endocarditis, or myocarditis are visible. Fibrinous vegetations are common in the heart, even when no inflammation exists. Pleurisy and pneumonia are also

not unfrequently present; and, rarely, signs of peritonitis, or of cerebral or spinal meningitis may be observed.

The blood contains excess of fibrinogenous elements, and becomes buffed and cupped during coagulation. The solids generally are diminished, but they are in excess in the serum. It is doubtful whether lactic acid can be detected in the blood.

**SYMPTOMS.** 1. **Invasion.**—An attack of rheumatic fever may set in gradually, being preceded by a state of general ill-health for some time; but usually the invasion is prominently marked by chills, or occasionally by distinct rigors. These are followed by pyrexia; and soon the joints or other structures become affected.

2. **Actual attack.**—When acute rheumatism is established, the symptoms are in most cases very characteristic, being both *general* and *local*, the latter being connected with the joints, but the two classes of symptoms are not always in proportion to each other.

*a. General.*—The patient complains of general soreness and stiffness, and presents an aspect of pain and suffering, combined with restlessness and weariness, but is unable to move, on account of the painful state of the joints, being often quite helpless. Usually there is copious perspiration, the patient being bathed in sweat, which has a peculiarly sour or acrid smell, and is generally very acid in reaction. Sudamina appear not uncommonly, and they may be extremely abundant, coming out in successive crops. There are the ordinary symptoms accompanying pyrexia. The pulse is generally full and strong. The tongue is thickly coated, while there is much thirst, with anorexia, and constipation. The urine is markedly febrile, deposits urates abundantly, and sometimes contains a little albumen. Patients often cannot sleep on account of the pain which they suffer, but there are no particular head-symptoms as a rule. Occasionally slight delirium is observed. In some instances the general symptoms tend to assume a typhoid character.

*b. Local.*—It is the *middle-sized* joints which are most commonly attacked in rheumatic fever, namely, the elbows, wrists, knees, and ankles, but the other articulations are by no means exempt. Usually many joints are involved in succession, the complaint exhibiting an erratic tendency, and often the symptoms subside in one articulation as they appear in another, but several may be implicated together. The same joint may be attacked more than once in the course of the disease. A disposition to symmetry in the joints which are involved is frequently noticed.

An affected joint is more or less red, either uniformly or in patches; swollen and enlarged; and hot. The amount of enlargement varies, and the swelling is due partly to infiltration of the tissues around the joint, partly to effusion into its interior. The skin sometimes pits on pressure. There is considerable pain and tenderness, which is aggravated at night; and any movement causes much distress. In character the pain is dull and aching as a rule, and may be so severe as to make patients cry. Frequently the suffering is less intense when the swelling is considerable.

**Temperature.**—The pyrexia of acute rheumatism is of an irregularly remittent type. The *ascent* usually lasts about a week, but it may be longer or shorter than this. The temperature in most cases ranges from 100° to 104°. The *stationary period* varies greatly in duration; there is generally a considerable difference between morning and evening temperature. *Defervescence* is gradual and indefinite in most cases,



*crisis* being rare. The implicated joints may indicate a higher temperature than other parts. Rheumatic fever is one of the diseases in which *hyperpyrexia* is most frequently observed, a remarkably sudden rise in temperature taking place, attended with grave symptoms, namely, severe rigors; marked general depression; prominent nervous phenomena; and sometimes jaundice, diarrhoea, or hæmorrhages; death usually speedily ensuing. The temperature may reach  $109^{\circ}$ ,  $110^{\circ}$ ,  $112^{\circ}$ , or more, and continues to rise after death. Irregularities in temperature are very common in rheumatic fever, even apart from any complications, and the latter are often not indicated by the thermometer in this disease. A disproportion between the temperature and the pulse is frequently observed.

**Subacute Rheumatism.**—A subacute variety of rheumatism is by no means uncommon, especially in hospital practice, which is very troublesome. There is but slight pyrexia, and one or more joints continue to be affected for a long time, with but little change, except that occasional exacerbations are liable to occur from slight causes, or even without any evident cause. The joints are not much deformed, nor are they structurally altered to any marked degree. The general condition is usually much below par.

**COMPLICATIONS AND SEQUELÆ.**—In most cases certain internal organs and structures are implicated in the course of an attack of rheumatic fever, and the resulting morbid conditions are ordinarily classed as *complications*, but in reality most of them are *parts of the disease*, and they may occur *without any joint-affection*. At present they will be merely enumerated, as their symptoms and signs are described in other parts of this work, but it must be specially noted that they may come on very insidiously, and should therefore be constantly watched for, especially those connected with the heart, this organ being examined at least once or twice daily during an attack of rheumatic fever. These complications chiefly include:—1. Cardiac affections, namely, pericarditis; endocarditis, with consequent valvular disease; myocarditis; and the formation of fibrinous deposits in the cavities of the heart. 2. Pulmonary diseases, including pleurisy, pneumonia, bronchitis, and pulmonary gangrene in exceptional cases. 3. Rarely peritonitis. 4. Cerebral and spinal meningitis very rarely, brain-symptoms formerly supposed to be due to meningitis being really attributable usually to hyperpyrexia. In alcoholic subjects delirium tremens may supervene; and a peculiar form of insanity has been described. The cardiac affections are by far the most common, and are especially frequent in the young, being met with in cases of all grades of severity.

Choreiform movements are not uncommon in acute rheumatism, or even a distinct attack of chorea, especially in children. The relation between them is a matter of doubt. It is supposed by some that the chorea is of embolic origin, being due to the plugging of the small vessels of some part of the brain with particles of fibrin, conveyed from the heart. This subject will be more fully discussed under chorea. Rheumatic subjects are liable to serious inflammatory affections in connection with the eye, namely, ophthalmia, sclerotitis, or iritis. Cutaneous complications may accompany rheumatism, such as erythema nodosum, urticaria, or purpura. Orchitis or inflammation of the tunica vaginalis is occasionally met with. Acute rheumatism and scarlatina may occur together, and some authorities consider that there is an important relation between them. There seems every reason to believe

that although essentially distinct diseases, rheumatism and gout may be present in the same subject, constituting true "rheumatic gout." Mr. Hutchinson says that probably rheumatic gout is far more common than pure gout, but infinitely less common than pure rheumatism.

Sequelæ very often follow rheumatic fever, the most important of these being permanent organic disease of one or other of the orifices or valves of the heart, especially the mitral, which leads to subsequent changes in this organ. In some cases pericardial adhesions remain. Not uncommonly the patient continues in a weak and anæmic condition for some time. In one case which came under my observation, the patient became imbecile after an attack of acute rheumatism.

It will be convenient to notice here certain subcutaneous nodules, specially described by Drs. Barlow and Warner as occurring in children and young adults, the subjects of rheumatism and chorea. They are observed in connection with fasciæ and tendons, especially near joints, most frequently over the back of the elbow, over the malleoli, and on the margins of the patella. They vary in size from a mustard seed to a bitter almond; are slightly movable usually; mostly symmetrical; and consist of loose fibrous bundles, sometimes very vascular, but never becoming bony. The skin over them is simply raised, without any heat, pain, redness, or infiltration; and often there is no pyrexia, marked fever being rare. The nodules may appear in one crop, or in succession; or they may subside partially or entirely, and then reappear.

COURSE, DURATION, AND TERMINATIONS.—Cases of rheumatic fever differ greatly in severity, and therefore their *course* and *duration* are exceedingly variable, but favourable cases generally become convalescent within from three to six weeks. Complications will necessarily materially influence their duration. *Relapses* are also frequent. The *termination* in the great majority of cases is in recovery, but permanent organic mischief often remains behind. Sometimes stiffness of joints continues for a considerable period, or they may become chronically affected; they are also liable to subsequent neuralgic pains. Death generally results either from internal complications; or from hyperpyrexia.

DIAGNOSIS.—Gout is the chief disease from which rheumatism has to be distinguished; the points of difference will be considered under gout. Articular rheumatism has also to be diagnosed from the other forms. Rheumatic fever may be simulated at first by erysipelas, pyæmia, trichinosis, dengue, or the early stage of glanders. The symptoms occurring during the apyrexial period of relapsing fever may also resemble those of acute rheumatism. Care must be taken not to mistake between local inflammation of a joint and rheumatism. It must, moreover, be remembered that during an attack of this disease the joints may not be implicated in the least. The presence of subcutaneous nodules is considered by Drs. Barlow and Warner as an aid in the diagnosis of a rheumatic condition, when associated with chorea and heart disease, although no history of rheumatic fever can be obtained.

PROGNOSIS.—As regards life and death, the prognosis is very favourable in cases of acute rheumatism, but in many instances it is grave with respect to the future condition of the patient, on account of the organic mischief which has been set up. The chief indications of immediate danger are a very high temperature, or one remaining high for some time; severe nervous disturbance; adynamic symptoms; extensive complications affecting the heart or lungs; cerebral or spinal



meningitis; and deficiency of excretions. Chorea is considered to be a highly dangerous complication, especially when accompanied with dysphagia. The subcutaneous nodules in children, although unimportant in themselves, are said to be of serious import, because in several cases the associated heart disease has been found actively progressive.

**TREATMENT.**—The indications which need attention in managing a case of rheumatic fever may be stated as follows:—1. To study the general comfort of the patient, and to protect in every possible way from exposure. 2. To encourage free excretion. 3. To get rid of or to neutralize the poison in the blood, if this can be effected. 4. To attend to the joints. 5. To relieve other symptoms. 6. To use every means of preventing complications; and to treat these should they arise.

Of course it is desirable that a patient suffering from rheumatic fever should be restored to health as speedily as possible, but it is a matter of much greater consequence that the attack should be passed through without any permanent organic mischief being left behind, than that convalescence should be established within this or that number of days or weeks.

**1. General Management.**—The patient should be placed in a comfortable bed, between soft blankets, the limbs being made as comfortable as possible by means of pillows. A flannel shirt should be worn, which must be frequently changed when there is much sweating. It is important to avoid anything like a chill, hence the bed should be carefully protected from all draughts, and patients should not be allowed to throw off the bed-clothes, which they are much inclined to do. It is my practice in severe cases to wrap up all the middle-sized joints, whether affected or not, in cotton-wool, and also to place a layer of this material over the anterior surface of the chest. The front of the shirt may be cut in such a way as to form a flap covering the region of the heart, so that by drawing it aside this region may be examined without disturbing the patient, or unduly exposing the chest. The cotton-wool must be frequently renewed, the surface being dried before each fresh application is made.

The *diet* ought not to be too low, but should consist of a good quantity of beef-tea and milk, regularly administered. Lemonade or barley-water should be freely allowed as a drink, as well as ice to suck. Alcoholic stimulants are not required in ordinary practice as a rule; in hospital practice, however, patients often need a little wine or brandy, and sometimes considerable quantities are required, should there be any marked tendency towards debility and prostration. The bowels should be kept regularly opened.

**2. Therapeutic treatment.**—Observations have been made to prove that rheumatic fever runs an equally favourable course without medicines, as when these are administered. This is true with regard to many cases, if the measures already mentioned are carefully attended to. At the same time a tolerably extensive experience has convinced me that good results frequently follow the *alkaline treatment*. Whether this treatment shortens the course of the disease or not it is impossible to say, but it certainly appears to exercise a beneficial effect over the joint-affection, and my belief is that alkalies are decidedly useful in preventing and relieving the cardiac complications. The bicarbonate of potash is the preparation which I usually employ, either given in doses of  $\mathfrak{z}\frac{1}{2}$  to  $\mathfrak{z}$  iij every two to four hours; or  $\mathfrak{z}\frac{1}{2}$  or more being dis-



solved in a quart of barley-water, and administered as a drink during the twenty-four hours. Much larger doses are recommended by some practitioners; while others prefer certain vegetable salts, such as the citrate or tartrate, which possess the advantage that they can be made into a pleasant drink.

Opium is another remedy of great value. It is best given in the solid form, in doses of gr.  $\frac{1}{4}$ -i. every three or four hours, according to the indications present. This drug not only relieves pain, procures sleep, and sustains the nervous system; but it also calms the heart's action, and by thus inducing rest for this organ, tends to prevent cardiac inflammations. Morphia is also very useful, and it may be administered by subcutaneous injection.

Salicine, salicylic acid, or salicylate of soda are still most in fashion at present in the treatment of rheumatic fever, being administered in doses of gr. x-xx at intervals of from one to three hours. The conclusions which I have arrived at from personal experience of the employment of these drugs are:—1. That in a certain proportion of cases they are decidedly useful, in the way of checking the progress of the disease; lowering the temperature; and relieving the joint-symptoms. 2. That they are more effectual in mild than in severe cases. 3. That these drugs utterly fail in a good number of cases, and therefore can by no means be implicitly relied upon, and certainly do not possess the almost *specific* action in the treatment of acute rheumatism which has been attributed to them. 4. That they are of little or no value in the prevention or treatment of cardiac complications; and may prove highly dangerous, if the action of the heart is much weakened or embarrassed. 5. That in some instances in which these medicines have signally failed, alkalies have proved undoubtedly efficacious. Dr. Spencer of Bristol has advocated the use of salicylic acid combined with opium to relieve pain, or with tincture of aconite if there are signs of much articular or cardiac inflammation. He gives salicylic acid gr. xv-xx, with liquor ammoniæ citratis  $\zeta$  iij, every two to four hours; and tincture of aconite  $\mathfrak{m}$ ij-iij, or extract of opium gr.  $\frac{1}{2}$ -i, every two, three, or four hours. The treatment by salicylic acid and alkalies may be advantageously combined in some cases.

Some of the numerous other plans which have been advocated for the treatment of rheumatic fever require brief notice.

The salts of soda are preferred by some practitioners to those of potash. Nitrate of potash has been much used, in quantities of from  $\frac{3}{4}$  i to  $\bar{3}$  i in the twenty-four hours. Iodide of potassium, phosphate of ammonia, benzoates, and various other salts have also been tried. Lemon-juice has been much recommended, in quantities of from  $\bar{3}$  iij to  $\bar{3}$  xij or more in the twenty-four hours. Having seen it employed in several cases, it did not impress me at all favourably. Some authorities prefer quinine or cinchona bark in full doses; the former may be conveniently combined with alkalies, as advised by Dr. Garrod. Dr. Reynolds has found tincture of steel efficacious. Potassio-tartrate of iron has also been well spoken of. Many practitioners use colchicum, but this drug is of doubtful value in rheumatism. Certain remedies which act powerfully upon the heart have been employed, namely, aconite, digitalis, and especially veratrum viride. They may possibly have the effect of diminishing the tendency to cardiac inflammations, but require careful watching during their administration. Trimethylamine, tincture of ergot, tincture of cantharides, and tincture of actæa

racemosa are among the numerous drugs which have been recommended in the treatment of acute rheumatism.

The treatment of acute rheumatism by blisters has of late been much discussed, and demands brief notice. It has long been a practice to produce a blister over or near an individual joint, for the purpose of relieving severe pain, or aiding absorption of effusion. The late Dr. Todd recommended small blisters, from the size of a half-crown to a crown piece, to be repeated in rapid succession over different parts of the joint. Dr. Herbert Davies advocates the following specific plan of treating rheumatism:—Every joint, large and small, attacked by pain, heat, and swelling, is surrounded simultaneously by circular and broad strips of emplastrum lyttæ or zones of blistering fluid, which are to remain above or below the inflamed joints, until a full discharge of serum is obtained, such a discharge being favoured by the application of large linseed-meal poultices. Dr. Harkin, in accordance with his theory of the endocardial origin of rheumatism, advocates the treatment of this disease by the early application of a blister over the cardiac region, so as to produce a derivative or counter-irritant effect upon the inflamed endocardium, and thus to prevent any further mischief.

Various *baths* have been much advocated in the treatment of rheumatic fever, namely:—the hot-air or vapour bath; different forms of cold bath; the wet-pack; the hot blanket bath; or sponging the skin with cold or tepid water.

**3. Local treatment.**—If possible, it is desirable not to apply anything to the joints except cotton-wool, but in some instances the pain is so severe that local applications must be resorted to. As a rule warm anodyne fomentations, or poultices containing opium, belladonna, or their active principles, give most relief. To be of any use they must be put on very hot; be well covered with macintosh; and frequently changed. I have often tried the local application of an *alkaline* solution, as recommended by some authorities, but in my experience this measure has not appeared to be of much service, unless opium is added to the solution. The use of a solution of salicylate of soda in this way has been advocated. *Cold compresses* have been much commended. Possibly the application of two or three leeches might be serviceable in exceptional cases. Free blistering with liquor epispasticus certainly not unfrequently gives speedy relief as regards the pain. It is not uncommon for a joint to show a tendency to remain chronically affected after the general symptoms have subsided. If this happens, the application of a blister or of tincture of iodine may be first tried, but if a speedy effect is not produced, I have found much benefit from strapping the articulation carefully and efficiently with ammoniacum and mercury plaster. If there is much effusion, it has been suggested to tap the joint by means of the aspirateur.

**4. Symptomatic treatment.**—The management of most of the symptoms which may call for interference in cases of rheumatic fever has been sufficiently indicated in the preceding remarks. The occurrence of hyperpyrexia indicates immediate recourse to the employment of *cold*, as described under fever, with quinine in full doses internally, and the free administration of stimulants. The cases successfully treated in this way prove that patients may thus be saved when in an apparently hopeless condition.

**5. The treatment of the several complications of rheumatic fever**

will be considered in their respective chapters. At present I will only express the opinion that very rarely is any kind of bleeding justifiable for the inflammatory affections, and that calomel should never be given as an antiphlogistic. Opium must be used with caution if the lungs are involved; and free stimulation is then demanded. In cerebral or spinal meningitis ice should be applied locally.

6. Much care is needed during **convalescence** from rheumatic fever, warm clothing being worn, with flannel next the skin, and every form of exposure avoided. The diet should be improved gradually. The patient ought to be kept under observation until quite convalescent; and should have full instructions as to how to guard against future attacks. Should any of the joints remain chronically affected, the local applications already mentioned should be persevered with, and iodide of potassium administered internally. If the patient remains weak and anæmic, tincture of iron is of signal value, either given alone or combined with quinine. Should the heart be implicated, special care is needed, and the patient must rest as much as possible, so as not to make any extra call upon this organ. A change of air to a warm district is often highly beneficial after an attack of rheumatic fever.

## 2. CHRONIC ARTICULAR RHEUMATISM.

**SYMPTOMS.**—This affection is common among old persons, usually coming on gradually as age advances, but occasionally following one or more acute or subacute rheumatic attacks. The fibrous structures connected with, as well as around the articulations, become thickened and stiff. Hence the movements of the joints are impaired, and more or less dull aching pain is felt, which becomes worse at night, and during damp or cold weather. There are no particular objective signs; and the joints are not much altered in form as a rule. A form of chronic arthritis is, however, met with, especially in old persons, in which only one, or at most a few joints are affected, gradual changes of a permanent character being produced, and causing deformity. This condition has been specially termed “senile arthritis” or “rheumatic gout.” Probably it may be associated with chronic changes in the valves of the heart.

**TREATMENT.**—Patients suffering from chronic rheumatism should wear flannel next the skin; and should avoid exposure to wet or cold, as well as rapid changes of temperature. Baths of various kinds are useful in different cases, such as warm, vapour, hot-air, Turkish, cold, salt-water, sulphur, or alkaline baths. These may also be employed locally, and douches are often very serviceable. Much good frequently results from systematic daily friction of the affected joints with some stimulating and anodyne liniment, such as camphor liniment with laudanum, tincture of aconite, or belladonna; as well as from shampooing and kneading. Local counter-irritation by means of blisters or tincture of iodide is sometimes beneficial. Good results frequently ensue from effectually strapping an affected joint with some plaster, such as ammoniacum and mercury plaster, red plaster, or Burgundy pitch plaster. It is always well to keep the joints bandaged. Patients should be encouraged to take a moderate amount of exercise. The local use of the constant galvanic current has proved highly serviceable in many cases of chronic rheumatism.

The internal remedies which yield the best result are *tonics*, such as



quinine, cod-liver oil, or tincture of iron. Iodide of potassium with decoction of bark is also very useful; or the iodide may be combined with quinine. Sulphur, guaiacum, sarsaparilla, *actæa racemosa*, and many other drugs have been recommended as specifics in chronic rheumatism. It is often necessary to give some anodyne to relieve pain and to procure rest at night, for which purposes chloral is very efficacious. Various mineral waters are in many cases serviceable, such as those of Buxton, Bath, Harrogate, Cheltenham, and some of the German spas. Vichy water may also be useful. The *diet* should be nutritious and easily digestible. A small quantity of some alcoholic stimulant is generally beneficial for persons suffering from chronic rheumatism.

### 3. MUSCULAR AND TENDINOUS RHEUMATISM—MYALGIA.

The muscles are frequently the seat of a very painful affection, supposed to be of a rheumatic character, the fibrous structures being also probably involved.

**ÆTIOLOGY.**—The *exciting cause* of muscular rheumatism is either exposure to cold and wet, or to a direct draught of cold air; or excessive exercise, fatigue from being for a long time in the same posture, or strain of the muscles. It is usually met with in adults, and some forms of the complaint are most common among labouring men, while others most frequently affect weak and anæmic women. One attack predisposes to another. Gout seems to increase the tendency to muscular rheumatism.

**SYMPTOMS.**—In most cases the first attack of myalgia is *acute*, and it often comes on quite suddenly, or sets in during the night. The symptoms are pain in the affected muscles; some degree of tenderness; and considerable stiffness, with difficulty of movement, by which the pain is also much increased. The degree of suffering varies considerably, but it may be extremely severe; sometimes the pain is only felt on moving the affected muscles. In acute cases heat frequently increases it, and it is also worse at night, so that patients suffer most when in bed. Steady pressure gives relief in many cases. There may be a tendency to spasm of the muscles. The objective signs are referable to the fact that the patient keeps the involved structures as much at rest as possible. Pyrexia is absent, but slight constitutional disturbance may be observed, due to the pain and want of sleep. There is no tendency towards any cardiac inflammation.

In the *acute* form myalgia only lasts for a few days as a rule, but it often becomes *chronic*, or may be so from the first, and is liable to return again and again. When muscular rheumatism is chronic, heat generally relieves, while cold and damp weather aggravates the pain.

**VARIETIES.**—Muscular rheumatism may affect the voluntary muscles in any part of the body, and it is even believed that it may attack the involuntary muscles. Its most frequent and important varieties, however, are as follows:—

1. **Cephalodynia** or rheumatism of the scalp, which is attended by a form of headache, increased on moving the muscles of the scalp, with much soreness on pressure.

2. **Torticollis, wry-neck, or stiff-neck.**—This is a very common variety, involving the muscles of the neck, especially the sterno-mastoid. Usually it is limited to one side, towards which the neck is more or less

immovably twisted, great pain being experienced on attempting to turn the head in the opposite direction. The muscles at the back of the neck may be implicated.

3. **Omodynia, Scapulodynia, Dorsodynia.**—These forms are very commonly observed, especially among labouring men, the muscles about the shoulders or upper part of the back being affected.

4. **Pleurodynia or Rheumatism of the chest-walls.**—The muscles of the chest are very often implicated, especially those of the left side. The intercostals, pectorals, or serratus magnus may be involved, and it has appeared to me that the pain is frequently seated over the interdigitations of the serratus magnus with the external oblique. It is very commonly situated in the left infra-axillary region. It may be exceedingly intense, and is increased by any movement which brings the muscles into play. Respiratory movements are restrained on the affected side, and such acts as coughing or sneezing cause much distress. Not unfrequently the chief pain is localized in a point, and is of a catching character, while pressure on this point increases it, though diffused pressure with the palm may give relief. In other instances it alters its position from time to time. Pleurodynia simulates pleurisy, from which it can only be distinguished by careful physical examination. It often comes on as a consequence of severe cough, and both sides may then be affected; this is frequently observed in phthisical patients.

5. **Rheumatism of the abdominal walls** is an exceedingly painful complaint, and may be mistaken for peritonitis. It not unfrequently results from straining during cough.

6. **Lumbago.**—The mass of muscles, along with the fasciæ, which occupy the lumbar region, are among the most common seats of muscular rheumatism. It may set in with peculiar rapidity, and is usually very severe. Generally both sides are affected. There may be constant aching pain across the loins, but this is increased greatly on any attempt being made to bring the muscles into action, and it then becomes of a sharp, stabbing character. The patient keeps the spine quite stiff, and generally a little bent forward; any attempt to stand erect, or, still more, to get up from the sitting posture, greatly aggravates the suffering. Sometimes the patient cannot stir in bed. Pressure intensifies the pain considerably, and heat produces the same effect in many cases.

In addition to these varieties, muscular pains are common enough in the limbs in different parts. Sometimes cases are met with in outpatient practice, in which the plantar fascia and muscles seem to be specially involved. The diaphragm is occasionally the seat of a rheumatic affection, which causes much distress. The muscles of the eyeball may also be affected.

**TREATMENT.**—In *acute* cases of muscular rheumatism, the first indication is to keep the affected muscles at rest, and in many cases this is all that is required. I treat pleurodynia by firmly strapping the affected side by means of broad strips of plaster extending from mid-spine to mid-sternum (as will be more fully described under pleurisy), and this rarely fails to give complete relief. In lumbago also the application of a wide piece of emplastrum ferri firmly across the back, and over this a flannel bandage passing twice round the body, almost always affords great comfort. In acute cases warm anodyne fomentations are frequently useful, mustard cataplasms, or turpentine stupes. Dry heat does not generally answer well, as it increases the pain, but sometimes

if persevered in it does good. Gentle friction is often beneficial. In lumbago the subcutaneous injection of a small quantity of morphia generally affords considerable relief. Internally the administration of bicarbonate of potash, alone or with iodide of potassium, after free evacuation of the bowels, seems to answer best. An opiate may be necessary in order to relieve pain. Exciting free diaphoresis, by administering a warm drink and then wrapping the patient in blankets, or by the use of a vapour-bath, in some cases brings about a speedy cure. In rare instances it might be advisable to take away a little blood locally, either by leeching or cupping; or dry-cupping may be advantageously employed.

In *chronic* cases the internal remedies which do most good are iodide of potassium, quinine, and chloride of ammonium. Sulphur, guaiaicum, arsenic, mezereon, and various balsams or resins are much used; and likewise colchicum, if there is any gouty tendency. Tincture of *actæa racemosa* has been much vaunted in the treatment of lumbago. Flannel should be worn next the skin. Rest, pressure, cold compresses, friction with stimulating and anodyne liniments, the application of sinapisms or blisters, and local baths or douches with shampooing, constitute the chief local remedies which are found efficacious in different cases of chronic muscular rheumatism. The use of the continuous galvanic current is sometimes attended with marked success in chronic as well as in acute muscular rheumatism. It may be advisable to have recourse to subcutaneous injection of morphia daily for a few days. Acupuncture; the use of Corrigan's irons; or ironing over the affected part with a common flat-iron, a piece of brown paper being placed next the skin, have proved beneficial in some cases.

#### 4. GONORRHŒAL RHEUMATISM.

**ÆTIOLOGY AND PATHOLOGY.**—An affection of the joints is liable to set in during the course of gonorrhœa or gleet, as the result of cold, especially in young and plethoric subjects. As regards its pathology, Dr. Garrod considers that the complaint is allied to pyæmia; while, on the other hand, Mr. Hutchinson regards it as a true rheumatism, and believes that the predisposing cause is usually the inheritance of arthritic tendencies.

**SYMPTOMS.**—The knee-joint is most commonly attacked in gonorrhœal rheumatism; but the ankles, the joints of the feet, the hip-joint, or, indeed, any or all of the joints, are also not unfrequently implicated. There is considerable pain, with a tendency to much effusion and exudation, which gives rise to great tension and swelling, but suppuration does not occur, or only very rarely. The inflammation is very apt to recur, and to lead to permanent changes in the affected joints, which may remain stiff for a long time, with a crackling sensation on movement; or destruction of the cartilages and subsequent ankylosis may ensue. This complaint may become chronic. It is often accompanied with much constitutional disturbance, anæmia, and debility.

**TREATMENT.**—The affected joints must be kept at rest, and well fomented. When the knee-joint is implicated, the limb should be extended on a splint, as it is apt to become bent. In the acute stage Dover's powder must be given, in addition to the ordinary remedies for gonorrhœa. Afterwards iodide of potassium is useful, with *tonics* and *stimulants* if the patient is weak. Friction, shampooing, counter-irrita-



tion, and passive movement of the joint must be carefully practised when the acute symptoms have subsided. It might be useful to strap the articulation.

## 5. RHEUMATOID ARTHRITIS—ARTHRITIS DEFORMANS.—CHRONIC RHEUMATIC ARTHRITIS.

**ÆTIOLOGY AND PATHOLOGY.**—This is a curious form of joint-inflammation, which is liable to bring about great deformity. Dr. Garrod and other authorities regard it as a special and peculiar disease, quite distinct from ordinary rheumatism or gout; others consider it to be merely a form of chronic rheumatism; Dr. Dyce Duckworth defines it as a rheumatic branch of the basic arthritic stock or diathesis; while Mr. Hutchinson thinks that it is the result of inheritance of a tendency to both rheumatism and gout, in short, a kind of “rheumatic gout.” Dr. Ord believes that this disease, as well as other forms of joint disease, may be referable in some instances to a lesion of the spinal cord set up by peripheral irritation. Rheumatoid arthritis occurs in those who are debilitated, and whose circulation is languid. Most cases are met with between twenty and forty years of age, and among females. The complaint is chiefly observed among the poor, who live badly; but it may attack those who are in the best circumstances. It is often attributed to cold or damp, or sometimes to injury; but there may be no obvious cause.

**ANATOMICAL CHARACTERS.**—At first rheumatoid arthritis is attended by redness of the synovial membrane of the implicated joints, and increase of synovia. After a time the capsular ligament becomes greatly thickened, irregular proliferations forming; while the synovial fluid is much diminished. The internal ligaments may be destroyed, leading to dislocation. Fibrous bands form within the articulation, and cartilaginous or bony masses may grow there. The interarticular fibro-cartilages break down and disappear, as well as the cartilages covering the ends of the bones. The latter are found to be smooth and eburnated to a greater or less extent; being also enlarged, sometimes considerably; and either regular or more commonly very irregular at the margins, owing to the growth of osseous protuberances. There is no trace of any deposit of urates.

**SYMPTOMS.**—Rheumatoid arthritis may be *acute* or *chronic*. In the former case several joints are involved, but there is no erratic tendency, such as is observed in ordinary rheumatic fever. Pyrexia is present, but there is no profuse sweating, nor does the heart show any disposition to become implicated. In the *chronic* variety one joint is first affected, being the seat of a slight degree of pain and swelling, but it soon recovers; in a short time the same articulation is again attacked, and remains permanently altered, becoming gradually worse. Other joints are subsequently involved in succession, until all those of the limbs may finally be observed in various stages of change, and even the temporo-maxillary and upper cervical articulations may become implicated. They become rigid, motionless, and either permanently bent or extended; there is more or less distortion and nodulation, with contraction and wasting of the muscles, which is held by some authorities to be of nervous origin, as in wasting palsy; and the patient may be finally completely crippled. There may be signs of fluid in the larger joints. In some cases disloca-

tion takes place. The pain may be very considerable, being sometimes extremely severe, especially at night. There are no special constitutional symptoms, but the patients are often weak, anæmic, and wanting in tone. The hands are usually crippled before the feet. On the former also little nodular thickenings of the epiphyses of the phalanges—*digitorum nodi*—are sometimes met with, especially in connection with the terminal phalanges, which are generally supposed to be due to rheumatoid arthritis, but others believe them to be of a gouty nature.

Other parts are occasionally involved in this disease, namely, the sclerotic, the internal ear, or the larynx.

DIAGNOSIS.—Rheumatoid arthritis, if regarded as a separate disease, has to be distinguished from gout; from acute or chronic articular rheumatism; and from gonorrhœal rheumatism. The marked structural changes and deformity distinguish the complaint from ordinary chronic rheumatism, as well as from the gonorrhœal form, the latter also having a different history. The diagnosis from acute rheumatism and gout will be pointed out after the latter affection has been considered.

PROGNOSIS.—Acute cases of rheumatoid arthritis may recover if properly treated. If the disease is chronic and advanced, some improvement may be effected, but not much as a rule.

TREATMENT.—Patients suffering from rheumatoid arthritis need a sustaining plan of treatment, which must be persevered in. The general health requires every attention; the *diet* must be nutritious and easily assimilated, and wine or some other form of alcoholic stimulant is decidedly beneficial. Warm clothing, an equable climate, some pleasant occupation, and moderate exercise, with daily baths, are also to be commended.

Iron, quinine, and cod-liver oil are the most efficient internal remedies. Syrup of iodide of iron, iodide of potassium, arsenic, guaiacum, tincture of actæa, and many other medicines have been favourably spoken of in different cases. Strychnine or nux vomica may be tried if the muscles have wasted to any marked degree. Different mineral waters and baths are sometimes beneficial, such as those of Bath and Buxton.

In early cases local counter-irritation is decidedly useful, but it does not produce much effect after a time. In a case under my care free bathing with salt and water, followed by friction, seemed to do most good. Systematic strapping of the joints, friction with various liniments, shampooing, and careful passive movement may also be attended with benefit. Galvanism might prove of service in some cases.

---

## CHAPTER XXX.

### GOUT—PODAGRA.

ÆTIOLOGY AND PATHOLOGY.—Gout is a markedly hereditary complaint, and this is shown partly in its development at an early period of life. It is rarely met with under 30 years of age except in hereditary cases, and then it may affect even children. Most first attacks occur between 30 and 35, and the disease does not often commence late in life. Males suffer much more frequently than females. Those who are of sanguine temperament, plethoric, and corpulent are most subject to the disease;

but thin, nervous, and wiry persons are also liable to be attacked. It was formerly thought that high mental endowments predispose to gout, but this is not the case; prolonged mental labour, worry, and other causes which depress the nervous system have some effect. Individuals who work in lead are decidedly prone to become gouty, and, on the other hand, gouty people readily suffer from lead-poisoning. Gout prevails chiefly in cold and temperate climates, especially in those which are at the same time damp and changeable. Spring is the most favourable season for gouty attacks, and then autumn.

The combination of circumstances most conducive to the development of gout is over-indulgence in certain alcoholic drinks; with excessive consumption of food, especially animal food; and deficient exercise, with general luxurious habits. The disease is in this way frequently produced *de novo*, being also considerably more prevalent among the better classes of society; as well as among publicans, butchers, butlers, and others who have special opportunities of indulging in these habits. There is a form of "poor gout" which is met with in persons who drink much beer, while at the same time living badly as regards food, but a hereditary tendency may be discovered in some cases of this class.

Wines and malt liquors are much more conducive to the development of gout than spirits. Port-wine stands first, then come burgundy, madeira, marsala, and sherry. The lighter wines are not so hurtful. Rum is said to be a frequent cause of gout. Sweet and unfermented cider is also believed to produce the complaint.

Numerous views have been advanced at different times as to the *pathological cause* of gout, but that which is at present generally accepted recognizes the disease as a manifestation of the *lithic* or *uric acid diathesis*, *lithiasis*, or *lithæmia*, being due to excess of uric acid in the blood, in the form of urate of soda. The excess may be the consequence of undue formation of the acid; its imperfect oxidation and destruction in the system; the presence of an abnormal amount of other acids in the blood, which from their greater affinity combine with the alkalis in this fluid, and thus diminish its alkalinity, so that it cannot hold uric acid or urate of soda in solution; deficient elimination; or of two or more of these causes combined. Dr. Garrod has shown that during an acute attack of gout uric acid may be detected in abundance in the blood-serum; and in long-continued chronic cases it may be obtained from this fluid at any time. It is also found in the fluid of blisters; in inflammatory serous effusions; and in dropsical accumulations. Lead interferes with the excretion of uric acid by the kidneys. Functional disorders of the liver are believed by many to have considerable influence in developing gout, by causing excess of urates in the blood; while certain conditions of the kidneys, and especially particular forms of organic disease, must also be recognized as an important factor in not a few instances.

A view recently advanced by Dr. Ord, and supported by Dr. Bristowe, attributes gout to a special form of degeneration in certain of the fibroid textures of the body, characterized by the excessive formation of urate of soda in the implicated tissues, whence it is discharged into the blood, and deposited here and there, especially in those parts which are least well supplied with vessels and lymphatics.

The *exciting cause* of a gouty fit may not be evident, but it is often traceable to some definite event, such as exposure to cold or wet; slight



injury to a joint; excessive exertion and fatigue; undue mental labour; violent or depressing emotions, such as rage or grief; over-eating or drinking; or indulgence in indigestible food. The occurrence of a "fit" of gout is explained by the sudden or rapid increase of uric acid in the blood from some cause: and two opposite views are entertained to account for the joint-inflammation, namely, that it is set up by the acid, which acts as a local irritant; or that it is the result of an attempt on the part of the articular structures to eliminate and destroy the acid.

**ANATOMICAL CHARACTERS.**—Gout is characterized by the deposit of urate of soda from the blood in various structures, especially in those entering into the construction of the joints, and in such tissues as are not very vascular. This deposit is usually accompanied with signs of inflammation. In an *acute* case there is increased vascularity, with swelling, and effusion into and around the affected joint. Even in the first attack a deposit of urate probably takes place, and this increases with each subsequent paroxysm. In the early period only the metatarsophalangeal joint of the great toe is usually affected; but subsequently other articulations become involved, so that almost the whole of them may be finally implicated. The deposit first occurs in the superficial part of the cartilages, in the form of fine crystalline needles or prisms, forming a more or less close network, and presenting different degrees of opacity. Subsequently the fibro-cartilages, ligaments, and synovial membrane become involved, the entire surface being rendered more or less irregular, and covered with white, chalky-looking deposits, consisting of urate of soda. The synovial fluid may also contain crystals of the same substance. Owing to the infiltration of the ligaments, the articulations become stiffened or fixed. In long-continued cases the joints become ultimately greatly distorted and nodulated, and the skin over them may be destroyed, exposing the chalky-looking masses, and leading to unhealthy suppuration and ulceration. The periosteum and various bursæ may also be implicated; and some authorities believe that even bone itself may become affected.

Deposits are often found in various other parts, such as the external ear, eyelids, nose, or larynx.

The kidneys become the seat of certain morbid changes, which probably begin at an early period. At first a deposit of urates forms within the renal tubuli, which afterwards extends into the intertubular tissue. White streaks are seen in the direction of the tubuli of the pyramids, and at the extremities of the papillæ. Ultimately the organs become greatly contracted and indurated, at the same time being the seat of extensive deposit; in short, a form of "granular kidney" is produced, which will be more fully described in its appropriate chapter.

Acute or chronic lesions of several other structures may be associated with gout, but these will be mentioned presently, and do not call for any special description here.

**SYMPTOMS.**—Cases of gout are divided into two classes, according as the symptoms are associated with the joints—**Regular** or **Articular gout**; or with one of the internal organs, or some structure other than the joints—**Irregular** or **Misplaced gout**.

1. **Regular or Articular Gout.**—At first this is an *acute* affection, but after a time it tends to become *chronic*.

a. *Acute gout.*—The first attack often comes on without any premonitory warning, but not always, and prodromata usually indicate the

approach of subsequent fits. The most frequent precursory signs are derangements of the alimentary canal, with heartburn, acidity, and signs of portal congestion; palpitation or disordered action of the heart; nervous disturbances, such as headache, giddiness, disturbed vision, drowsiness and heaviness, irritability of temper, languor, restless sleep with unpleasant dreams, starting of the limbs, or cramps in the calves of the legs or in other parts; asthmatic attacks, or sudden laryngeal congestion with obstructed breathing; profuse sweats; and changes in the urine, this secretion either becoming scanty and depositing much sediment, or being very abundant and watery. Dr. Garrod has found albuminuria in several instances some days before a fit of articular gout, which has continued during the attack, but has afterwards completely passed away. In some cases the patient feels unusually well, both mentally and bodily, before the occurrence of an attack. This may also be preceded by some uncomfortable local sensations in connection with particular joints.

The onset of the attack usually takes place during the night, especially from 2 to 5 A.M. In the large majority of cases the *metatarsophalangeal articulation of the great toe* is the joint which is first affected, generally on one side, especially the right, but sometimes on both, or they may be attacked in succession. In some instances this joint is alone implicated during several attacks, but in most cases other articulations soon become involved, a number of them being affected in the course of a gouty fit, either simultaneously or in succession, though the complaint tends to be limited for a considerable time to the smaller joints of the feet and hands. Exceptionally the knee or ankle is first affected, but very rarely the larger articulations of the upper extremity.

*Characters of the joint-affection.*—The subjective sensations are extremely severe. The pain rapidly increases until it becomes agonizing and unbearable, being described as burning, tearing, plunging, boring, or piercing. There is exquisite tenderness, so that the slightest touch cannot be endured. These sensations are usually much worse during the night, and remit during the day. Soon the joint becomes much swollen, owing to effusion of fluid into its interior, the skin over it being red, tense, hot, and shining; after a while there is considerable surrounding oedema, pitting on pressure, some relief accompanying these objective signs. The superficial veins are also enlarged. As the inflammation subsides, desquamation of the cuticle takes place, and troublesome itching is often experienced. Oedema may continue for some time.

It is in early attacks, and in full-blooded persons, that the phenomena just described are most marked. Subsequently the pain and other symptoms become greatly diminished; and in weakly individuals, especially females, the characteristic features of the joint-affection are not nearly so pronounced.

*Constitutional symptoms* of greater or less severity are present during a paroxysm of gout, being in proportion to the intensity of the local symptoms, and to the number of joints involved. Chills or even distinct rigors are felt at the outset, followed by pyrexia, usually attended with perspiration, but not excessive. Marked remissions are generally observed towards morning. The urine is very scanty and dark, and deposits urates abundantly, of variable colour; uric acid is actually deficient, though relatively in excess. The patient is very restless and



sleepless, irritable, and not uncommonly suffers from cramps in the legs. The digestive and hepatic functions are much deranged. At the close of the paroxysm there may be critical perspiration, diarrhœa, or an abundant sediment of urates in the urine.

The *duration* of a gouty fit varies from four or five days to several weeks, in the latter case being interrupted by remissions or intermissions. It becomes more prolonged as the case progresses. *Recurrence* is a characteristic feature of gout, though it does not invariably happen. At first the attacks generally come on once a year, in the spring; then twice, in spring and autumn; and afterwards at more frequent intervals.

Some patients do not recover their usual health for a considerable time after a fit of gout; others are much the better for it. In a short time the affected joints become the seat of permanent changes.

*b. Chronic gout.*—This term is applied to those cases in which the joints have become permanently much altered in structure and form; and where the attacks are frequent, as well as chronic in duration and intensity, indeed in some instances being never altogether absent; while at the same time other structures besides the joints are involved. The articulations become stiff, immovable, enlarged, nodulated, and deformed, owing to the extensive deposits of urates in their structures. The skin appears blue and congested over them, the veins being enlarged. Finally it may rupture, masses of urate being exposed, named *chalk-stones* or *tophi*, which may be discharged as a yellowish-white substance; or suppuration and unhealthy ulceration may be set up.

In course of time other structures are affected, such as tendons, bursæ, the periosteum covering the shafts of bones, aponeuroses, and the sheaths of muscles. Gouty abscesses may form in connection with these deposits. Small deposits may also be observed in the helix of the external ear, the cartilages of the eyelids, the nose, and the sclerotic. At first they are liquid, and when punctured a whitish matter is discharged, containing abundant crystals of urates; ultimately they become solid, and form little hard nodules or beads. These gouty concretions sometimes set up inflammation, and hence they are not always of uniform composition; a little phosphate of lime may be mixed up with the urates.

Patients suffering from chronic gout are almost always weak and wanting in tone. They may have a pale and sallow aspect; or are sometimes plethoric, but flabby-looking. They suffer from various disorders of digestion; as well as from disturbances about the heart, in the way of palpitation or irregular action; being also irritable or depressed and restless, and subject to cramps, twitchings, tic douloureux, and other nervous disorders. A peculiar grinding of the teeth has been noticed in gouty subjects. From time to time there may be a little feverishness. The urine is generally pale, of light specific gravity, deficient in solids, and often slightly albuminous; it sometimes contains casts. In some cases of gout the nose is subject to daily paroxysms of heat and redness.

**2. Irregular,\* non-articular, misplaced, suppressed, or retrocedent gout.**—Some of the phenomena which have been mentioned as being associated with gout, as well as the deposition of urates in other structures besides the joints, may be regarded as strictly belonging to the category now to be considered. The terms given above apply, indeed, to all conditions due to the gouty state which are not connected with the articulations, though they are often more particularly associated



with certain internal and special manifestations of the diathesis. Irregular gout may occur when there is no sign of any articular affection, but usually the joints are more or less involved. The severity of the symptoms is often in an inverse ratio to that of the joint-affection, and the two classes of symptoms may exhibit a remarkable tendency to alternation. *Suppressed gout* is an expression implying that internal symptoms are due to a want of, or imperfect development of, the external phenomena; while *retrocedent gout* means that from some cause or other the joint-inflammation suddenly or rapidly subsides, and marked irregular symptoms are simultaneously developed, a kind of *metastasis* being supposed to take place, but probably the phenomena are really due to elimination of uric acid being checked. It will only be practicable here to give a brief summary of the chief conditions usually regarded as manifestations of irregular gout, premising that they may be either acute or chronic, and of the nature of functional disorders, inflammatory affections, or chronic organic lesions of a special kind, many of them being attended with the deposit of urates in different structures. They may be thus arranged:—

a. *Nervous disorders*, such as attacks of severe headache and vertigo; mental disturbance, the intellect being impaired, or delirium or acute mania setting in as the result of retrocedent gout; epileptiform seizures; various forms of neuralgia, sometimes very acute, and probably due to neuritis in some instances; morbid sensations of other kinds; startings of the limbs, severe cramps, or local paralysis. It is believed by some authorities that gout may set up a form of meningitis. Apoplexy is common among gouty subjects, but this is probably due to the fact that the vessels are generally diseased in such persons; a temporary apoplectic attack may be due to cerebral congestion. A gouty form of sciatica has been described, due to implication of the sheath of the sciatic nerve, which may extend up to the spinal cord and its membranes.

b. *Derangements of the digestive organs*. The stomach is one of the organs most commonly affected in gouty persons. There may be actual gastritis, with severe bilious vomiting; or merely a neurotic disturbance, indicated by sudden spasmodic pain, of great intensity, but relieved by pressure, with a sense of oppression, much anxiety and distress, and sometimes marked prostration or collapse. Dysphagia is occasionally complained of. In some cases enteralgia or intestinal colic and diarrhoea are associated with gout; or there may be acute enteritis. Signs of hepatic disorder are often evident; and fatty liver is frequently developed in gout.

c. *Vascular disorders*. There is no true gouty cardiac inflammation, such as is met with in rheumatism, but the organic changes which may be set up in connection with the heart include white patches on the pericardium; chronic valvulitis and degeneration of the valves; and hypertrophy, followed by fatty degeneration. This organ is also liable to mere functional disorder. The vessels often become atheromatous, or arterio-capillary fibrosis is set up, with hypertrophy of the muscular coat of the small arteries. Symptoms and physical signs indicative of these conditions will be developed; and the cardiac action is liable to become gravely disordered at times, as evidenced by severe palpitation, or it may be very weak, or very slow or rapid, or irregular or intermittent; while at the same time the pulse is feeble and small, and there may be a tendency to syncope or collapse. Painful or disagreeable sensations are often experienced at the same time over the

cardiac region, accompanied with a feeling of constriction, dyspnœa, and much anxiety ; while true anginal attacks are not uncommon.

*d. Pulmonary affections.* A form of asthma is often met with in gouty patients, and also dry bronchial catarrh, which is attended with much cough. Emphysema is of frequent occurrence in these subjects. Pulmonary congestion may arise ; but pneumonia is very uncommon, and there is no special form of gouty pneumonia.

*e. Urinary complaints.* The abnormal conditions of the urine in gout have already been sufficiently alluded to. When renal changes are developed, the urine presents corresponding morbid characters. Chronic cystitis and urethritis are not unfrequent, especially among old people. Gouty persons often suffer from gravel and calculus ; and oxaluria is observed in many cases.

*f. Cutaneous affections.* The chief skin-diseases which may be associated with gout are acute or chronic eczema, erythema, urticaria, psoriasis, local or general prurigo, and acne.

*g. Miscellaneous complaints.* Under this head may be mentioned lumbago and other forms of muscular rheumatism. Mr. Hutchinson thinks that many obscure joint-affections in young persons are due to hereditary gout ; and he has also described a peculiar form of iritis, coming on insidiously, and almost painlessly, and ending in destruction of the eye.

DIAGNOSIS.—The chief points of difference which are usually recognized between gout, rheumatism, and rheumatoid arthritis are indicated in the following table :—

	GOUT.	RHEUMATISM.	RHEUMATOID ARTHRITIS.
1. <i>Hereditariness.</i> . .	Very marked.	Less marked.	Doubtful
2. <i>Social position of patient</i> . . . . .	Among the better classes ; or those who over-feed and drink.	Among the poorer and hard-working classes chiefly.	Among the poor and ill fed most commonly.
3. <i>Age</i> . . . . .	Very rare in early life. Most first attacks from 30 to 35.	Common in early life ; chiefly from 16 to 20.	Usually from 20 to 40.
4. <i>Sex</i> . . . . .	Much more prevalent among males.	More among males, but to less degree.	Chiefly among females.
5. <i>Mode of onset.</i> . .	Often no obvious cause of first attack ; this is frequently preceded by digestive derangements, and other premonitory symptoms.	Usually follows an obvious cause, viz., exposure to cold ; and frequently no precursory symptoms are observed.	Exciting cause may be evident or not. Preceded by much exhaustion and debility in some cases.
6. <i>Joint-affection</i> . .	The smaller joints are most affected, especially the great toe ; no erratic tendency. Local symptoms very intense, with much œdema, a shining appearance of the skin, enlarged veins, and desquamation after the attack. In time permanent enlargement of the joints, with distortion, and deposit of urates.	Medium-sized joints most involved ; erratic, several joints being usually attacked in succession. Symptoms less severe, and less œdema present than in gout ; no enlargement of veins or desquamation.	All joints equally attacked ; not erratic. Symptoms are not severe, but tend to long continuance. Ultimately deformity is produced, but there is no deposit of urates in joints.

	GOUT.	RHEUMATISM.	RHEUMATOID ARTHRITIS.
7. <i>General symptoms</i> . . .	Pyrexia, variable in amount; much constitutional disturbance; considerable morning remissions.	Variable degree of pyrexia, but usually considerable; more continuous than in gout.	Only slight pyrexia. Symptoms of debility and want of tone.
8. <i>Perspiration</i> . . .	No special characters.	Very profuse and acid	No acid sweats.
9. <i>Course, duration, and progress</i> . . . . .	Early paroxysm of short duration; great tendency to recurrence, and to periodicity.	Attack of much longer duration; much less tendency to recurrence; not periodic.	Subacute and gradually progressive; often no complete intermission; not periodic.
10. <i>Complications</i> . .	Affects especially the stomach, brain, and kidneys; also gives rise to nervous disturbance of the heart, but not to inflammatory affections.	Very liable to cardiac inflammations; also to pulmonary inflammations.	Nothing in heart or other organs.
11. <i>Uric acid in blood</i> .	Present.	None.	None.
12. <i>Tophi in auricle, &amp;c.</i>	Present in many cases.	None.	None.
13. <i>Urine</i> . . . . .	Deficiency of urates before and during the fit, followed by excess; albuminuria common; may have casts, indicating kidney-disease.	Febrile; sometimes slight albuminuria.	No special characters.

Although in the great majority of cases the diagnosis between these several affections can be readily made, by attending to the points indicated in the foregoing table, yet it must be remembered that cases do occur in which the distinctions are by no means so marked, and where it becomes difficult to make out the precise nature of the complaint. In some instances undoubtedly there is a combination of gout and rheumatism, constituting true *rheumatic gout*.

**PROGNOSIS.**—Acute gout is rarely immediately fatal, but when the internal organs are involved there is considerable danger. The complaint is always liable to return, but much depends upon the mode of living which the patient adopts. The future prognosis is worse in proportion to the youth of the patient; to the degree of hereditary predisposition; and to the frequency of the attacks. Chronic gout decidedly tends to shorten life. The most serious signs are those indicating advanced renal disease, with non-elimination of uric acid. Gout materially diminishes the chances of recovery from acute diseases and injuries.

**TREATMENT.**—1. **During the paroxysm.**—It is well to give a brisk purgative at the commencement of a fit of gout, such as a calomel and colocynth pill, followed by a black draught. Colchicum is the specific remedy for this affection, but it must be given with due care. From  $\mathfrak{m}$  x-xx of vinum colchici should be administered every six or eight hours, and it may be combined with the bicarbonate or some vegetable salt of potash, freely diluted; or with carbonate of lithia.

A free action of the skin should be kept up by the use of *diaphoretic*



drinks; or the vapour or hot-air bath might be advantageously employed in some cases. A low *diet* is generally indicated at first, which should be gradually improved as the attack passes off, but it may be necessary to permit weak patients to have a good quantity of liquid nourishment throughout. As a rule all stimulants ought to be withdrawn, especially when the patient is young, but sometimes it is advisable to allow a certain quantity of brandy, well-diluted. If there is much pain and restlessness, opium must be given at night, in the form of Dover's powder; or subcutaneous injection of morphia may be called for.

As regards *local treatment*, rest is of course essential, and an elevated position should be adopted for the affected parts. These should be wrapped up in flannel or in cotton-wool, covered with oil-silk. If the local symptoms are very severe, warm fomentations or poultices containing opium, or localized steaming may be tried; or it may be advisable to apply *anodynes*, such as belladonna liniment, tincture of aconite, or a solution of atropine or morphia. Local removal of blood is extremely rarely called for; but sometimes the application of a blister is useful. As the inflammation subsides, slight pressure by means of a bandage or an elastic apparatus is often serviceable, in order to get rid of the œdema and other conditions which are liable to remain; gentle friction may also be employed with advantage.

*Irregular gout*.—Should gout attack internal organs, it is requisite to endeavour to excite inflammation in the joints, by means of friction, heat, or sinapisms. In the neurotic affections, opium with *stimulants*, such as ammonia, ether, camphor, and musk, are the remedies indicated; as well as alcoholic stimulants. External heat and sinapisms are also of much service in these conditions. Inflammatory complications may require the application of a few leeches or of a blister, but in most cases gouty patients will not bear much depletion.

**2. During the intervals.**—There is no disease in the management of which a careful study of the patient, and of everything connected with him, is more necessary than in the case of gout. In the early stage a practical cure may often be effected, especially in acquired cases, in so far that freedom from further attacks can be ensured, provided that due attention is paid to certain rules. Even when the gouty diathesis is hereditary, it may be prevented from becoming actively developed, or at all events the complaint can be put off until a later period in life, by the exercise of proper care and precautions.

*Diet* requires strict regulation, but must necessarily vary much in different subjects. The food should be nutritious and easily digestible, consisting of a due proportion of animal and vegetable constituents, but the consumption of nitrogenous and saccharine substances must be limited. Moderation in eating, and regularity of meals, are important points to be attended to. Tea and coffee may be allowed to a limited extent; also soft and stewed fruits, provided they do not contain much sugar; but pastry ought to be avoided. It has been suggested that common salt should be entirely avoided by gouty persons, and decided benefit seems to have followed strict attention to this matter. Abundance of pure water is of great value as a drink in cases of gout.

The use of *alcoholic stimulants* is a matter calling for serious consideration. For young persons who are hereditarily predisposed to gout, total abstinence is to be decidedly recommended, as well as for other gouty subjects when there is no obvious necessity for stimulants.

The nature and quantity of any stimulant which is permitted should be always definitely stated. Malt liquors and all strong wines ought to be rigidly denied. Lighter wines of good quality, such as claret, hock, moselle, or chablis, may be allowed in moderation; or in some cases a small amount of dry sherry. Small quantities of spirits, freely diluted, are also permissible in some instances, either brandy, whisky, or gin, and these may be taken mixed with good potass- or lithia-water, but soda-water must be avoided. When a change is being made as regards the diet and drink of a gouty patient, it should be carried out gradually, and not abruptly.

With respect to *general hygienic management*, the following are the chief matters which need to be enforced:—Moderate daily exercise in the open air, and the avoidance of sedentary habits; proper ventilation; daily bathing, followed by friction, and the occasional use of a warm, Turkish, or tepid salt-water bath; the wearing of warm clothing, with flannel next the skin; abstinence from undue mental labour, and freedom from all sources of worry or irritation; the avoidance of late hours at night, of heated and ill-ventilated rooms, and of lying in bed late in the morning; if possible, residence in a warm and equable climate, but if this is not practicable, protection against all sudden alterations of temperature or exposure to wet and cold, with a change to a favourable climate during the winter months. Wet-packing and other forms of hydropathic treatment are decidedly useful in some cases of gout.

The digestive organs must be attended to. The bowels should be made to act daily, a mild *aperient* being given if necessary. Much mercury is injurious, but there is no harm in giving a dose of blue-pill or calomel occasionally. It may be requisite to administer remedies with the view of improving digestion; or of relieving portal congestion.

*Mineral waters* are often useful, but must be duly regulated in their administration. Those which are considered most beneficial include the waters of Buxton and Bath in this country; and various German and other foreign waters, especially those of Vichy, Wiesbaden, Baden-Baden, Aix-la-Chapelle, Carlsbad, Kissengen, Marienbad, Homburg, Ems, and Wildbad.

It is scarcely necessary to add that every cause should be avoided which is known to have any tendency to bring on an acute attack of gout.

*Therapeutic treatment.*—Colchicum is a valuable medicine even in the intervals between the attacks of gout, when carefully administered; it is best given in the form of extract at night, with extract of henbane or gentian; or a few minims of vinum colchici may be added to other medicines. In a considerable proportion of cases of this disease *tonics* are indicated. Quinine, or tincture or infusion of cinchona; some mild preparation of iron, such as the ammonio-citrate; arsenic; mineral acids; and bitter infusions are frequently serviceable. Guaiacum and ammoniacum are strongly recommended for asthenic gout in old persons. Iodide and bromide of potassium are also beneficial in some cases. Alkaline salts and those of the alkaline earths are valuable in many cases, given freely diluted and on an empty stomach, especially the carbonates and phosphates of potash, magnesia, or lithia. The salts of lithia have been specially advocated, either the carbonate (gr. v-x), or the citrate (gr. viii-xii). Most of the mineral waters owe their efficacy to

some of these salts being dissolved in them. Carbonate of alumina, benzoate of ammonia, phosphate of soda and ammonia, lime-juice, and various other medicines have also been recommended in the treatment of chronic gout.

*Local treatment.*—When the joints become much altered in gout, but little can be done locally. I have, however, seen much benefit derived, in cases not far advanced, from the continued application of wet bandages over the affected joints. Friction, shampooing, and pressure by means of strapping might be carefully employed. Ulceration requires the application of some simple dressing. A dressing consisting of a solution of a potash or lithia salt has been favourably spoken of for this condition.

## CHAPTER XXXI.

### SCORBUTUS—SCURVY.

*ÆTIOLOGY.*—Scurvy is a distinct and peculiar disease, though the term is not uncommonly ignorantly applied to a variety of skin-affections. It is met with chiefly among seafaring men, being consequently by far most commonly observed on board ship, or in the hospitals of seaport towns. During the early part of my connection with the Liverpool Northern Hospital a large number of cases were admitted within the year, but subsequently they became less numerous, owing to better regulations being carried out for the prevention of the disease.

Scurvy has been attributed to many causes, among others to the use of salt meat or of putrid meat and bad water, and to imperfect hygienic conditions; my own experience, however, would lead me to agree with those who believe that the complaint is almost always due to the *want of fresh vegetable diet*, or of some appropriate substitute. This was the invariable cause in the seamen in whom I observed the disease; and in some cases of *land-scurvy* which have come under my notice, the complaint could be distinctly traced to the same cause. Scurvy seems, however, to have broken out under circumstances which indicate that it might possibly arise in other ways. During the siege of Paris numerous cases of the disease occurred, which were attributed to insufficient food and bad hygienic conditions.

Advanced age; a cold and damp climate or season; exposure; fatigue; and despondency, have been set down as *predisposing causes* of scurvy.

*ANATOMICAL CHARACTERS.*—In fatal cases of scurvy much emaciation is usually observed, with œdema of the legs. The blood is very dark and liquid, and its colouring matter stains the tissues; while the corpuscles are more or less altered or dissolved. Extravasations are met with in the subcutaneous tissue, as well as between, or sometimes even within the muscles, and they are often firmly coagulated or partially organized. Serous and synovial effusions are common, especially pericardial effusion, these frequently containing an admixture of blood. The organs are loaded with blood, and present ecchymoses and extravasations, being also relaxed and softened. The heart exhibits ecchymoses, and its muscular tissue is the seat of granular or fatty change. This



change has also been observed in the secreting cells of the liver and kidneys. Ecchymoses may be seen under the serous membranes; while the mucous membranes are red and swollen in some parts, and also present blood-stains. Granular fatty degeneration of the voluntary muscles is said to occur. The appearances which are characteristic of scurvy during life continue to be visible after death.

**PATHOLOGY.**—The pathology of scurvy is by no means settled. In what manner deficient consumption of vegetable matters acts in producing the disease is still a disputed question, and all that can be positively stated is that this leads to some alteration in the composition of the blood. This change has been attributed to a want of potash salts, of vegetable albumen, of organic acids, and of various other constituents of vegetable substances.

Dr. Raffe has investigated the pathology of scurvy, and has drawn the following conclusions from his observations:—

1. That the primary change that occurs in scurvy is a *chemical alteration* in the quality of the blood.

2. That this chemical alteration, as far as can be judged from inferences drawn from the analysis of urine in patients suffering from scurvy, and analysis of “scurbutic” and “antiscorbutic” diets, points to a *diminution of the alkalinity of the blood*.

3. That this *diminution of alkalinity* is produced in the first instance (physiologically) by an increase of acid salts (chiefly urates) in the blood, and finally (pathologically) by the withdrawal of salts having an alkaline reaction (chiefly alkaline carbonates).

4. That this *diminution of the alkalinity* of the blood finally produces the same results in scurvy patients as happens in animals when attempts are made to reduce the alkalinity of the body (either by injecting acids into the blood or feeding with acid salts), namely, dissolution of the blood-corpuscles, ecchymoses, and blood-stains on mucous surfaces, and fatty degeneration of the muscles of the heart, the muscles generally, and the secreting cells of the liver and kidney.

**SYMPTOMS.**—Scurvy sets in gradually and insidiously, and cases are met with of all grades of severity, but in a well-marked example the symptoms are very striking. The patient presents a peculiar unhealthy aspect, the face being sallow and of a dirty-yellowish hue, combined with puffiness about the eyelids, and anæmia, the latter being well seen in the mucous membranes. There is more or less emaciation, but wasting may not be very marked even in bad cases. The subjective sensations are those of languor, debility, fatigue, shortness of breath, faintness, pains and soreness in the limbs, mental depression and despondency. These symptoms vary in degree, sometimes amounting to complete prostration, with a tendency to sudden syncope, which may even prove fatal. Scurbutic patients often exhale a peculiar odour from the skin.

The mouth affords certain characteristic signs in scurvy. The gums are either more or less swollen, turgid, dark, and spongy, sometimes reaching to, or even much beyond the level of the teeth; or they become ulcerated and gangrenous, dropping off in masses, and exposing the teeth or jaws. From the first they readily bleed, and after a time blood oozes constantly from them. The teeth feel very tender, so that chewing becomes difficult or impossible; and they also soon become loosened, or may even drop out. Necrosis of the jaws occasionally occurs. The breath has a peculiar and excessively foul odour.

The legs present small purple spots, corresponding to and being the result of extravasations of blood into the hair-follicles. They are chiefly observed below the knees, but are not uncommon, though less abundant, on the thighs, being rarely seen on the abdomen or arms. In addition to these petechial spots, more or less extensive and irregular ecchymotic patches are visible, presenting various hues, according to the changes which the colouring matter of the extravasated blood has undergone. Brawny indurations can be felt, due to deeper extravasations, especially in the hams and calves, and these are often very painful and tender. Œdema of the feet and legs, and desquamation of the cuticle, are common symptoms. There is much stiffness and pain on movement; while a constant feeling of aching and contusion in the legs is experienced.

In some cases hæmorrhages take place from various mucous surfaces. Ulcers are occasionally formed; or old ulcers may break out anew, or assume an unhealthy aspect, being covered with large bleeding granulations. It is said that old fractures sometimes become disunited, that bones soften, or that epiphyses become separated.

The alimentary canal is usually disordered. The appetite is impaired as a rule, though sometimes patients feel inclined for food, but cannot chew it, and certainly the appetite generally returns as soon as they are able to masticate. In severe cases there may be nausea and vomiting. Obstinate constipation is the rule, but occasionally the bowels are relaxed, and the stools may contain blood, or actual dysentery may be present as a complication. There is no pyrexia usually, and the temperature may be below the normal. The pulse is infrequent, weak, and small. The patient often passes restless nights. The urine is deficient in quantity, dark-coloured in some cases, and tends to decompose rapidly. Urea, phosphates, and potash are deficient. Occasionally blood is mixed with the urine.

Morbid conditions of the blood have been described in scurvy, but there is no certainty about this matter. Garrod states that potash is much diminished in quantity. Dr. Leven affirmed that in cases in which he examined the blood during the siege of Paris he found fibrin in excess, and corpuscles diminished by one-half.

**DIAGNOSIS.**—The only disease likely to be confounded with scurvy is purpura; the diagnosis will be pointed out after the latter has been considered.

**PROGNOSIS.**—All the cases of *sea-scurvy* which came under my notice rapidly recovered, with one exception, and in that case death resulted from an accidental complication, namely, apoplexy. Therefore the prognosis is highly favourable, if proper treatment can be adopted. In my experience sporadic cases of *land-scurvy* do not seem to be so easily cured, and in one instance acute gastro-enteritis and pneumonia set in without any evident cause, ending in speedy death; while in another no treatment seemed to produce any effect, and the patient sank from the direct consequences of the disease, death being preceded by high pyrexia.

**TREATMENT.**—*Sea-scurvy* is one of the most satisfactory diseases to treat, speedy recovery being brought about in the great majority of cases, provided the necessary remedies can be obtained, namely, plenty of fresh, soft, and succulent vegetables; with from  $\bar{3}$  iv to  $\bar{3}$  viij of lime- or lemon-juice daily. Potatoes and cabbages constitute the best forms of vegetable food. Oranges, lemons, citrons, and other fruits of this class are also most valuable. Water-cress, garden-cress, mustard, scurvy-

grass, Sauer-kraut, spruce, fir, and various other vegetable growths have obtained repute in the treatment of scurvy, and might be tried if the more reliable remedies cannot be procured. It is stated that vegetables act best when uncooked, but ordinarily it is quite unnecessary to give them in this condition.

Liquid nutritious food is needed, such as beef-tea and milk, often in considerable quantity, and as soon as the patient can chew, meat should be allowed. Alcoholic stimulants are frequently indicated, but should be given carefully, and in small quantities. In a few days it is advisable to administer some *tonic*, such as quinine with tincture of iron, which aids recovery by exciting the appetite, strengthening the patient, and improving the quality of the blood.

The mouth must at first be frequently washed out with some dilute *antiseptic*, of which one of the best is Condy's fluid. At a later period a mild *astringent*, such as a solution of alum, may be used. Constipation is generally a troublesome symptom, and is best overcome by the use of enemata. Fomentations may be applied to the painful swellings in the legs. If serious hæmorrhage occurs, *astringents* must be given. Ulcers may be dressed with lime-juice, but they improve rapidly under its internal administration.

Many remedies have been recommended in the treatment of scurvy, such as salts of potash; solution of various vegetable acids, especially citric; and phosphoric acid. In my experience these have all proved quite unreliable. Raw meat and seal's flesh have been employed, it is said, successfully for the cure of this disease.

The treatment of land-scurvy must be conducted on the same principles as that of sea-scurvy, but it is much more difficult to cure, the symptoms often lasting a considerable time, and, as already stated, death may occur from this complaint in spite of all treatment.

The *prevention* of scurvy is a most important matter in connection with those persons who lead a seafaring life. There can be no question but that the disease may be completely prevented by the use either of fresh vegetables, preserved vegetables, or of proper lime- or lemon-juice, which should be served out daily. Many of the specimens of so-called lime-juice used on board ship are worthless; often only a solution of citric acid is used, which decidedly does not prevent the disease. The free employment of vinegar, and of the vegetable salts of potash, has been recommended. It is important to attend to all hygienic and other measures for maintaining the general health of the sailors; and they must also avoid undue exposure.

---



## CHAPTER XXXII.

## PURPURA.

**ÆTIOLOGY AND PATHOLOGY.**—Purpura is due to a peculiar unhealthy condition of the blood and tissues, which may probably be originated in many ways, but its causes are not at all clearly defined. The disease is quite distinct from scurvy, although the purpuric condition is one element in this complaint. It may depend upon unhealthy hygienic conditions, deficiency of proper food (? vegetables), or intemperance and other lowering agencies; or it may arise in connection with various acute fevers (typhus, small-pox, measles, &c.), or in the course of many chronic affections, such as albuminoid disease, syphilis, cancer, Bright's disease, cirrhosis of the liver, or skin-diseases, while it often accompanies jaundice. Sometimes the condition follows the administration of iodide of potassium. Amenorrhœa has been set down as a cause. Strictly the term *purpura*, as applied to a distinct disease, is only associated with cases where the symptoms are independent of any obvious local lesion, or general specific affection. Purpura may affect persons apparently in perfect health, even in its worst forms. The old and young are most liable to be affected with purpura.

The pathology of purpura is very uncertain. Changes in the blood have been described, but they are not constant, and it may be quite normal. The complaint has been attributed to a primary morbid condition of the capillaries and other small vessels, of a degenerative character.

**ANATOMICAL CHARACTERS.**—Purpura is characterized by rupture of the capillaries, and escape of blood in various parts, indicated by petechiæ and other forms of extravasation in connection with the skin; hæmorrhages from mucous, and sometimes from serous surfaces; and extravasations into the cellular tissue or muscles, or into certain organs, such as the brain, lungs, and pelves of the kidneys. These organs are often found in a diseased condition, this being the cause of the purpuric state. The muscular fibres of the heart have been found in a state of extreme fatty degeneration in prolonged cases of purpura, ending fatally from repeated hæmorrhage.

**SYMPTOMS.**—Purpura is described under two chief forms—**Purpura simplex** and **Purpura hæmorrhagica**. In the former the hæmorrhages are only observed in connection with the skin; in the latter they also take place from mucous surfaces, as well as sometimes into serous cavities, or into the substance of organs, while the cutaneous hæmorrhages are generally more abundant.

The skin presents various forms of extravasations, namely, minute points or stigmata; petechiæ; vibices; or extensive ecchymoses. These are observed chiefly on the legs, and may appear in successive crops, each crop lasting a variable number of days. Their occurrence is often favoured by much standing. From their first appearance pressure produces no effect on the colour, which is often bright-red at the outset, but afterwards becomes darker, changing to purple, violet, or almost

black in some cases. The usual alterations in colour which blood undergoes are seen as the extravasations disappear. The spots are generally roundish, and have a well-defined outline at the commencement, but subsequently they gradually fade into the surrounding skin. They are not at all raised, but there is often hardening and swelling of the subcutaneous tissue. Blebs containing sanguineous serum sometimes form under the cuticle; and in rare instances the skin becomes actually gangrenous.

The hæmorrhages from mucous surfaces which may be met with are epistaxis, hæmorrhages from the gums and mouth, hæmatemesis, melæna, hæmaturia, hæmoptysis, and menorrhagia. These may be slight, or profuse and frequently repeated. In rare instances bleeding takes place from the ear. Extravasations may also escape into or beneath the mucous membranes, such as the conjunctiva, or the membrane covering the palate, cheeks, or gums. Little blisters containing bloody serum sometimes form on the tongue or cheek. Occasionally hæmorrhages from the choroidal vessels occur, even sufficient to cause blindness. Extravasation into organs is rare, but death has occurred from pulmonary or cerebral apoplexy in exceptional cases of purpura.

The *general* symptoms vary much, and are considerably modified by the conditions under which the purpura arises. There are often premonitory symptoms for some time, such as general pains, languor, and debility, but not always. The attack may be ushered in by pyrexia, and occasionally febrile symptoms of a hectic type have been noticed. Pains in the abdomen, especially in the epigastrium, loins, chest, and limbs, are often complained of. The digestive organs may or may not be impaired in their functions. There is usually more or less debility, and a sense of depression, and in severe cases this becomes extreme, being accompanied with marked anæmia if much blood has been lost, and a tendency to faintness or syncope. The pulse is usually feeble, quick, and compressible. The urine may be albuminous apart from the presence of blood, and sometimes contains casts.

Purpura has a very variable duration, being either *acute* or *chronic* in its progress. It usually terminates in recovery, if uncomplicated.

VARIETIES.—In addition to the varieties already mentioned, the following are described:—1. **Senilis**, which is met with in old people, owing to degeneration of their vessels, and especially on the exposed and irritated arms of aged women. 2. **Rheumatica**, occurring in rheumatic individuals, and attended by much pain. 3. **Urticans**, where *purpura simplex* accompanies urticaria. 4. **Papulosa**, which is merely a form of lichen.

DIAGNOSIS.—Scurvy is the main disease from which purpura has to be diagnosed. It might possibly be mistaken for ecchymoses from injury; flea-bites; typhus fever; or the hæmorrhagic form of measles. This complaint must also not be confounded with the *hæmorrhagic diathesis*.

In addition to the difference in the ætiology of the two diseases, purpura not being obviously due to the want of vegetable food, and the use of this class of diet or of lime-juice not having any material influence in its cure or prevention, scurvy presents the following characters which distinguish it from purpura:—1. The peculiar colour and sallowness of the skin. 2. The state of the gums. 3. The greater extent of the ecchymotic patches. 4. The presence of brawny indurations in the substance of the limbs, accompanied with much pain and stiffness. 5. Extensive desquamation of the cuticle.

PROGNOSIS.—The prognosis of purpura will depend greatly on its cause, and especially whether the complaint is associated with any organic affection. The *hæmorrhagic* variety is very dangerous, but I have known recovery take place, apparently spontaneously, when all treatment had failed, and the case had been given up as hopeless. The *simple* form is often very tedious in its progress towards recovery; and it is also liable to return, sometimes periodically.

TREATMENT.—In the first place it is necessary in the treatment of purpura to inquire into all *hygienic conditions*, and improve these if they have been at fault; at the same time giving a nutritious *diet*, consisting of both animal and vegetable constituents, alcoholic stimulants being also in many cases required in moderation.

Rest in the recumbent posture is generally advisable; or, at any rate, the legs should be kept up. Attention must be paid to any cachexia present; or to any organic disease on which the purpura may depend. If there is much plethora, *saline purgatives* may be given at the outset.

The chief remedies employed for the cure of purpura are tincture of iron in full doses; turpentine; tincture of larch bark; and arsenic. The first mentioned is most useful, and it may be combined with quinine and dilute sulphuric acid. In the *hæmorrhagic* form, gallic or tannic acid, alum, ergot of rye, acetate of lead, or subcutaneous injection of ergotine are the remedies indicated. Other astringents may also be freely administered, but they often fail to produce any effect. Local astringents, pressure, and cold, especially by means of ice, may be tried where their application is practicable. Careful bandaging of the legs, or the use of elastic stockings may prove advantageous in many chronic cases of simple purpura affecting the lower extremities.

---

## CHAPTER XXXIII.

### RACHITIS—RICKETS.

ÆTIOLOGY AND PATHOLOGY.—Although rickets is an exceedingly prevalent complaint amongst children in this country, there is still much uncertainty as to its nature and causation, and many diverse opinions are entertained. The subject was discussed by the Pathological Society in 1880, and also at the International Medical Congress in 1881, and in the ensuing remarks an endeavour will be made to incorporate the points brought out in these discussions which seem worthy of notice.

Some of the most prominent changes observed in rickets are connected with the bones, but it must not on that account be regarded as a mere local disease of these structures. Unquestionably it is a *general* disorder, and may be fairly considered as a special *diathesis*, using this term in its widest sense, as defined by Sir William Jenner and Mr. Hutchinson.\* Trousseau strongly maintained the identity of rickets and osteo-malakia in the adult, and Mr. Hutchinson seems to favour this view. The general opinion, however, is that these are entirely

\* See "Pathological Society's Transactions," Vol. XXXII., page 313.



distinct maladies. Dr. Goodhart thinks that the histology of rickets points to imperfect development rather than to disease, and in this light it has no relation to mollities ossium or senile fragilitas ossium.

A very important question, which may next be conveniently considered, is the relation of rickets to *congenital syphilis*. M. Parrot strenuously maintains the theory that the constant cause of rickets is hereditary syphilis, but that at the period when syphilis produces rickets the syphilis has spent itself; it has made its last effort; it exists no longer, but has substituted for itself a new affection. He regards this as an incontestable example of transformation of disease. Such a theory is contrary to almost universal experience; there are strong arguments against it; and it is opposed by all the best authorities. It may be, however, that congenital syphilis ranks in some cases as one of the causes of rickets, as suggested by the late Dr. Fagge. Moreover, syphilis may, on its own account, produce conditions of the bones simulating those of rickets. Mr. Parker holds an entirely opposite view to that of M. Parrot, and affirms that "without rickets there would be little or no congenital bone syphilis." He regards rickets as "a lesion of nutrition of the primordial cartilage, out of which the bony skeleton is formed;" and thinks "that the changes which M. Parrot has described as due to syphilis, and sometimes leading to rickets, might be better defined as the manifestations of congenital syphilis in the already rachitic skeleton."

Allusion may next be made to *tuberculosis*. Most observers have concluded that there is no relation between this diathesis and rickets, but that they are entirely distinct. Dr. Eustace Smith says that rickets never occurs in children in whom the tubercular disposition is well-marked; and a table of cases prepared for Sir William Jenner appears to show that the offspring of phthisical parents are less likely to have rickets than those of non-phthisical parents. On the other hand, one diathesis certainly does not exclude the other, and some writers think that a tubercular predisposition may favour the development of rickets.

Recognizing rickets as an independent general disease, can it be hereditarily transmitted? On this point there is again much difference of opinion. Vogel, Parker, and others regard heredity as an important ætiological factor. It is maintained that although the external manifestations of rickets in the parents may have disappeared, the constitutional peculiarity still remains, especially under social conditions favourable to this disease, and that the constitutional predisposition may be transmitted, which is developed under suitable conditions. It has even been stated that the rachitic conformation may be hereditarily continued. Mr. Hutchinson thinks that there is nothing improbable in the idea of the hereditary transmission of a constitutional tendency to rickets, that is, "of a tendency to defective nutrition on the part of the organs of assimilation, which may, either with or without the help of contributory causes, at some time bring about such a defect in the composition of the blood as would interfere with the nutrition of the bones." In the great majority of cases it certainly does not appear that there is any distinct hereditary transmission of rickets; at the same time the health of the parents not uncommonly has an important influence in developing the disease. Sir William Jenner doubts whether impairment of the father's health has any tendency to induce rickets; but the health of the mother is

generally recognized as an ætiological factor of much importance. If the general nutrition and state of health of the mother are below par, and her blood impoverished, the development of rickets in the child is decidedly favoured. In this way it may be a congenital disease, without being in the strict sense of the term hereditary, the fœtus becoming affected during intra-uterine life, in consequence of deficient nutrition from the mother. Rickets has been attributed to early marriage, inter-marriage, and advanced age of the father, but on no adequate grounds.

Rickets is essentially a disease of infancy or childhood. The general opinion is that it is not congenital, but some maintain that in many cases the complaint is actually present at birth. Parker is strongly of this opinion, and states that among patients of one month old the chest deformity and slight curves of the bones are not infrequently seen. There is a special condition termed "fœtal rickets," but this is usually regarded as quite distinct from the ordinary disease. It may be affirmed that, as a general rule, rickets is not distinctly present before six or seven months after birth. It appears far more frequently during the first or second year than subsequently. Dr. Gee states that if a child is not rickety at a year old, it may be assumed that it will not become so. The complaint may, however, commence as late as seven, or even nine years of age.

While recognizing the influence of the causes already considered, there can be no doubt that rickets is commonly a disease which originates *de novo* in a child, and is artificially produced, being traceable to some obvious cause or combination of causes which lead to imperfect nutrition. Moreover, these causes powerfully assist in developing the complaint, even where any hereditary predisposition exists. Amongst them improper diet holds a prominent place. It is impossible to ignore the almost universal experience as to the effects of bad or unsuitable feeding in originating rickets, although Vogel makes no mention of it. In this country, at any rate, it is a most powerful cause. A very large proportion of cases occur in young infants who have been brought up by hand, either throughout, or from an early period, and who have been improperly fed artificially, especially with various farinaceous articles of food. In another class prolonged suckling is the cause, the child being thus fed for sixteen or eighteen months or even longer, the milk of the mother being quite unfit for its proper nourishment, especially if at the same time, as is often the case, she is weak and anæmic. In addition, not uncommonly some kind of improper food is given in the intervals. Too frequent suckling has an influence in some cases, by deranging the child's digestion. Again, the abrupt change from the mother's milk to various kinds of food may have some effect in originating rickets. It is a matter of doubt as to how these different dietetic errors produce the disease, but probably they may do so in more ways than one. There may be the premature withdrawal of the mother's milk, which is the infant's natural food; as well as the use of farinaceous and other articles which cannot be digested. In either way nutrition is impaired. And it is important to recognize the fact that rickets often results, not from any deficiency in the quantity of the food, the child being, indeed, not uncommonly overfed, but because it is wrong in quality. Another way in which improper food may help in producing rickets is by deranging digestion, and irritating the alimentary canal. The complaint has even been attributed solely to chronic intestinal catarrh thus set up, but this is certainly not correct; although long-



continued catarrh of the stomach and intestines, with diarrhœa, does assist in developing the affection. The condition of the digestive and assimilative organs must be recognized as one element in the causation of rickets. Mr. Hutchinson would refer this disease to the class of "diet diatheses," or "assimilation diatheses," and he thinks that while defective food is probably the main cause, defective assimilation in many cases helps, and in a few cases accomplishes the whole task. His view as to the hereditary transmission of defective organs of assimilation has already been referred to.

Other causes which, at any rate, often assist in producing rickets are deficient ventilation and want of fresh air, upon which Vogel lays great stress; want of sun-light; cold and deficient clothing; and unfavourable sanitary surroundings generally. In some instances it appears to follow some previous debilitating complaint.

From what has been stated as to the causes of rickets, it will be evident that it is much more prevalent among the poorer classes. It is not, however, by any means uncommon amongst the well-to-do classes, and it has been said that amongst the rich the complaint is generally observed in the eldest child, on account of the improper feeding to which it is subjected, owing to want of experience. The children of the better classes frequently suffer also from want of fresh air. Amongst the poor it is the later children who are affected, and it is very common for all, after the second or third child, to be the subjects of the disease. This is partly due to the condition of the mother, who is exposed to the same unfavourable hygienic conditions as the child, and is usually very badly fed.

Rickets is much more prevalent in large towns and cities than in country places and small towns, for obvious reasons. With regard to its geographical distribution, the complaint is most frequent in cold and damp climates. It seems to be seldom met with in its typical form in America, India, and certain other parts; but is exceedingly common in this country.

Allusion must be made to the lower animals in relation to the causation of rickets. It has been affirmed that the complaint occurs amongst certain animals, when they are placed under similar conditions, as to air and food, which produce it in the child. Experiments have also been made with the view of producing rickets in the lower animals. Guerin fed dogs of three or four weeks old on bread, meat, and especially raw meat, but no milk, and produced all the phenomena of rickets. Tripier, however, failed to produce the disease in this way; but the results obtained by other experimenters support Guerin's conclusions. Dr. Baxter made a series of experiments upon puppies, kittens, rabbits, guinea-pigs, and white mice, placing them as soon as possible after their birth upon a diet of pure arrowroot-jelly, with a variable quantity of added milk. The starch-jelly passed through the intestines unchanged, and the animals died more or less rapidly of inanition, but in no instance were the changes in the bones characteristic of rickets produced. Wegner, by administering minute doses of phosphorus to young animals, and withholding lime-salts from their food, developed an affection of the bones exactly like rickets. Heitzmann has stated that by feeding animals on lactic acid, and injecting it under the skin, he has produced a similar result. The bearing of some of these experiments will be again alluded to.

**ANATOMICAL CHARACTERS.**—The bones present some of the most



obvious morbid changes in rickets, and these changes have of late years been carefully investigated. At first there is an arrest, and then a perversion of organization and growth. It will be well in the first instance to give an account of the histological changes, founding this mainly upon Cornil and Ranvier's description. They are observed chiefly in the cartilage of ossification; in the medulla; and in connection with the periosteum.

In normal ossifying foetal cartilage an even layer, presenting a semi-transparent and bluish appearance, is visible, placed between the ossiform and cartilaginous tissues, and named *chondroid* by Broca, but really consisting of cartilage in a state of proliferation. In rickets, from its very commencement, this layer is altered. It increases in thickness, often considerably; and its surfaces become very irregular and sinuous. Sometimes it sends out long processes into the bone, which frequently become detached. The layer is furrowed by medullary canals of cartilage, containing dilated blood-vessels. On microscopic examination it is found to resemble cartilage undergoing physiological proliferation, but the primary capsules are much larger, and contain a greater number of secondary capsules, which are also larger. Thus rounded masses of cells are formed, of enormous size, and the cells themselves present an abnormal appearance, which has been termed "dropsical" by Klebs.

Another important change observed is that underneath the layer of cartilage just alluded to, a peculiar tissue forms, named "*tissu spongoïde*" by Guérin, on account of its spongy appearance. In normal ossification a thin layer exists here, named *ossiform* by Cornil and Ranvier, and consisting of trabeculae of cartilaginous matrix infiltrated with calcareous salts, the alveoli containing embryonic medulla and blood-vessels. The spongoid tissue is not due to a mere increase in this layer, but has a special origin. It often extends from the margin of the cartilage to the diaphysis, and may invade it. There is a distinct line of demarcation, though a sinuous one, between the spongoid tissue and the cartilage; but it sometimes presents islets of hyaline cartilage in its midst, and it is often quite impossible to indicate precisely where the old bone ends. At the level of the periosteum it is blended, especially on the margin of the diaphysis, with a tissue formed of nested ossiform lamellae and a soft tissue. The spongoid substance is red and very vascular, and of the consistence of fine sponge, or of bone partially softened by an acid. It is made up of very irregular alveoli, the trabeculae forming which contain angular corpuscles irregularly scattered through a granular and non-laminated substance. The corpuscles are larger than bone-corpuscles, but have no anastomotic canaliculi. The trabeculae consist of portions of cartilaginous tissue not properly ossified, but infiltrated with calcareous salts. This infiltration not only involves the ground substance, but also the secondary capsules, which are not dissolved, as happens in physiological ossification. Thus the cartilaginous tissue is invaded throughout with distinct calcareous granules. While the calcareous incrustation is taking place, the vascular canals of the cartilage, springing from the medullary cavities of the old bone, enlarge by solution of the calcified tissue surrounding them, and open into each other, thus forming a cavernous system.

Coming now to the medulla, that which is contained in the spaces of the spongoid tissue is at first fluid, red, and contains round or angular cells, some pigmented, as well as numerous red blood-corpuscles. In the older medullary spaces it is more consistent, while the cells become

stellate in form, and are separated by a slightly fibrillated ground substance. A similar attempt at fibrous organization of the medulla occurs in the old cancellous tissue and Haversian canals, as well as within the central canal of long bones, and under the periosteum. In the central canal the external layers are most modified, and in pronounced cases of rickets, while the centre of the marrow is red, fluid, and in a foetal condition, the periphery is organized into a kind of young connective tissue, which has the appearance of a medullary membrane.

There is a sub-periosteal layer of medulla in young subjects, but the periosteum and bone can generally be easily separated. In rickets this layer changes first into a soft connective tissue, and afterwards becomes more solid. It adheres to and unites the periosteum and bone, so that they cannot be separated, and often attains considerable thickness. In it subsequently appear refractive trabeculae, which curve over and unite with one another, and contain cells. The tissue in which these trabeculae are developed is called *osteoid tissue* by Virchow. Stellate bodies, which apparently anastomose together, are found in the trabeculae and the connective tissue, being larger and more defined in outline in the former.

In advanced rickets delicate nested lamellae are found beneath the osteoid tissue, forming complete cylinders round the bone, and separated from each other by a soft and vascular connective tissue. The lamellae consist of cancellous bone, and their cavities are also filled with young connective tissue. This condition results from partial absorption of bone already formed, with fibrous transformation of the medulla, and incomplete sub-periosteal ossification. Ultimately the medulla of the Haversian canals undergoes fibrous change throughout the whole thickness of the compact tissue of the diaphysis; at the same time the osseous trabeculae are absorbed, and the liberated bone cells mix with the medulla cells.

In the course of his observations in the discussion at the International Medical Congress, M. Guerin gave a description of the changes observed in long bones in rickets, of which the following is a summary:—The concentric lamellae of the long bones become the seat of a nearly colourless fluid effusion, which separates them from each other. This becomes gelatiniform, reticulated, and pink—*tissu spongoide*. If the disease tends to get well, calcareous deposits form in the spongy tissue, and can be seen with the naked eye as islets or lamellae. Finally it acquires the consistence of normal bone. The old osseous tissue is first arrested in development, then loses its consistence, and its lamellae waste and are partly absorbed. That which remains becomes incorporated with the newly-formed tissue. In extreme cases, where the disease does not tend towards a cure, all the old bone disappears, and gives way to a mass of spongy tissue, reddish in colour, areolar, elastic, and very yielding. In lesser degrees some lamellae remain, but undergo changes in form, from their defective resistance to the weight of the body and to muscular actions.

The histological changes which take place in rickety bones during the process of cure are not known. Some think that merely a process of calcification takes place.

With regard to the chemical composition of rickety bones, according to the best authorities the proportion of calcareous salts is diminished. Friedleben found from 33 to 52 per cent.; and they have been stated to be as low as 21 per cent. Lehmann affirmed that on boiling rickety bones do not yield ordinary gelatine, but an animal matter resembling that of malignant bony growths.

As the result of the changes just considered, the bones in rickets



come to present more or less obvious alterations. Cornil and Ranvier distinguish three periods in the disease—in the first the bones are not deformed; in the second there are considerable deformities; and in the third, consolidation of the affected bones occurs. The more prominent appearances are more or less enlargement of the ends of the long bones, especially in breadth; thickening of the flat bones, particularly at their growing edges; and various curvatures and deformities. All the bones are more or less softened, the flat bones becoming spongy, and the long bones may be so soft that they can be cut with a knife or scissors, and easily bent or broken, while partial (green-stick) or complete spontaneous fractures are not uncommon. Where a long bone is bent, its medullary canal is narrowed, and may become ultimately completely filled up by callus. After fracture of a rachitic bone very abundant callus is usually formed, but it is composed of osteoid tissue and not of true bone. There is much difference observed in different cases in the relative amount of enlargement of the ends of the bones, and of softening of the shafts. The curvatures are generally observed after the enlargements, and usually proceed from below upwards.

It will be impracticable to enter into any lengthy description of the distortions to which rickets give rise, and a very general outline must suffice. In the limbs the wrists and ankles are usually most enlarged, and then the elbows and knees. The bones of the forearm are generally curved backwards, but may also be twisted; the humerus is often bent at the attachment of the deltoid. The clavicles are thickened at the ends, and their natural curves tend to be increased, so that they are shortened, and the shoulders are narrowed. The bones of the lower extremity usually bend forwards and outwards, but when rickets comes on late, the curve is often in the opposite direction, and “knock-knee” is produced. Mr. Clement Lucas has described a “knock-knee tubercle” developed in such cases on the inner side of the shaft of the tibia, just below the tuberosity. The spinal column becomes bent, and often more or less twisted. The normal curves are usually exaggerated, and this is frequently accompanied with lateral deviation, resulting from rotation of the bodies of the vertebræ upon their axes. In young infants the lumbar curve is lost in the dorsal, and a “bowed” spine is produced, with the convexity backwards. The bones of the pelvis tend to be flattened and expanded above, while the sacrum is thrown forwards, and the lateral parts in the neighbourhood of the acetabulum are approximated to each other; thus the pelvic cavity is narrowed, and a section of it would be more or less triangular. The chest usually presents distinct changes. The earliest of these is the “beading” observed at the junction of the ribs with their cartilages, constituting what has been termed the “rachitic garland.” This is a very significant and early sign of rickets, beginning and being most marked in connection with the 5th, 6th, and 7th ribs (Barlow). Fagge states that the beading is often more marked towards the pleural surface than externally. In course of time the thorax often becomes peculiarly deformed. Many authorities class all cases of chest deformity from rickets under the head of “pigeon-breast,” but undoubtedly a pigeon-breast may be produced without rickets, and this disease originates a very distinct form of thorax, which will be described in the chapter on PHYSICAL EXAMINATION. The skull is often much altered in rickets, but the descriptions given of it by different observers do not exactly correspond. The head is certainly enlarged, although it has been stated that it only appears to be so, because it is



out of proportion to the size of the face. It has a long antero-posterior diameter, with a high square brow, or often a projecting forehead, and unduly prominent frontal and parietal eminences, while it is flattened on the top. The sutures remain long ununited, and the fontanelles widely open; while the bones are especially thickened at a little distance from their edges, so that grooves are noticed along the sutures, with elevations on each side. Mr. Clement Lucas affirms that there is always increased width between the eyes, showing that the base of the skull is increased as well as the vertex; and he describes an extreme form of rickety head, which he calls the "scallop-shell head," composed of four convexities like four scallop-shells loosely articulated together, corresponding to the frontal, parietal, and occipital bones. A peculiar condition of the skull has been associated with rickets, which is observed in a certain proportion of cases, and named *craniotabes* by Elsasser, who originally described it, and considered it a form of the disease peculiar to and characteristic of sucklings. It consists in an abnormal thinness of portions of the parietal and occipital bones, causing them to yield to moderate pressure, and to impart to a finger pressed upon them a sensation like that derived from stiff parchment or from the surface of a bladder. The bones may be actually perforated. The investigations of Drs. Barlow and Lees have led them to the conclusion that *craniotabes* is not due to rickets, nor is it simply a part of general marasmus, but that congenital syphilis is the largest factor in its causation. Their own observations, however, as well as those made by other workers, seem to show that this condition may be due to rickets alone.

The degree and extent of the various deformities and changes now described vary widely in different cases of rickets. If the rickety process is arrested, the bones rapidly calcify, and often become unnaturally strong and dense. If the alterations in shape have been slight, they may ultimately gradually disappear. As a rule, however, they are permanent, more or less deformity remaining, and the body being stunted in growth. In bad cases persons who have suffered from rickets become very deformed dwarfs. It is said that the skull often continues to enlarge, and may attain a great size.

It will be convenient here to offer a few remarks with regard to the *pathology* of the rickety process in bones, and the immediate causes of the deformities which it produces. The rickety changes have been regarded as the result of a subacute or chronic inflammatory process, and consequent perversion of growth and nutrition. Another view is that there is merely a deficiency or malposition of calcareous salts; but this is not an adequate explanation of the phenomena. Rindfleisch defines rickets as depending upon an acceleration of the changes which usher in and prepare the way for the formation of bone, without the actual ossification keeping pace with them. There is an old "lactic-acid" theory, which has been recently revived, and is supported by Senator, who supposes that rickets is the result of an irritant action upon the growing bones of lactic acid, formed in excess in the alimentary canal from milk or other articles of food; there being also a deficiency of phosphate of lime, from insufficient supply, or because it is carried away by diarrhoea.

The various deformities are the immediate consequence of the softening of the bones. They result mainly from pressure and gravitation, and therefore are modified according to the age of the patient, whether it is able to sit up, crawl, or walk, the mode in which it is supported

and carried, the ordinary position assumed, and other circumstances. They are not produced by muscular action, but, on the contrary, muscular weakness allows many of the curvatures to take place. It is said that epiphyses which are most worked or liable to be injured show the greatest enlargement. With regard to craniotabes, this has been attributed to pressure by the brain, due to the mechanical action of its rhythmical movements, its rapid growth, and its own specific weight. Sir W. Jenner thinks the condition results from the pressure of the pillow. Parker looks upon the thin spots as islets of undeveloped bone.

The structures generally tend to become relaxed and wanting in tone in rickety children; and those connected with bones are arrested in growth. The muscles become more or less atrophied, pale, and flabby. No special microscopical change has been observed in the muscular tissue.

With regard to the blood, Dr. Goodhart has found in some cases deficiency of corpuscles, in others deficiency of colouring matter; and he has also specially noticed a remarkable variety in the size of the red corpuscles, along with a quantity of free granules, beaded strings, and dumb-bell accumulations.

Of late the spleen, liver, and lymphatic glands have attracted particular attention in rickets. Formerly these organs were believed to be the seat of albuminoid disease, but this was probably a mistake. Dr. Dickinson regards the changes in these viscera as an essential part of the rickety condition; and has observed that where the visceral change is most marked, that in the skeleton is seldom extreme. The affected organs are enlarged, but especially the spleen, without any material alteration in shape or smoothness. The increase is said by Dr. Dickinson to be a form of hypertrophy, which bears resemblance to that which occurs in bones. There is no new growth or deposit, but a large increase in the interstitial tissue, and some in the epithelial elements. In the spleen the trabecular tissue is increased, and its delicate threads swollen; but there is no sago-transformation in the Malpighian bodies. The portal cellular tissue in the liver is also augmented, soft, and loose. The absorbent glands are least affected, but of these the mesenteric show the most decided swelling, the superficial glands being only shotty to the touch. None of the involved structures give the tests of lardaceous disease; and they are deficient in earthy salts. It must be stated, however, that other observers do not fall in with Dickinson's views. It has been said, indeed, that the spleen and liver are not enlarged at all, but merely displaced. Gee affirms that the appearance of the spleen differs in no respect from that seen after ague, or in inherited syphilis, and thinks that its enlargement is the result, not of the rickets, but of the general state of health which causes it.

The conditions found within the cranium in rickets are also disputed. The brain may be small, with fluid between it and the skull (Gee); or it is enlarged, but the enlargement is due to increase of the neuroglia, or to albuminoid infiltration (Jenner), and not to true hypertrophy. Ordinary chronic hydrocephalus may occur; and Fagge observed in one case a form of chronic cerebritis.

The remaining morbid conditions which may be met with in cases of rickets need only to be enumerated at present, and these are:—1. Collapse and emphysema in certain parts of the lungs, in connection with the deformed and weak thorax. 2. Bronchial catarrh or extensive bronchitis. 3. Pleurisy. 4. White patches on the pericardium and spleen, due to the distorted chest. 5. Gastro-enteric catarrh.

**SYMPTOMS.**—At an early period the symptoms of rickets are often very indefinite, the onset of the disease being insidious. Generally there is marked disturbance of the alimentary canal, with some degree of pyrexia, the pulse being quick and irritable. The child alters in disposition, becoming dull and sad, or peevish and irritable; is restless or drowsy; complains of headache; and is languid, refusing to play or to be amused. Either no attempt at walking is made; or, if the patient has commenced to walk, unsteadiness of gait is first observed, followed by inability to walk. At the same time wasting may be observed, the tissues becoming flabby, and the face pale. These and other non-characteristic symptoms may be present, but there are three signs which Sir William Jenner looks upon as pathognomonic of early rickets, namely:—1. Profuse sweating about the head, neck, and chest, especially during sleep, attended with enlargement of the veins, the other parts of the body being often at the same time hot and dry. 2. General soreness and tenderness of the body, not only affecting the bones, but also the muscles, such as those of the loins and abdomen; gradually increasing in severity until the child cries on being touched or moved, or even on the approach of any one; cannot bear to be washed or played with; and finally keeps quite still, avoiding every movement. Older children do not suffer so much, but only complain of pain in the limbs. 3. A febrile condition, causing the child to throw off the bedclothes at night, in order to try to get cool. At this time also the urine may be very copious; and it may contain abundant calcareous salts or phosphates.

Sooner or later the *changes in the bones* are revealed, and in outpatient hospital practice it is generally found that these are more or less obvious when a child suffering from rickets is brought to the hospital. The enlarged ends of the bones can be distinctly seen or felt, so that the joints appear swollen and knobby, especially those which are least covered, such as the wrists and ankles; and they also have a loose feeling. A string of nodules is usually felt along each side of the chest, at the junction of the ribs with their cartilages. The limbs and trunk are found to be distorted and curved in various ways and degrees, as already described. In some cases the enlargement of the joints is most evident; in others the distortion of the limbs attracts most attention. Rickety children may become flat-footed, owing to weakness of the plantar aponeurotic and muscular structures, and of the ligaments.

The head and face usually present well-marked rickety characters, and these may be extreme. The cranium is often unsymmetrical. A blowing sound has been said by Rilliet and Barthez to be commonly present over the cranial sutures of rickety children. The hair on the scalp is generally thin. The face looks small, especially in contrast with the large head, and is often turned upwards, owing to the head being thrown back or sinking between the shoulders, in consequence of the increase in the curve of the cervical spine. The child assumes a staid, sedate, or pensive expression, and becomes “old-looking.”

The teeth appear very late, in many cases none having come through at the end of a year or more; while they also rapidly decay or fall out, being deficient in enamel.

The *general* symptoms advance with the progress of rickets. Emaciation, flabbiness of tissues, debility, and loss of power become more or less marked, the child being sometimes completely helpless, and unable to sit up or move in the least, the head dropping in any direction. In



some cases, however, there is no wasting, but rather undue fatness, and Sir William Jenner regards the emaciation as due to the changes in organs, especially the lymphatic glands. In acute cases the loss of power may become so marked, without any evident changes in the bones, that the condition simulates infantile paralysis. The skin often becomes thick and opaque, and covered with downy hairs; while profuse sweating continues. The alimentary canal is more or less deranged, and there is often much flatulency. Enlargement and protrusion of the abdomen is commonly a prominent symptom in rickets. This is due chiefly to distension of the alimentary canal with flatus; partly to depression of the diaphragm and smallness of the pelvis, to enlargement of the spleen and liver in some cases, and to weakness of the abdominal muscles. The spleen is in some instances easily recognized, and it may attain a large size; more commonly it has to be felt for, and is found with some difficulty. The liver may be felt projecting two to three inches below the margin of the ribs. The glands in the neck have frequently a shotty feel, and those in the groin and axilla may be similarly affected. Pyrexia may continue, and hectic symptoms may set in. Some writers, however, state that the temperature is below normal, and in twenty cases Dr. Day found the morning and evening temperature so nearly normal, that the difference was not worth notice. Development and growth are arrested more or less, the body remaining short and stunted.

Much difference of opinion exists as to the state of the intellect in rickety children. Some believe that their mental powers are above par, but this is certainly not the case, although they may appear to be very precocious, and are often amusing and old-fashioned in their ways and mannerisms; in some instances they become stupid, or even almost imbecile, this being evident in the expression of the face. Rickety children are late in beginning to talk; and their memory is defective.

The urine is pale and abundant; contains an excess of earthy phosphates and lactates; and sediments of oxalate of lime often form. Urea and uric acid are said to be deficient, but deposits of uric acid may occur. Urinary calculi are not uncommon in connection with rickets.

During the course of rickets symptoms indicative of the *complications* already mentioned frequently appear. Among the most common and the most dangerous complications are bronchitis and gastro-enteric catarrh. Laryngismus stridulus and general convulsions are also very liable to occur. It has even been stated that laryngismus is never observed except in connection with rickets, but this is a questionable statement. This complaint has been attributed to local irritation of the recurrent laryngeal nerve by the growing extremities of enlarged tracheal rings, or where it passes under the inferior corner of the thyroid cartilage, or at the jugular foramen (Lucas). Tetany may also occur in rickets. A fatal termination in cases of rickets is generally due to some complication; but it may be the mere result of the cachexia attending the disease.

Cases of rickets present all grades of severity and advancement. When a favourable termination ensues, the symptoms gradually subside; strength is restored; and the bones ossify, though usually more or less permanent distortion remains, which, however, may frequently be greatly diminished by appropriate treatment. Children who have suffered from rickets in a marked form rarely attain the ordinary height; and they may become deformed dwarfs.

A class of cases recently described as "Acute Rickets" by German

authors, has been satisfactorily proved by my colleague Dr. Barlow\* to be truly scorbutic, although occasionally complicated with rickets.

**DIAGNOSIS.**—In a well-marked case of rickets the diagnosis presents no difficulty, but in its earlier stages the malady is not so easily recognized. It should always be borne prominently in mind among the diseases of children, especially of those belonging to the poorer classes in large towns, though it must not be forgotten that the complaint may also be met with among the better classes. Should there be any reason to suspect rickets, inquiry must be made with regard to its more characteristic early symptoms; and the history of the mode in which the child has been fed may also help the diagnosis. Delayed dentition, or the fact that a child shows no disposition to walk, not uncommonly first draws attention to the presence of the complaint. The nodules on the chest must be particularly looked for as one of the earliest signs of the changes connected with the bones, special attention being paid to the 5th, 6th, and 7th ribs.

**PROGNOSIS.**—Most cases of rickets may be cured, if taken at a sufficiently early period, and treated properly. Its complications are very serious, and the presence of rickets adds materially to their gravity, especially in the case of chest-affections. The thoracic and pelvic deformities which it originates may prove highly injurious in course of time; and the latter are specially important in relation to parturition.

**TREATMENT.**—1. **General management.**—The first matter requiring attention in the treatment of rickets is the *feeding of the child*, which is almost always at fault, and about which thorough inquiry should be made, with the view of correcting whatever may be wrong. The breast should only be given at regular stated intervals, and for a certain time; or the child should be weaned if suckling has been continued beyond the proper period, or may be partly artificially fed. Often the health of the nursing mother requires to be improved. The feeding of children artificially needs the greatest care in this disease. Milk mixed with a fourth part of lime-water should constitute the principal article of diet, to which may be added a little cream and milk-sugar. This combination must be given in regulated quantities, and the feeding-bottle should be kept scrupulously clean. It is a common custom to give young infants considerable quantities of arrowroot, corn-flour, and various artificial foods of a farinaceous character, and these certainly do much harm. Only a very small quantity of such articles should be allowed, if any. Ass's and goat's milk are useful if they can be obtained. For older children, beef-tea in small quantities and milk-puddings are valuable; and at a still more advanced period they may have a small amount of pounded meat, or be allowed to suck a piece of underdone beef, or given the juice pressed out of it. Potatoes well mashed with gravy may also be permitted in moderation. All indigestible substances must be avoided.

*Hygienic management* also demands special attention. The ventilation of the bedroom must be looked to, and the child should have a separate bed, if possible, which must be kept very clean and dry. After the early symptoms have subsided, the patient should be a good deal out of doors whenever weather permits, in the sun, a dry bracing air of moderate temperature answering best. The clothing must be sufficiently warm, and the common custom of inadequately covering the lower part of the body in children ought to be avoided. A change to the seaside is very beneficial. The body should be washed over twice a day with warm water; and later

\* "Med.-Chir. Trans.," Vol. LXVI., page 159.

on warm salt-water bathing, followed by friction, is useful. It is important to look to the position assumed by the child, and also to the movements carried on, so as to prevent deformity. Straight wooden splints lightly applied along the legs, and extending a little beyond the feet, are of use for the purpose of preventing the child from attempting to walk. The abdomen should be well supported by a bandage. Any distortion of the limbs must be removed, as far as possible, by systematic but judicious efforts to straighten them.

**2. Therapeutic treatment.**—It is almost always necessary to treat the alimentary canal in cases of rickets, as this is usually out of order. A combination of rhubarb with carbonate of soda, magnesia, or chalk answers very well, and an occasional dose of castor-oil may be given. Grey powder is useful now and then, when the stools are offensive, but should not be habitually administered. Lime-water also improves the condition of the stomach and bowels. *Alkalies* and bitters are recommended by some practitioners. At a later stage the two great remedies for rickets are cod-liver oil and some preparation of iron, care being taken that the limbs are straightened as much as possible before these are administered. The cod-liver oil should be taken after meals, in half-a-teaspoonful or teaspoonful doses. In the case of infants it may also be rubbed into the arm-pits, and a flannel moistened with some of the oil may be worn over the abdomen. The best preparation of iron is steel wine, but others are useful, especially the potasso-tartrate, ammonio-citrate, saccharated carbonate, syrup of the phosphate or iodide, or Parrish's food. Chalybeate waters are also of service. Sometimes it is advisable to combine quinine with the iron.

**3. Complications.**—When any inflammatory affection arises in rickety children, lowering measures are not well-borne, but supporting treatment is indicated. Laryngismus stridulus and convulsions require *tonics*, and warm baths with cold douching. The slightest sign of bronchial catarrh ought to receive immediate attention, as rickets renders this complaint extremely dangerous, while it aids materially in producing deformity of the chest.

---

## CHAPTER XXXIV.

### CONSTITUTIONAL SYPHILIS.

IN the present chapter it is not intended to enter into a full description of syphilis, but merely to give an outline of the course of the affection as it results from *direct contagion*, with the constitutional effects thus induced; and to consider the phenomena presented by *congenital syphilis*. The lesions produced by this complaint in connection with the more important organs will be discussed in fuller detail in the chapters devoted to the diseases of these several organs.

**SYPHILIS FROM DIRECT CONTAGION.**—Syphilis is classed by many authorities along with the specific fevers, the differences observed being supposed to be due to its prolonged course. As a *primary* disease it can only be transmitted from one individual to another by *direct inoculation*, or by *contact* of the specific virus, either with certain mucous surfaces, or with a wound or abrasion. This produces a *specific ulcer*, having an indurated base, with but little tendency to suppuration, the neighbouring



lymphatic glands being also hard and somewhat enlarged. Then follows a period of incubation, of from one to three months' duration, at the close of which secondary symptoms arise. These are preceded and accompanied by some general disturbance, indicated by languor; pains in the bones and joints, especially at night; debility and loss of flesh; slight pyrexia; impaired digestion; and a tendency to anæmia. The secondary phenomena consist of:—1. A cutaneous eruption, of very variable character, being either a mere rash, papular, scaly, vesicular, pustular, bullous, or tubercular. It usually presents a coppery tint, and is most marked in the bends of the limbs. 2. Ulceration of both tonsils, the ulcers being grey, abruptly cut, scarcely at all painful, and without any tendency to spread. 3. Enlargement of the glands of the neck, especially of those situated behind. 4. Superficial inflammation of the mucous lining of the mouth, tongue, palate, pharynx, or larynx, sometimes with slight ulceration. 5. Mucous tubercles or condylomata in connection with the tongue, angles of the mouth, pharynx, larynx, anus, penis, labia, and other parts. 6. Loss of the hair, which also becomes dry and thin. 7. Onychia. 8. Iritis or retinitis, these being rather late phenomena. 9. Slight and transient periostitis, especially on the cranium. More or less of these morbid conditions may be present, and this stage lasts from six to twelve months usually, but may in rare instances extend to eighteen months or even longer. Secondary lesions exhibit a remarkable tendency to symmetry.

After this succeeds a period, differing greatly in duration in different

FIG. 12.



Gummy growth of liver. a. Central portions of growth, consisting of granular debris. b. Peripheral granulation tissue. r. A blood-vessel.  $\times 100$ . (After Cornil and Ranvier.)

cases, during which there are either no symptoms at all, or only occasional slight cutaneous eruptions are observed, or little ulcers on the tongue or lip. This interval is in many cases followed by tertiary symptoms or sequelæ. The chief pathological tendencies of tertiary syphilis are to produce certain lowly-organized growths, of the nature of fibro-plastic, fibro-nuclear, or fibroid tissue, which are very prone to suppurate or ulcerate.

Many growths are met with in tertiary syphilis which merely result from proliferation of ordinary connective or fibrous tissue, and which present the characters of this tissue, but those which are peculiar to syphilis constitute what are termed *gummy tumours* or *gummata*; there is, however, no marked line of demarcation between these two kinds of growths, both often existing together, and the former becoming converted into the latter. Gummata are not of the nature of an exudation, but result from hyperplasia of the connective-tissue elements previously existing, this process beginning in the walls of the vessels, the new elements invading the normal tissues, and being mixed up with or displacing them. At first gummata are soft, translucent, greyish-white, and almost homogeneous; but afterwards they become firmer, tough, yellowish, opaque, non-vascular, and caseous-looking, owing to degeneration and gradual drying-up of their structure. On section they often present a central yellowish mass, or several distinct yellowish spots, surrounded by a translucent fibrous layer, which sometimes looks like a capsule, but this cannot be separated from the sur-

rounding tissues, into which it gradually passes. Gummata vary much in size, and some of the larger masses seem to be formed by the union of smaller nodules. In structure they resemble at first granulation-tissue or embryonic connective-tissue, consisting of an amorphous matrix, with minute spherical or ovoid finely-granular cells, enclosing obscure nuclei. The matrix becomes fibrillated more or less; while many of the young elements degenerate, and ultimately break down into mere granules of fat and cholesterin, which are embedded in a small amount of fibrillated stroma. These several stages may be seen in the same growth, the central yellowish portion of the nodule being that which is most advanced in the process of degeneration. A few vessels are present in the recently-formed tissues, but these subsequently disappear.

Syphilitic gummata may be absorbed more or less completely; or their fibrous stroma may be left, which tends to shrink, giving rise to deep cicatrices or seams; or in certain structures they are liable to suppurate or to ulcerate. Several tissues and organs are often implicated at the same time, this being one of the prominent characteristics of syphilitic deposits.

The chief morbid conditions which are liable to be met with in *tertiary syphilis* may be enumerated as follows:—1. Skin-eruptions and ulcerations, namely, erythema and psoriasis of the hands and feet; or ulcerations originating in tubercles, subcutaneous gummata, or syphilitic lupus. These ulcers are of a horse-shoe or kidney-shape, and spread in a serpiginous manner. 2. Ulceration of the pharynx and palate. The ulcer may commence at any point, often starting simultaneously in two or more places. It is unsymmetrical, and spreads very irregularly and deeply, causing much destruction of tissues, and forming an excavation with hard borders. It is liable to extend to the larynx, thus inducing very dangerous symptoms; or it may even reach the œsophagus. When cicatrization takes place, much induration and contraction often result, which may lead to serious obstruction. 3. Induration of the tongue, followed by hard, well-defined, unhealthy, painful ulcers. 4. Ulceration of the rectum occasionally, accompanied with dysenteric symptoms, and very apt to be followed by stricture. 5. Growths in, or ulceration of the larynx, the latter almost always beginning on the epiglottis, and presenting the usual syphilitic characters. The cicatrix following a syphilitic ulcer in this part is firm, pink, shining, retracted, and surrounded with growths, hence causing serious interference with breathing; or it may lead to more or less stenosis. The laryngeal cartilages often necrose. 6. Fibroid thickening of the trachea and larger bronchi, leading to diminution of their calibre. 7. Gummata in the subcutaneous tissue, or “cellular nodes,” most frequent among females, and generally observed on the legs, being either single or multiple. These finally form ulcers. 8. Inflammation of a bursa, especially that over the patella, followed by ulceration. 9. Gummata in the voluntary muscles, often forming indurated tumours. 10. Periostitis and disease of the bones. Periosteal nodes form chiefly over the tibia and skull, sometimes in large numbers, but they may be observed over almost any bone. They are usually attended with severe pain, especially at night, and feel very sore and tender. They may undergo absorption, ossification, fibrous development, or suppuration; or they may become truly gummatous. Syphilis often originates caries or necrosis of bones, portions being exfoliated, and much destruction of



tissues being thus caused. This is not uncommonly seen about the nose, palate, and skull, and it may set up intracranial inflammation. I have met with several instances of syphilitic caries of the ribs leading to pleurisy. Syphilitic caries presents a peculiar worm-eaten appearance. 11. Enlargement of the testicle, due to gummata. 12. Chronic enlargement of the lymphatic glands, with but very slight tendency to supuration. 13. Gummata or fibrous growths in connection with internal organs, especially the liver and nerve-centres; or, not unfrequently, albuminoid disease or fatty degeneration. 14. Morbid changes in the arteries, which may lead to the formation of aneurisms. 15. Growths in the placenta, leading to abortion or miscarriage, which events may also happen from the direct effects of the syphilitic poison upon the ovum.

The different morbid changes just enumerated are accompanied with more or less *constitutional cachexia*; and they necessarily give rise to *local symptoms* corresponding to the part affected. In tertiary syphilis the lesions present no tendency to symmetry.

CONGENITAL OR HEREDITARY SYPHILIS.—Syphilis affecting the infant is occasionally evident at birth, and various internal lesions may be developed in the fetus *in utero*; more commonly, however, the disease is not manifested earlier than from three weeks to a month or two after birth, and very rarely it is revealed as late as six months. In a well-marked case the appearance of the child is highly characteristic. There is great emaciation, with anæmia, all fat having disappeared, while the muscles feel flabby, the skin hangs in loose folds, and growth is retarded. The face has a peculiar shrivelled, aged, decrepit look, which is particularly seen when the child cries; the skin covering it being also dark, opaque, and earthy or muddy-looking. The nose is often broad or depressed. The entire skin feels dry, harsh, rough, and inelastic; and the cuticle desquamates. Various cutaneous eruptions are liable to break out, which tend to be of a moist character; among the most frequent of these is a dull-red or coppery, shining, erythematous condition of the palms and soles, of the surface around the anus, and of the thighs and genitals. Roseola, lichen, psoriasis, eczema, impetigo, ecthyma, or pemphigus may be observed. Small yellowish patches sometimes form on the skin, like hard scales, which on separating leave superficial ulcers. The hair is often very deficient; and the nails grow slowly, being also prone to ulceration. The mucous membranes may be either inflamed; or the seat of tubercles or condylomata; or ulcerated. The mouth is often hot and swollen, and the lips are fissured. Among the most characteristic phenomena of congenital syphilis are a peculiar hoarse, cracked cry; and snuffling (popularly termed “the snuffles”), with nasal discharge, which tends to clog the nostrils, and interferes with breathing. Ulcers may be visible about the nose, the angles of the mouth, the anus, or the labia. Mucous tubercles are also frequently observed about the mouth and anus; on the labia or scrotum sometimes; near the umbilicus; or in the larynx. Condylomata are occasionally present. Discharges from the eyelids or ears are not uncommon. Occasionally iritis or some other inflammatory affection of the eye is set up. Syphilitic children are more liable to serous inflammations than others. The internal organs may be implicated, enlargement of the spleen and liver being not uncommon. The lymphatic glands may be also affected. Obstructive disease of the pulmonary artery has occasionally appeared to owe its origin to hereditary syphilis. In excep-



tional cases there are well-marked evidences of congenital syphilis without any particular emaciation or anæmia.

The investigations of Cornil and Ranvier, Wegner, and Parrot, have revealed some peculiar changes which take place in the bones in connection with congenital syphilis. In the first months of life complete separation of the epiphyses is often seen at the line of ossification. Cornil and Ranvier found the ossiform tissue much thickened, and composed of trabeculæ of cartilage infiltrated with calcareous salts, without any formation of true osseous tissue; and they attribute the separation to greater friability at the level of this tissue, and think that it is caused by the movements and weight of the body. Wegner thinks that the initial morbid process is an excessive calcareous infiltration of the cartilage undergoing ossification, which causes the death of the invaded parts; these act as foreign bodies, and irritate the neighbouring living tissues, causing inflammation, so that embryonic tissue is formed, and the bone becomes partially absorbed, which process leads to a solution of continuity and separation of the epiphyses. Parrot has shown that neoplasms, showing a marked tendency to the formation of osteoid or osseous tissue, are of common occurrence in hereditary syphilis. They are found on flat bones, as those of the skull, as well as on long bones. At first soft, they eventually form osteophytes, large exostoses, or osteomata; these consist of perfect or incompletely ossified trabeculæ, which anastomose together, leaving irregular spaces of variable size, which contain a vascular and more or less fibrous medulla. Ultimately the trabeculæ thicken, and the spaces become narrower. Craniotabes has been attributed to syphilis by Barlow and Lees, and no doubt this is its cause in many cases.

Mr. Hutchinson has drawn attention to some important distinctions between *congenital* and ordinary *constitutional* syphilis. He states that in the former the secondary and tertiary phenomena sometimes occur together, but the secondary are then not well-marked; as a rule, however, there is a considerable interval between them, the child apparently recovering more or less completely, while tertiary symptoms do not set in until between five years of age and the time of puberty, or even later. In the meantime the health may be good, but the aspect of the patient is hardly ever satisfactory, while growth and development are sometimes much retarded. As special *secondary symptoms* of congenital syphilis, Mr. Hutchinson mentions diffuse stomatitis without ulcers, and diffuse inflammation of the mucous membrane of the nares; and among *tertiary symptoms* a form of phagedenic lupus, and interstitial inflammation of the cornea or keratitis, in which the cornea becomes uniformly hazy with a few interspersed whitish dots, and is very vascular. Deafness and amaurosis are also stated to be far more common in the inherited disease, but paralysis of single nerves is not observed. Another point of difference is that in all its stages congenital syphilis tends to exhibit symmetry.

The characters presented by the teeth in congenital syphilis have attracted much attention, but there are still different opinions on the subject. Some observers regard teeth of bad colour, eroded and honey-combed, as indicative of syphilis, but Mr. Hutchinson considers these changes as merely due to stomatitis, mercurial or other; they are, however, usually associated with the malformation resulting from congenital syphilis. In accordance with the best authorities, it may now be affirmed that there are no special peculiarities in the temporary teeth of syphilitic

infants. It is the permanent set which present the characteristic changes, and especially the upper central incisors, such changes being due to peculiar arrests of development, and to these teeth alone Mr. Hutchinson attaches much importance. He states that the commonest and most trustworthy condition is an arrest in the growth of the middle denticle, leaving a single central notch in the tooth, but there is usually also a general dwarfing in all its dimensions, so that it is short and narrow, but broader at the neck than at the cutting edge, and this appearance is called "pegged." The defects are usually symmetrical, but sometimes not so. The cutting edge soon breaks, or wears away irregularly, leaving a single shallow semilunar notch at the centre. At the bottom of this notch the enamel is deficient, and the dentine exposed. The other teeth besides the incisors are also often malformed, but not in a characteristic manner. They are badly developed and stunted; and sometimes slant together, or are widely separated. Mr. Moon has pointed out that the permanent first molars are often reduced in size, and of a dome-like shape, from suppression of their angles, and that the enamel is absent from the grinding surface. These teeth also show in greatest degree, and most frequently, the changes due to stomatitis. Next come the four incisors and the canines; while the bicuspid are remarkably exempt. The affected teeth become rough, pitted, eroded, and of bad colour; often showing a transverse furrow, which crosses all the teeth at the same level.

It has been suggested that the virulent character which scarlatina sometimes assumes in individuals or in families during a mild epidemic may be the result of a hereditary syphilitic taint.

There are certain important points bearing upon the *transmission of syphilis* which it will be expedient to allude to here. It seems certain that the disease may be communicated to the mother through the fœtus, usually only tertiary symptoms being then produced, and these are not of a severe character. There is reason to believe that the taint may be transmitted to a third generation. Probably syphilis may be originated by the milk of a syphilitic nurse; and some authorities believe that, on the other hand, a syphilitic child may infect the nurse.

**DIAGNOSIS.**—It is only intended here to offer a few remarks respecting the general diagnosis of constitutional syphilis, whether resulting from inoculation or from hereditary transmission. The possibility of the existence of a syphilitic taint should always be borne in mind, and in any doubtful case it is requisite to make rigid investigation in order to clear up this point. If direct information cannot be obtained, it may often be procured indirectly by inquiring about the ordinary secondary and tertiary symptoms of syphilis, such as sore-throat, rash, &c. Examination of the throat, mouth, tongue, and eyes, as well as over the tibiae and skull, may give evidence of past or present disease, in the form of cicatrices, iritic adhesions, nodes, and other lesions. The existence of paralysis of a single nerve, especially one of the cranial nerves, is strongly indicative of acquired syphilis. Nocturnal pain is also a suspicious sign. In not a few cases the results of treatment afford ample proof of the presence of the disease.

*Inherited syphilis* may in many instances be recognized at an advanced period by pallor and an unhealthy aspect of the face; arrest of growth or development; a sunken bridge of the nose; the peculiar teeth; pits and scars, or even actual ulcers, on the skin, about the angles of the mouth, or in other parts; keratitis or its remains; double deafness without

otorrhœa; amaurosis; the changes in connection with bones; or a very prominent forehead, resulting from meningitis.

TREATMENT.—For *constitutional syphilis* the two great remedies are mercury and iodide of potassium, the former being especially valuable during the secondary stage, the latter during the tertiary. Mercury may be introduced into the system by the mouth, by inunction, or by the mercurial bath, and it is in many cases requisite to bring the patient rapidly under its influence, of course due care being taken to avoid the injurious effects of this powerful drug. Calomel, blue-pill, and bichloride or iodide of mercury are the preparations which are usually administered, and both the bichloride and iodide are often very beneficially combined with iodide of potassium in the later stages. Local applications of mercurial ointment, or of black-wash, are valuable in many cases.

Iodide of potassium should be given at first in doses of gr. v three times a day, and then gradually increased to gr. x, xv, xx, or even xxx. When this drug cannot be borne in such large quantities, great benefit may be derived in some cases from its administration in very small doses. It is often advantageously combined with decoction of cinchona and ammonia. Some authorities prefer iodide of sodium or ammonium.

*Congenital syphilis* decidedly requires the administration of mercury. It may be given in the form of hydrargyrum c. cræta, gr.  $\frac{1}{2}$  twice or thrice a day; or the ointment may be rubbed into the arm-pits and inner surface of the thighs; or the milk may be used to convey it, blue-pill being administered to the mother or nurse, or the milk of a mercurialized goat being employed. At the same time all *hygienic conditions* must be carefully attended to, and the child properly fed. Cleanliness is essential, and simple local applications are often necessary, or black-wash may be needed. Toilet powder should be freely used over the seat of any eruption. The use of cod-liver oil, either internally or by inunction, is frequently attended with much benefit.

In the advanced stage of congenital syphilis mercury often disagrees seriously, and in the early stage it sometimes cannot be borne. Iodide of potassium must then be substituted. A general *tonic* plan of treatment is often serviceable in cases where specific treatment is not obviously indicated, or even along with this mode.

## CHAPTER XXXV.

### TUBERCULOSIS—SCROFULOSIS.

THESE terms are applied to certain supposed *constitutional diatheses*, attended with definite and peculiar lesions. Many believe in the identity of *tubercular* and *scrofulous* diseases; others consider them as essentially distinct. Tuberculosis is met with as an acute and chronic affection; scrofula is always a chronic complaint. There are still many questions relating to these subjects which are in a very unsettled state, and at the present time, in consequence of certain investigations and supposed discoveries, the difficulty of giving any definite account of the various points bearing upon them is materially increased.



**ÆTIOLOGY.**—Looking upon tuberculosis as a *constitutional* disease, it has almost universally been regarded as having a hereditary origin. Undoubtedly various forms of tubercular disease run in families, but many pathologists are of opinion that only a constitutional debility is transmitted, with a tendency to inflammations of a low type, the products of which rapidly become caseous, and may thus lead to tubercle. This was the view strongly maintained by Niemeyer, and he believed that the same result would follow in the case of children born of parents debilitated from any cause. Occasionally tubercle is actually congenital, being observed at birth. Intermarriages, very early marriages, and advanced age of the father have been set down as causes of inherited tuberculosis, or as aggravating an inherited tendency. It is not improbable that syphilis in the parent has considerable influence in its development in some cases.

Age materially affects both the occurrence of tubercle, and its seat. It is by far most frequently met with in children and young persons. The majority of deaths from tubercular affections as a whole occur between 20 and 30 years of age, but of 50 cases of acute tuberculosis observed by Frerichs and Litten, the largest number in each decade (14), occurred between 30 and 40. In children the disease tends to involve a number of organs; in adults it is more localized. The glandular system is very frequently implicated in the former. In acute tuberculosis affecting infants the meninges are very frequently the seat of tubercle.

Sex seems to have considerable influence as regards the occurrence of acute tuberculosis, for out of the 50 cases observed by Frerichs and Litten 43 were males. This disease also appears to be most prevalent in April, May, and June; and sometimes it assumes a quasi-epidemic character.

A number of causes which tend to lower the state of the general health, have an important influence in predisposing to tuberculosis, or to the local development of tubercle. Among these may be especially enumerated imperfect ventilation, want of fresh air, and close confinement; overcrowding; want of exercise; constant residence in a humid atmosphere; unhealthy, insufficient, or indigestible food, not forgetting the milk of the mother or nurse; intemperance; interference with the free expansion of the chest, due to clothing or occupation; previous diseases, such as measles, hooping-cough, and various fevers, as well as many chronic affections; long-continued dyspepsia; prolonged lactation; excessive sexual indulgence; undue mental labour, and depressing passions. Many of these are frequently found acting in concert, especially among the poor and hard-worked inhabitants of large towns. Unfavourable hygienic conditions, combined with improper diet, are particularly liable to affect children injuriously.

The questions which are now receiving special attention in relation to tuberculosis are those dealing with its development by infection. These will be more conveniently discussed under the head of **PATHOLOGY**.

**ANATOMICAL CHARACTERS.**—Tuberculosis is characterized by the formation of a morbid product, known as *tubercle*, which it will now be convenient to describe.

**General description.** The typical variety of true tubercle almost universally recognized at the present day consists of certain minute bodies, termed *grey granulations* or *miliary tubercles*. These appear as small nodules or granulations, about the size of a mustard or millet-

seed; generally of a roundish form, but sometimes slightly angular; well-defined; usually firm, but occasionally soft; of a greyish-white or pearly-grey colour; more or less translucent; and non-vascular. These may be quite separate and distinct; or collected into irregular groups, their individual outline being then rendered indistinct. In some structures, however, tubercle is more diffused, and appears as a greyish *infiltration*, which presents a smooth and dense section. In its earliest stage tubercle is not visible to the naked eye, and it is by the continued growth and agglomeration of fresh tubercles that it becomes perceptible, appearing either as granulations or infiltrations according to their mode of arrangement.

What has been described as *yellow tubercle* consists of nodules or masses of caseous matter, derived either from tubercle, or from various inflammatory and other morbid products which have undergone cheesy degeneration. True tubercle may be mixed with this material; while it also tends to excite inflammation around, and thus its physical characters may be more or less modified.

**Microscopic structure.**—The histological elements found in tubercle have been very differently described by different observers, but they are stated to be more or less of the following nature:—1. *Lymphoid corpuscles*, which are very small, round, colourless, translucent, and slightly granular, each containing a single nucleus. 2. *Epithelioid cells*, of larger size, very delicate, and hence liable to rupture and to set their nuclei free. 3. *Connective-tissue corpuscles*, enlarged, and sometimes containing two or more new cells. 4. *Embryonic corpuscles*, shrivelled and granular. 5. A *giant-cell*, which consists of a mass of finely-granular protoplasm of very varied form, often presenting processes at its margin, and having imbedded in it a great number of round or roundly-oval nuclei, from twenty to forty or many more, usually collected chiefly at its periphery, and sometimes regularly arranged, each containing one or sometimes two bright nucleoli. 6. *Free nuclei*. 7. An *intercellular substance*, which may be either amorphous, homogeneous, and hyaline; or granular; or in the form of a fine reticulum or net-work of delicate fibres. There is much difference of opinion among observers as to the presence, arrangement, and relative proportions of these elements; and it will be readily understood that they may vary considerably according to the seat of the tubercle, its age or stage of development, its rapidity of growth, and other circumstances. The lymphoid cells are generally considered to be most abundant, but Schüppel describes tubercle as being made up chiefly of epithelioid elements surrounding the giant-cell. This giant-cell has attracted much attention. It occupies the centre of each tubercle, and great importance has been attached to it by Schüppel and others, but it has now been conclusively shown that this element is not essential to the constitution of tubercle, and that it is found in many healthy and morbid structures, so that it is by no means characteristic of this special morbid product.

Tubercle has been distinguished by some writers as *cellular* and *fibrous*, according to the proportion of cells and fibrous reticulum entering into its formation. Friedlander, however, as well as Cornil

FIG. 13.



Elements from grey tubercle-miliary granulation.  
(Jones and Sieveking.)

and Ranvier, insist that in recent tubercle no fibres are visible, and that the appearance is due to the hardening processes employed in its preparation for microscopic examination. Tubercle does not contain any vessels or lymphatics of its own, but it may involve those belonging to the original tissue in which it is formed; and it may also enclose

FIG 14.



Giant-cells. *a.* Rounded (*Virchow*); *b.* With processes; from a muscular tumour (*Billroth*).

pigmentary matters. As tubercle undergoes degenerative changes, its microscopic appearances necessarily alter materially. The presence of a special organism—the *tubercle bacillus*—will be considered later on.

**Changes and terminations.**—Some very important changes are liable to take place in connection with tubercle. 1. *Absorption*.—It is probable that tubercle may be absorbed after it has undergone degenerative changes. 2. *Caseous degeneration*.—The want of vascularity, and consequent low vitality of tubercle, renders it very liable to this change. The deficient vascularity is probably mainly due to the pressure exercised by the cells upon the minute vessels, but it has also been attributed to a proliferation of the endothelium lining these vessels. The process of degeneration begins in the centre of the granulations, causing them to become yellow and opaque. Ultimately the material often becomes so softened as to be converted into a purulent or curdy-looking fluid, simulating an abscess; or a firm cheesy mass is produced, which may become encapsuled. As the cheesy degeneration proceeds, the microscopic appearances change, the cells shrivelling and breaking up, while granules, oil-particles, and cholesterin appear in abundance, finally nothing but a granular debris remaining. 3. *Calcification*.—This frequently follows caseation, the material consequently becoming inert. Sometimes the calcareous matter is subsequently discharged, or becomes surrounded with a fibrous capsule. 4. *Elimination and its results*.—After the process of softening has been completed, the material is often eliminated, thus giving rise to ulcers on mucous surfaces, or to cavities in organs, as is well exemplified in the case of the intestines and lungs. These ulcers and cavities may ultimately heal up, and a permanent cure may result, the cicatricial tissue formed being very prone to contract. More commonly, however, the destructive process extends, owing to the formation of fresh tubercles on the walls of the ulcer or cavity. Occasionally a cavity results from the death and discharge of a quantity of



tubercle *en masse*. 5. *Fibroid change*.—There is sometimes an increase of the fibrillated stroma, perhaps more frequently than is generally believed, coincident with a disappearance of the cells, and finally a tuberculous granulation may consist almost entirely of fibroid tissue, becoming dense and hard. This is probably identical with the change which has been described as *cornification* or *obsolescence*, in which tubercle becomes opaque, horny, and bluish-grey.

**Tissues and organs affected.**—Lymphadenoid tissues are specially prone to the development of tubercle, but it may involve any of the connective tissues. Several organs or structures are frequently affected at the same time, but in adults tubercle is often confined to one organ in chronic cases. In most cases of *acute tuberculosis* grey granulations are seen throughout almost every organ in the body, and it is one of the characteristics of this disease that it attacks many structures. Parts actively growing are most liable to be implicated. The most frequent seats of tubercle are the lungs and respiratory passages; the bronchial, mesenteric, and other absorbent glands; the small intestines; the pleura, peritoneum, and pericardium; the pia mater; the liver and spleen; and the thyroid gland in acute cases. It is not uncommonly met with in the kidneys and genito-urinary passages; in the testes; or in the brain and spinal cord. In the cases of acute tuberculosis collected by Litten, ulcers were observed on the back of the tongue in about 10 per cent., having elevated edges, and being the seat of miliary tubercles. These tubercles were also present in the choroid coat of the eye-ball in a large proportion of cases, but were rare in the retina. Among the less frequent seats of tubercle in acute tuberculosis are the prostate gland, the supra-renal capsules, the bladder, the walls of the heart, and the marrow of the bones. It is only very exceptionally met with in other structures, and in Litten's cases it was never found in the pancreas, salivary glands, or voluntary muscles. Tubercle has been described in connection with morbid products, such as scrofulous ulcers, chancres, and cancerous growths. Louis laid it down as a law, that if tubercle is found after fifteen years of age in any part of the body, it is certain to be present in the lungs. In acute tuberculosis these organs are almost invariably, and may be solely affected.

**PATHOLOGY.**—Until within a comparatively recent period tubercle was almost universally regarded as a *specific exudation from the blood*, at first fluid, but soon coagulating into a molecular blastema, the molecules aggregating together to form cells. This view is not yet extinct, though the researches of late years have satisfactorily proved that it is absolutely incorrect. Observers, however, are by no means agreed as to the *nature* of tubercle. Some still consider that many of its cells at any rate come directly from the blood, being of the nature of leucocytes; others regard these cells as being the result of retrograde metamorphosis of pre-existing tissue-elements or morbid products. Virchow and his followers believe that tubercle arises from proliferation of the fixed corpuscles of the connective tissues. On account of its structure, which mainly resembles that of *lymphatic* tissues, tubercle is regarded by many pathologists as being of this nature, and as merely resulting from a *hyperplasia* or *excessive growth of pre-existing adenoid or lymphatic tissue*. This is the view maintained by Burdon Sanderson, and it is one which has found much favour. It is supported by the fact that adenoid tissue is usually very extensively distributed, having been discovered in the perivascular sheaths of the small arteries in various structures; beneath the

epithelium of serous membranes; around the minute bronchia; under the mucous membrane of the alimentary canal; in the spleen and other glands; beneath the conjunctiva; and in other parts. In the perivascular sheaths of the arteries tubercle is described as being very commonly observed, the cells increasing at separate points, so as to compress or even ultimately to close the vessels.

Other investigators have described tubercle as originating in the lymphatic vessels themselves, by a metamorphosis of their endothelium and outer tissues; around the lymphatics, as the result of a peri-lymphangitis; or in connection with the small blood-vessels. Rindfleisch believes that the lymphoid cells are derived from the endothelium of blood-vessels and lymphatics; the epithelium of serous membranes, of the lungs, and of the kidneys; and the muscular tissue of the bronchia. Some pathologists, however, deny that true tubercle can originate from epithelium or from muscular elements. The formation of the *giant-cell* has received much attention. It has been attributed to changes in a small blood-vessel, within which an accumulation of molecular matter takes place; to alterations affecting the endothelium of the vessels; to a morbid modification of protoplasmic buds from the walls of vessels; to the aggregation of leucocytes; to the fusion of smaller cells; to the development of free protoplasm; and to a lymph-coagulum in a lymphatic. Virchow holds that giant-cells are a very peculiar form of regular cell-formation. Klein affirms that in the lungs he has followed the development of giant-cells from the epithelium of the alveoli with all possible certainty, originating either by their fusion, or by the excessive development of a single epithelial cell.

Another most important pathological question relating to the formation of tubercle has reference to its *mode of origin* or *direct causation*. The chief views held on this point may be stated as follows:—

1. That tubercle is merely the local development of a peculiar *constitutional diathesis*, either hereditary or acquired, which is recognized as *tuberculosis*.

2. That it may originate from some direct *local irritation* under certain circumstances, especially in structures where lymphatic tissues exist, quite apart from any constitutional condition, the tubercle being simply the result of a local inflammation. Mr. Treves's observations on the formation of tubercle in lymphatic glands have led him to regard the process as simply inflammatory in its origin, and he maintains that tubercle only indicates a certain stage, occurring in the most chronic or least intense form of the inflammatory process.

3. That it is the product of *infection*. At the present time two very distinct views are to be recognized in relation to the supposed infective nature of tubercle.

- a.* It is believed that tubercle may be the secondary result of the absorption of caseous matter and other morbid products into the blood, which somehow act as a poison, and give rise to a specific inflammation originating tubercle; in short, that a kind of *auto-infection* takes place. This view was first advanced by Buhl, and experiments performed upon living animals by Villemin, Lebert, Wilson Fox, Waldenburgh, Clark, Burdon Sanderson, Cohnheim, Feltz, Chauveau, and others, have shown that bodies resembling miliary tubercles can be artificially produced in the lungs, glands, and other organs of animals, especially of the guinea-pig. This has been effected by the inoculation of tuberculous matter under the skin, as well as of various other cheesy morbid products; by



the insertion of putrid meat; by putting a seton in the skin, or even causing a simple wound; by injecting cheesy matter into the serous cavities, bronchia, blood-vessels, or heart; and even by feeding animals on caseous tubercle. The artificial tubercles thus produced are associated with very numerous inflammatory growths, which rapidly become caseous. These experiments give weight to the infective theory now under consideration, though some observers deny that the morbid appearances thus produced have any real analogy with tubercle. The more recent experiments of Martin seem to show, however, that inoculation with true tuberculous matter is alone capable of producing true general tuberculosis, and that the lesions set up by introducing non-tuberculous materials are not true tubercle, but what he calls "pseudo-tubercle." Virchow is in favour of this theory of auto-infection, and he believes that by a multitude of processes a substance may be produced that can infect the neighbouring tissues, and also the whole body, and produce the tuberculous eruption. He has seen secondary tuberculous deposit at the periphery of a cancerous tumour.

b. The theory which seems now to be gaining ground, however, is that which attributes tubercle to the action of a *morbific agent coming from without*, and introduced in some way into the system. This is the view maintained by Creighton, who agrees with Klebs, and he believes that disseminated tuberculosis is analogous to the several stages of syphilis, inasmuch as it is due to a special virus introduced from without. He differs from Schüppel, who is of opinion that tuberculosis may originate as a primary new formation in the lymphatic glands, and in the serous and synovial membranes; and also from Rindfleisch, who, while admitting a primary, secondary, and tertiary tuberculosis, explains that succession as being a subordinate one on the analogy of tumour infection, as contrasted with the analogy of syphilitic infection.

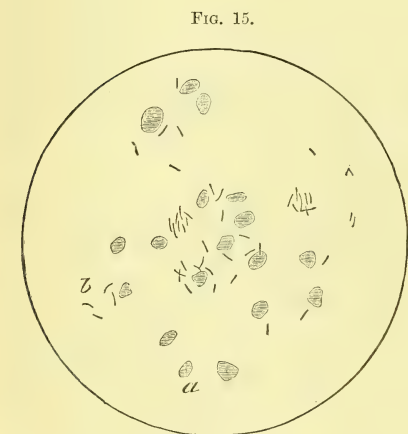
Creighton's investigations have led him to conclude that "bovine tuberculosis" may be communicated to man, and that the disease may arise from eating the flesh and drinking the milk of animals affected with this disease. Gerlach's experiments are in favour of this view; while Virchow's experiments and observations are opposed to it, and he regards Creighton's deduction as at least premature. The most important development, however, of the theory of the origin of tubercle in a specific virus entering the system from without, is that which attributes the disease to infection transmitted from one human being to another, through the medium of the breath or the expectoration in phthisical cases, as well as in other ways. It has also been supposed that it may be conveyed by vaccination. This is no new theory, and in some countries pulmonary consumption has always been looked upon as a highly contagious disease. It has received a marked impetus through the discovery by Koch of the *tubercle-bacillus*, which he holds to be the proximate cause or *materies morbi* of tubercle. This bacillus is a minute organism, motionless and rod-like, from a quarter to half the diameter of a blood-corpuscle in length, having no distinct morphological characters, but specially affected by certain dyes. The rods sometimes present oval spores. Tubercle bacilli are very rarely united in pairs, and never massed in the so-called *zoogluæ* form. They are found in tubercles in all parts of the body, and are said to abound in recent and rapidly-spreading tubercles, and at the periphery of collections of tubercles or cheesy masses, but especially in the giant-cell; while they are absent or comparatively few in parts which have undergone degeneration.



Koch calls particular attention to the presence in the centre of the tubercle cell of the minute organism which he believes has created it. This organism has also been found in caseous bronchopneumonia, in recently extirpated scrofulous glands, and in certain cases of synovial degeneration of joints; and it has been observed in the tuberculosis of the lower animals. It is, moreover, present in the sputa of phthisical patients, especially where rapid destruction of lung-tissue is taking place; and is said to be absent from other forms of expectoration. Koch claims to have cultivated the bacilli outside the body, and

apart from tubercle, and by inoculating guinea-pigs and rabbits with the organisms thus grown, to have developed tuberculosis in these animals. They require a degree of heat approaching that of the human body for their growth, the limits of temperature between which they can grow and multiply being  $86^{\circ}$  and  $104^{\circ}$ . Koch's observations have been corroborated by others, and especially by Watson Cheyne.

Such are the chief points bearing upon the supposed infectious nature of tubercle. It is still premature to come to any positive general conclusion on the subject, but there seems to be no doubt as to the possibility of tuberculosis being developed



Koch's *Bacillus Tuberculosis* in Sputum from a case of Phthisis (last stage). *a.* Pus corpuscles. *b.* Bacilli.  $\times$  about 320 diameter. (Horsley.)

by infection from within and from without, under certain circumstances.

*Acute general tuberculosis* in most cases follows, and is the consequence of infection from some local lesion, especially where caseous matter is formed. In 28 out of 52 cases collected by Litten, it was associated with pulmonary phthisis, and this accords with general experience. At the same time this affection is not always the result of infection, and it may arise without any apparent cause. In some instances it becomes developed during convalescence from one of the acute exanthemata, which probably brings out a pre-existing constitutional tendency. Litten states that in 3 cases it followed the rapid absorption of pleuritic effusion, and he attributed it to the hereditary predisposition being lighted up by the irritation caused by the removal of the pressure from the pulmonary vessels, and by the rapid expansion of the lung. *Localized tubercle* is also not uncommonly traceable to infection, as in certain instances when it is developed in the lungs or lymphatic glands. Niemeyer was of opinion that caseous matter generates tubercle by a *local* rather than by a *general* infection. The primary channels by which the infecting materials are generally supposed to be conveyed are the lymphatics, by means of which these materials may be carried into the blood, being then distributed throughout the body.

With regard to the anatomical characters of *scrofulous disease*, these will be pointed out under the signs of the diathesis. It may be stated

here, however, that scrofulous persons are very liable to the development of tubercle; and, as already mentioned, the tubercle-bacillus has been found in recent scrofulous glands, and in scrofulous joints.

**SYMPTOMS.**—Much difference of opinion has always prevailed as to whether there is a true *tubercular diathesis*, indicated by any characteristic signs. A very large number of persons unquestionably become tuberculous who present no obvious peculiarities, but the following characters are looked upon as evidencing a tendency towards the disease in children and young persons:—They are tall, slim, erect, and delicate-looking, having scarcely any fat; while they present usually a pretty oval face, a clear complexion, bright eyes, and large pupils. The skin is very thin, soft, and delicate, and through it bluish veins are visible; the hair is fine and silky, often light, the eye-lashes being long. Tubercular subjects cut their teeth early, and are generally precocious and clever, walking and talking soon. They are excitable and active in body and mind. The ends of the bones are very small and firm, their shafts also being thin and rigid; while the cartilages seem to be very soft and flexible. The thorax is small, being either long and narrow, or flattened anteriorly.

The well-developed *scrofulous* or *strumous diathesis* is characterized as follows:—The body is short, thick-set, and heavy; the face plain, tumid-looking, with expanded and thick *alæ nasi*, a low forehead, a large upper lip, and a dull, pasty complexion. The skin is thick and opaque, and is very subject to obstinate eruptions of a moist character, with a great tendency to the formation of scabs. Chronic abscesses or exudations are also liable to form in the subcutaneous tissues. Scrofulous children often exhibit the phlegmatic temperament, being inactive and languid in mind and body, as well as backward in intellect, this being evident in their expression. The bones are thick, with rather large ends, being very liable to caries or necrosis. Chronic disease of the joints is also common. The teeth often decay early. Derangements of the alimentary canal are of frequent occurrence, and the belly is generally tumid. The lymphatic glands usually exhibit marked changes. They become chronically enlarged, the enlargement being attributed to chronic inflammation, to hyperplasia of the normal lymphatic tissues, or to the actual development of tubercle; and this is very apt to end in caseous degeneration, or in slow and unhealthy suppuration. Unhealthy inflammation of mucous membranes is very frequently observed, the products being rich in cells and of a sticky character, while the membrane itself is the seat of an exudation containing abundant cells; the inflammation often ends in ulceration. Ophthalmia, tinea tarsi, ozæna, otitis with otorrhœa, throat-catarrh, and catarrh or more serious inflammation of the alimentary or respiratory mucous tracts, are of common occurrence. Scrofulous subjects may suffer from pyelitis, cystitis, or catarrh of the vagina or vulva.

The *local* development of tubercle as a *chronic* disease is attended with corresponding local symptoms; and also with indications of more or less general disturbance as a rule, in the way of fever, wasting, debility, anæmia, night-sweats, and other symptoms. These will be more particularly considered when treating of the individual diseases of organs.

**Acute tuberculosis.** It is necessary briefly to allude more particularly to the symptoms which are associated with the deposit of tubercle as an *acute* affection. Generally almost all the organs in the body are involved under these circumstances, but only the lungs, brain, and

spleen usually reveal any local signs, and even these are often very obscure.

Three forms of acute tuberculosis have been described, namely, the *insidious*, *acute febrile*, and *adynamic*, but all grades are met with, while the *course* and *duration* of the disease are very variable, the latter usually ranging from two to eight weeks. At first there may be merely languor, heaviness, irritability, or restlessness; derangement of the digestive organs, with offensive stools; irregular fever, the temperature being sometimes very high; and rapid wasting. Or repeated rigors occur, followed by high fever, with an extremely rapid pulse; much constitutional disturbance and prostration; and profuse sweating. Occasionally the pyrexia is not high from first to last. Head-symptoms are usually severe; and there is a great tendency towards typhoid symptoms, such as a dry brown tongue, sordes on the teeth, an exceedingly feeble pulse, and low nervous phenomena. Dyspnoea is a prominent symptom, breathing being very hurried, and the patient may become cyanotic; there may be more or less cough; but no marked *physical signs* can be detected in connection with the lungs, there being usually only some dry rhonchal sounds, or signs of accumulation of air in the lungs may be noticed. The dyspnoea is partly due to the condition of the lungs, and to the high fever, but Litten thinks it is also the result of the direct irritation of the peripheral fibres of the vagus nerve by the tubercles, the stimulus being conducted to the respiratory centre in the medulla oblongata. The spleen is usually enlarged, sometimes very much so; it is painless, and there being generally no tympanites, the enlarged organ can be readily recognized. Ultimately evidences of the presence of tubercle in certain structures often appear, in the form of tubercular meningitis, peritonitis, or other lesions. It must be remembered, however, that marked nervous symptoms do not necessarily imply implication of the nerve-centres, for they may be due to the high pyrexia, and to the poisoned blood circulating through these centres. Much importance has been attached by some to the discovery of tubercles in the choroid, by the aid of the ophthalmoscope. They usually appear late in the course of acute tuberculosis, but may be visible some weeks before death. These choroidal tubercles are in the form of yellowish-white, roundish spots, usually very minute, but sometimes, when confluent, as large as the optic disc; they shade off gradually into the surrounding pigment. They may be watched in their growth from day to day, and their changes may also be observed. Vision is rarely affected by these tubercles. In some cases of acute tuberculosis herpes appears about the lips.

**DIAGNOSIS.**—It is always important to recognize any tendency to tubercular disease when it exists, especially in young persons, which is indicated by the family history, and by what the examination of the patient reveals. It must be remarked, however, that affections commonly regarded as tubercular are very frequently observed in individuals who present none of the characteristic features of the diathesis. The diagnosis of the local formation of tubercle rests upon the presence of local clinical signs; accompanied with the usual constitutional symptoms.

*Acute tuberculosis* is by no means easy to recognize with certainty in many cases, and the diagnosis must be founded upon the combination of clinical phenomena present. The condition may closely resemble certain fevers, especially typhoid fever. It should always be remembered in obscure cases occurring amongst children. The absence of the peculiar



symptoms of the exanthemata, or of any eruption; high fever from the first, with the irregular range of temperature; extreme frequency of the pulse; very quick breathing; severity and rapid course of the illness; and the local symptoms which supervene, will usually enable the diagnosis to be made. The discovery of tubercles in the choroid is highly important. Litten observed that occasionally much difficulty arose in the diagnosis between diffuse bronchitis with emphysema in elderly persons, and acute tuberculosis, at any rate at first. The abundance of the râles, and the amount of the expectoration are the chief distinctions of the former condition.

PROGNOSIS.—The existence of well-marked signs of the tuberculous or strumous diathesis is a matter of serious moment, and any case in which such signs are present requires to be carefully watched. The prognosis of chronic cases in which the local development of tubercle has taken place will depend upon the seat and extent of the mischief, and various other circumstances. *Acute tuberculosis* is an extremely grave condition, the termination being almost invariably fatal. Cases have, however, been brought forward by Dr. M'Call Anderson and others, believed to have been of this nature, which have recovered.

TREATMENT.—When there is any tubercular or strumous tendency, all *hygienic conditions*, as well as the *diet*, should be rigidly attended to. Fresh air and sunlight; proper exercise; warm clothing; change to the sea-side, with salt-water baths; nutritious diet carefully regulated, with plenty of good milk; and the avoidance of undue mental labour, are chiefly indicated. The *digestive organs* must be kept in order, and all sources of irritation in connection with these organs should be at once removed. Every cause likely to originate lung-affections must be carefully avoided, and it will be well to examine the chest from time to time, as well as to treat the slightest pulmonary complaint without delay. Cod-liver oil; iron in various forms, especially steel-wine, or syrup of iodide of iron in some cases; and *tonics*, properly administered, do a great deal of good in these cases. The treatment of local formations of tubercle will be considered in their respective chapters. In *acute general tuberculosis* no measures are, as a rule, of much avail, but the administration of quinine in full doses, and the application of cold externally, with ice to the head, might be tried; along with supporting diet and stimulants. If the disease seems to be confined to particular structures, such as the lungs or peritoneum, better results may possibly be expected from treatment, as will be again pointed out.

---

## CHAPTER XXXVI.

## CARCINOMA—CANCER—MALIGNANT DISEASE.

THOUGH mainly a surgical disease, cancer not uncommonly comes under the observation of the physician, and therefore a brief general consideration of the subject is called for in this work.

**ÆTIOLOGY.**—Cancer is decidedly a hereditary disease. Age exercises a marked influence as regards its occurrence, nature, and seat. It is rare in the young, being by far most common after middle life, and the mortality from this disease increases as age advances. In early life the softer varieties of cancer are met with; and the lymphatic glands are very liable to be involved. Primary cancer is chiefly observed in organs which have been in a condition of high functional activity, but whose functions have ceased to be performed. Females suffer most on the whole, on account of the frequency with which the uterus and mammæ are attacked. The digestive organs, bones, and skin are most affected in males. Anxiety, mental overwork or distress, and a depressing climate seem to have some influence as *predisposing causes* of cancer.

Injury, excessive use of a part, or some other form of irritation may act as the *exciting cause* of the local development of a malignant growth. Some pathologists consider cancer as essentially local in its origin.

**ANATOMICAL CHARACTERS.**—Different classifications have been made of the varieties of cancer, but those which it is necessary specially to recognize here may be brought under the following groups:—1. **Scirrhus.** 2. **Encephaloid.** 3. **Colloid.** 4. **Epithelioma.** 5. **Adenoid or Tubular.**

1. **Scirrhus, fibrous, or hard cancer.** This variety either infiltrates tissues, or forms distinct tumours, which are irregular in shape, but never attain a very large size. The growth is often depressed, and causes puckering of overlying structures. The consistence is very hard and firm, sometimes approaching that of cartilage. A section is grey, bluish-white, or whitish, and glistening, while opaque fibrous bands may be seen intersecting the surface. This form of carcinoma presents but very slight vascularity. The outer part of the growth is less dense than its central portion, and yields a milky juice on scraping.

2. **Encephaloid, medullary, or soft cancer.** Assuming the form of tumours, or being infiltrated, encephaloid increases with great rapidity, forming considerable masses, which are more or less lobulated. The substance is soft and brain-like, and on section presents a pulpy appearance, especially towards the centre of a growth, varying in colour from white to crimson according to its degree of vascularity, and not uncommonly presenting small extravasations of blood. A large quantity of juice can be expressed. This form of cancer may produce very vascular fungous growths, being then termed *fungus hæmatodes*. All grades are met with between the encephaloid and scirrhus forms of carcinoma.

3. **Colloid, alveolar, or gelatiniform cancer.** Many regard this variety of malignant growth as merely one of the other forms which

has undergone colloid degeneration. It generally infiltrates tissues, but sometimes forms lobulated masses, which have a tolerably firm and uniform consistence. On section roundish spaces or alveoli are seen, having fibrous walls enclosing the colloid substance, which is of more or less glue-like consistence, glistening and translucent in appearance, and either colourless or greyish-yellow.

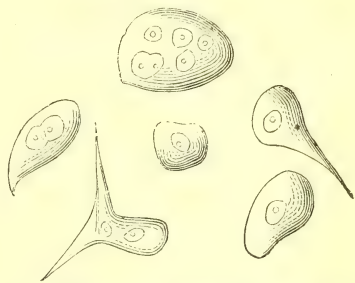
4. **Epithelioma, epithelial cancer, or canceroid.** Generally observed in connection with the skin or a mucous membrane, epithelioma commences either as a hard nodule, or as a small excoriation or ulcer. The latter has indurated edges, with an irregular, grey or bloody surface, which is often papillated and villous, or nodulated. The consistence is generally firm, but may be soft and friable. The cut surface is greyish-white, or presents numerous opaque specks and white lines of fibrous tissue; a small quantity of milky granular fluid can be expressed, which will not mix with water.

5. **Adenoid or tubular.** This variety forms various-sized tumours, very like those of encephaloid cancer, highly vascular, soft, and yielding an abundant milky juice. It is of rare occurrence, and usually originates on a mucous surface, but occasionally starts in the liver or some other solid organ. The lymphatic glands and other parts are liable to be involved secondarily. From its structure, this form of cancer is named *columnar* or *cylindrical epithelioma*, and it consists of tubules with a very small amount of fibrous stroma. The tubules are usually cylindrical, of tolerably uniform size, lined with spheroidal or columnar epithelium, and generally present a central cavity or canal. They are arranged irregularly. Adenoid cancer is said to present a marked resemblance to sections of the cortical substance of the kidney, deprived of malpighian bodies.

Other varieties of cancer which have been described are named *melanotic*, which contains much pigment, the growth being most commonly of an encephaloid character; *cystic*, where cysts are developed; *chondroid* or *cartilaginous*; *osteoid* or *bony*; and *villous*, which affects mucous surfaces, presenting villous processes. *Sarcomatous* growths may also be of a malignant nature.

**General and microscopic structure.** All the forms of cancer consists of *cells*, enclosed in the meshes of a *fibrous stroma*, these elements differing greatly in their relative proportions in the different varieties. The *cells* are of large but very variable size; present diverse and curious forms; and contain one or more nuclei, as well as usually a number of fat molecules. Each nucleus is large, clear, and well-defined; eccentric; round or oval in shape; and it encloses one or more nucleoli. Abundant free nuclei are often present. The expressed juice contains a quantity of these cells, as well as nuclei and free granules. The *stroma* is generally firm and fibrous, the fibres being either delicate or coarse; but if it has developed rapidly, it presents an embryonic structure. The vessels are solely distributed in this stroma,

FIG. 16.



Cells from a mammary cancer, magnified 300 diameters. (After Billroth.)



and its fibrous bundles intersect in all directions, forming a communicating network, within the alveoli of which the cells are grouped. Lymphatics have been found accompanying the blood-vessels, and they communicate with the alveoli.

In *scirrhus* the cells, though they may be abundant at first, speedily disappear, and the fibrous stroma is greatly in excess, especially towards the centre of the growth, where finally no cells at all can be discovered. In *encephaloid*, on the other hand, the cells are greatly in excess, developing rapidly and as speedily degenerating, becoming granular, and

their nuclei being set free. There is but little stroma, which is soft, delicate, and very vascular. *Colloid* is in great part structureless, but some cells are present, which are large and spherical, often having a lamellar outline, and containing some of the colloid material. *Epithelioma* presents generally a large number of cells, which, with few exceptions, are exceedingly like those of squamous epithelium, but are subject to great alterations in shape from mutual pressure. They tend to form peculiar *concentric globes* or *nests*, or so-called *epithelial pearls* (fig. 18), which increase from within, so that the outer layers become hardened and flattened. Ultimately the entire groups of cells may become dry, firm, and brownish-yellow. A variable amount of stroma is present. The structure of *adenoid* cancer has already been described.



Primary cancer of rectum. (Creighton.)

Cancerous growths are very liable to *fatty degeneration*, especially the softer forms, and as a result the cells become more granular, and softening takes place; or parts of the growth may assume a caseous appearance. *Calcification* is very uncommon. *Melanosis* and *colloid* are generally regarded as forms of cancer which have undergone these peculiar degenerative processes. All cancerous growths tend to ulcerate, the ulcers having no disposition to heal, but being on the other hand inclined to spread.

**Organs and tissues affected.** *Scirrhus* is usually observed in the mammary gland, uterus, stomach, rectum, or skin. *Encephaloid* affects chiefly the bones, testicles, eyes, and internal organs, especially the lungs, liver, kidneys, brain, and spleen. *Colloid* particularly involves the stomach, but is sometimes seen in the omentum, intestines, and other parts. *Epithelioma* grows in connection either with the skin or a mucous surface, but by extension it may implicate any tissue. Its ordinary sites are the lower lip, the tongue, eyelids, cheeks, scrotum, prepuce, labia, uterus, or bladder. In exceptional cases internal organs are involved.

Several parts may be attacked with cancer, either simultaneously, or

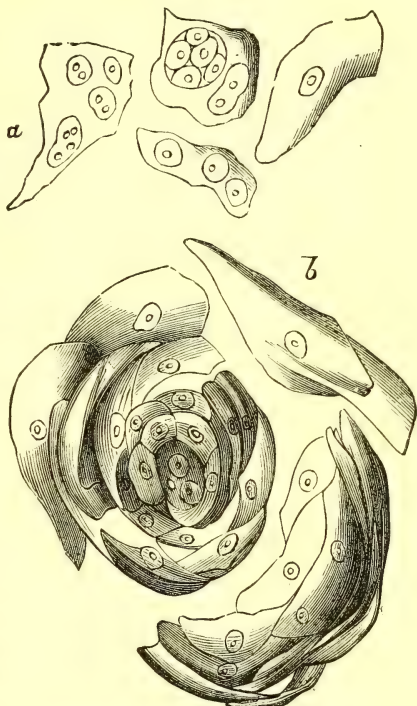
usually in succession. In the latter case the original formation is said to be *primary*, and subsequent growths are named *secondary*. Secondary deposits are frequently observed in internal organs, being generally of the same variety as the primary growth, but scirrhus is often followed by encephaloid in internal organs. Malignant formations usually show a marked tendency to spread, and to infiltrate surrounding tissues, so that no line of demarcation can be observed; in rare instances a kind of capsule forms around a cancerous growth.

**PATHOLOGY.**—Two very opposite views are entertained as to the *nature* and *origin* of cancer, namely:—1. That it is primarily a *constitutional* or *blood-disease* or *cachexia*, of which the formation of malignant growths is but a local manifestation. 2. That it is in the first instance a *local disease*, produced by some direct irritation; and that the system is only affected secondarily, as the result of absorption of morbid materials from the primary growth. It is not at all improbable that both these theories are correct in different cases. *Secondary* growths arise in consequence of absorption by the blood-vessels and lymphatics, by which the cancerous elements are conveyed to distant parts, especially those more immediately associated with the structure first affected. Neighbouring absorbent glands are very liable to become involved.

The stroma of cancer consists partly of the original cellular tissue, but is chiefly derived from hyperplasia of the connective-tissue elements. The cells originate in the proliferation of pre-existing cells; some pathologists are of opinion that they are only derived from epithelial structures, but others believe that they are also developed from connective-tissue corpuscles, leucocytes, and other cells.

Dr. Creighton, in his investigations respecting malignant tumours, has arrived at the following conclusions:—In *secondary* tumours the parenchymatous cells, and not the cells of the connective tissue of an organ, are the elements that undergo transformation. This transformation consists in a vacuolation of the protoplasm of the cells, with other associated changes; the products remaining as tumour cells, or as indifferent cells which may further develop into connective tissue. Vacuolation has been otherwise described as endogenous cell-formation, a mode of cell-growth that is essentially heteroplastic, the products being alien to the original tissue. Both in physiology and pathology it is a familiar process, as, for example, in the development of colostrum-cells, spermatozoa, mucous and salivary corpuscles, and the goblet-cells so

FIG. 18.



Epithelial Carcinoma. a. Separate cells; b. Epithelial pearls. (After Billroth.)



abundantly found amongst the epithelium of mucous surfaces; and Dr. Creighton concludes that the process of secretion in the epithelial cells of mucous surfaces is essentially a process of endogenous cell-formation. This being the case, a true physiological basis has been established for the theory of the formation of secondary malignant tumours. The theory may be extended to the origin of *primary* malignant tumours in epithelial parts, for it is evident that if the epithelium, in the exercise of its functions, endogenously produces solid elements instead of fluid substance, the result will be a heteroplastic growth. The growth of the malignant tumours in the mammæ, for example, may be explained in this way.

*Primary* and *secondary* malignant tumours have a distinct genetic relation to each other. The primary precedes the secondary in point of time; a remarkable family-likeness can in most instances be traced between them; and, what is of the greatest significance, the process of vacuolation or endogenous cell-growth, by which the secondary tumour is developed, bears a remarkable resemblance to the first changes in the ovum after impregnation. To explain the origin of secondary tumours in a healthy organ, the influence of the primary or parent tumour must manifestly be assumed, an extraneous influence operating by the mechanism of endogenous cell-formation. This influence, Dr. Creighton contends, is comparable to a spermatoc influence, which is exercised in some unknown manner by the parent tumour. Dr. Creighton attempted to produce cancer artificially, but without success.

**SYMPTOMS.**—Cancer usually gives rise to two classes of symptoms, namely, *general* and *local*. The *general* symptoms may precede the local, and include more or less wasting; a peculiar sallowness, cachectic look, with a yellowish, earthy tint of countenance; a careworn, gloomy expression; debility and languor, which may culminate in a feeling of extreme depression and exhaustion; anæmia and its accompaniments; and irregular fever. These vary much in their intensity according to the part affected; as well as with the rapidity of growth and nature of the cancer, being most marked in connection with the scirrhus variety. The *local* symptoms may be summed up as pain and tenderness or other subjective sensations, often very severe, the pain being frequently of a lancinating or burning character; symptoms resulting from interference with the functions of the part affected, these being in many cases of a serious character; those due to pressure upon, or irritation of neighbouring structures; and objective or *physical signs* afforded by the growth itself. The *duration* of cases of malignant disease is very variable, but it is rarely prolonged, and sometimes they run an acute course.

**TREATMENT.**—The treatment of cancer belongs chiefly to the domain of surgery, the morbid growth, when conveniently situated, and when other circumstances are favourable, being removed by operation, or destroyed by the use of caustic applications and other methods. When cancer attacks internal parts, no medicine is of any avail as a curative agent, and it may confidently be affirmed that never will any remedy for this disease be discovered. All that can be done usually is to support the patient by means of good food and other appropriate measures; and to treat the case symptomatically, according to the structure which happens to be involved. Experience has shown that operations for the removal of malignant growths in connection with internal structures may be resorted to with advantage in exceptional cases.



## CHAPTER XXXVII.

## I. DIABETES MELLITUS—GLYCOSURIA.

**PATHOLOGY AND ÆTIOLOGY.**—The pathology of diabetes is still very uncertain, and it is difficult to determine the class of diseases to which this complaint should be referred. Under these circumstances it may for the present be conveniently discussed among the constitutional disorders, as the general system is often profoundly affected in diabetes, although its phenomena probably depend upon derangement of a special function which is carried on in the body, and in many cases may be referred to a local cause of a definite character.

Before considering the pathology of diabetes, it is requisite to offer a few remarks respecting certain physiological questions which have a bearing upon this complaint. In the first place it has been conclusively proved that sugar is always present in the blood during life, no matter what the nature of the diet may be. Claude Bernard concluded from his experiments that in the normal state its amount fluctuated between from 1 to 3 parts per 1,000, and that it is essential for the due maintenance of nutritive action, the sugar being used up in the process of nutrition. Further, this eminent physiologist believed that it can only be destroyed within certain limits, and that about 3 per 1,000 represents the limit of the capacity of the blood for sugar, beyond which amount it overflows through the renal organs, and is excreted with the urine. He also held that there is a marked difference between arterial and venous blood, as to the relative proportion of sugar contained in each, the mean difference in several analyses representing about 0·300 part per 1,000 excess in arterial blood. Dr. Pavy, however, affirms that the experiments which led to these conclusions were fallacious, and that naturally the blood only contains a very small quantity of sugar, while the difference in the amount of sugar belonging to arterial and venous blood is insignificant.

With regard to the urine, it is generally believed that this fluid is perfectly free from sugar in the normal state. Pavy states that when sufficiently delicate tests are employed, it can be shown that healthy urine always does contain sugar in minute quantity; that a sufficiency to give a slight reaction under ordinary testing is not uncommon; and that sometimes as much as 5 to 8 parts per 1,000 may be present as an incidental occurrence. Consequently, he maintains that there is no abrupt line of demarcation, or distinction of an absolute kind, between the urine of health and that of diabetes; and that the difference, as regards the urine, is one of degree and not of kind. The chief circumstances under which this temporary glycosuria may occur, so that the presence of sugar can be appreciated by the ordinary tests, are after taking food containing much sugar or starch; after the administration of chloroform; in poisoning by strychnine or woorara; in various conditions which interfere with respiration, such as during paroxysms of asthma or hooping-cough; in certain nervous diseases, for example, epilepsy, tetanus, or apoplexy; and in connection with injuries affect-

ing the nervous system, the liver, and other parts. Glycosuria can also be induced experimentally in several ways.

The next question relates to the explanation of the presence of sugar in the blood and urine. As regards the blood, a certain quantity is probably taken up directly from the alimentary canal by the absorbents, and conveyed along the thoracic duct to the general circulation. Bernard and those who agree with his views account for the existence of sugar in the blood chiefly by the so-called *glycogenic* theory. This observer discovered that in health a substance is formed in the cells of the liver, derived from the sugar which is absorbed by the blood-vessels from the alimentary canal, and conveyed by the portal vein into the liver. This substance is of an amyloid nature, and is capable of being readily converted into grape-sugar; it has received various names, the chief being *glycogen*, *amyloid substance*, *zoamylin*, *animal* or *hepatic dextrine* or *starch*, and Pavy has suggested that it should be called *Bernardin*, after its eminent discoverer. It is further believed that a peculiar ferment exists in the blood, which has the power of converting this glycogen into grape-sugar. According to Bernard and his followers, this conversion is being constantly carried on in the liver in the normal state, the sugar being then conveyed by the hepatic vein into the circulation, and gradually undergoing combustion in the peripheral capillaries, especially in those of the muscles, being converted into water and carbonic acid, and contributing to force-production. This destruction of the sugar is supposed to account for the difference alleged to exist in the relative proportion of sugar contained respectively in arterial and venous blood. Bernard also affirmed, from his experiments, that during life the liver contains from 1 to 3 parts per 1,000 of sugar, derived from the glycogen. Pavy is strongly opposed to these views, and his theory is, that the liver is a sugar-assimilating and not a sugar-forming organ; that the sugar which reaches this organ is converted into, and stored up as amyloid substance (he objects to the term glycogen), but that this material is not re-converted in the normal state into sugar; and that one of the functions of the liver is to detain and appropriate the sugar which passes through it, changing this element into a substance which can be afterwards used up in the economy, and preventing it from passing into the general circulation as sugar, which is incapable of utilization. He, moreover, maintains that amyloid substance can also be formed from nitrogenous matter, owing to a re-arrangement of its elements, and this is in consonance with the views of some physiologists, who maintain that the liver is the organ in which urea is produced. As to the amount of sugar present in the liver, Pavy holds that this is very small, that the large proportion found by Bernard was due to *post-mortem* change, and that if the liver is frozen immediately after death only a minute quantity of sugar can be obtained from it. In this he is supported by many eminent experimenters in physiology. With regard to the sugar present in the blood in health, he affirms that it is not derived from any normal change in the glycogen, but that it is mainly either the excess of the ingested sugar which has not been appropriated by the liver, and has thus been permitted to reach the general circulation; or that it is derived from an unnatural change in the amyloid substance, which possesses a strong tendency to pass into sugar under the influence of contact with bodies of the nature of ferments. Dr. McDonnell has advanced a theory that hepatic dextrine is not converted into sugar at all, but that it unites

with nitrogen to form a new protein-compound resembling casein. Some authorities hold that the muscles form glycogen as well as the liver, and that conversion of this material into sugar takes place in these structures, as well as the destruction of sugar.

With reference to the presence of sugar in the urine in a state of health, according to the glycogenic theory this represents the excess of the saccharine element which is not destroyed in the system, whether in consequence of excessive ingestion, or of undue production from glycogen, and which is therefore eliminated by the kidneys. The supporters of this view hold that only exceptionally, and under particular circumstances, does the urine give evidence of the presence of sugar within the limits of health. On the contrary, Pavy states, as has been already mentioned, that sugar is always present in minute quantity in the urine, corresponding to the amount existing in the blood; and he denies that it represents any excess over what is destroyed, or that sugar is specially eliminated by the kidneys, but merely passes through the vessels of these organs by a process of diffusion, as it does through all the vessels of the body, and thus appears in the urine.

Another point bearing upon the pathology of diabetes is the connection which physiological experiments have proved to exist between the nervous system and glycosuria. Bernard discovered that glycosuria may be induced by penetrating a certain spot in the floor of the fourth ventricle. Subsequently Pavy ascertained, and his experiments have been corroborated by other observers, that the same result follows injury to certain portions of the sympathetic nerve. Thus sugar appeared in the urine when the filaments ascending from the superior thoracic ganglion to accompany the vertebral artery were divided, when the superior cervical ganglion was removed, and in some instances when the gangliated cord in the chest was divided. The influence of the nervous system in the production of glycosuria is generally believed to be exercised through the vessels going to the liver, paralysis of their walls being induced, with consequent dilatation; but some physiologists are of opinion that the nerves directly affect the amount of glycogen formed, and the rapidity of its conversion into sugar. With regard to the effect of the vascular paralysis, most authorities consider that it merely leads to a state of hyperæmia of the liver, in consequence of which a more intimate and speedy contact of the glycogen and ferment is brought about, and Schiff believed that a special ferment was developed under these circumstances. Pavy, however, has found experimentally that when defibrinated arterial blood is injected into the portal vein, marked glycosuria is induced, and this condition is also noticed when the blood generally is maintained in a highly oxygenated state, as when artificial respiration is performed, or carbonic oxide is inhaled. This authority maintains that these observations explain the relation of the nervous lesions to glycosuria, a state of vaso-motor paralysis of the vessels of the chylipoietic viscera being induced, which permits the blood to reach the liver through the portal vein without being properly de-arterialized, and it is the presence of oxygenated blood in this vein which causes the glycosuria, though in what precise way it acts he is not prepared to state, whether by interfering with the formation of amyloid substance, and thus allowing the sugar to pass through the liver unchanged, or by facilitating the reconversion of the amyloid substance into sugar.



Having discussed the physiological questions relating to glycosuria, the main theories at present held as to the pathology of diabetes may now be enunciated.

1. According to Bernard and his followers diabetes consists in an *increased formation* of sugar in the liver from glycogen, in excess of what normally takes place; hence it accumulates in the blood, and is excreted by the kidneys. A modification of this view is that there is *diminished destruction* of sugar in the system, either of that normally formed, or along with excessive production. The muscles are also supposed to be at fault in some cases, and to assist in the causation of diabetes.

2. Pavy's views are entirely opposed to those just stated. As previously remarked, he regards diabetes, so far as the presence of sugar in the urine is concerned, as being a mere exaggeration of the condition observed in health, the difference being one of degree and not of kind. He holds that this complaint consists either in a *want of assimilative power* on the part of the liver over the saccharine principle, so that it is not appropriated and converted into amyloid substance, but is allowed to pass on into the general circulation; or in an *abnormal change* of this amyloid substance into sugar, a change which does not take place in health. The excess of sugar in the blood is evidenced by the presence of a corresponding amount in the urine.

3. Most observers agree that in a large proportion of cases at any rate some morbid condition of the *nervous system* is the primary cause of diabetes, and the modes in which such a lesion may be supposed to act have already been sufficiently discussed. This view is not only borne out by physiological experiments, but also by clinical observation, and *post-mortem* examinations. The nature and seat of the morbid conditions in the nervous system are still matters of dispute, but doubtless they differ in different cases. Dickinson has described special lesions in the nerve-centres, which will be presently alluded to. Pavy inclines to the opinion that some kind of textural change in the brain stands at the foundation of diabetes, and considers that this disease may arise in either of two ways, namely, from a lesion affecting or involving a loss of power in vaso-motor centres, with consequent direct paralysis of the muscular coat of the vessels; or a lesion in some part or other of the cerebro-spinal system, leading to an inhibitory influence being exerted upon these centres. This authority suggests an alliance between diabetes and locomotor ataxy or progressive muscular atrophy, as regards the progressive character of the disease.

It seems highly probable that the exact pathology of diabetes differs in different cases, and in the present state of knowledge at any rate no exclusive view can be adopted. At the same time there is every reason to believe that in a large proportion of cases the nervous system is primarily at fault, though the nature of the lesion may vary.

**Exciting causes.**—The exciting causes of diabetes in individual cases are usually by no means evident. Among those to which the disease has been mainly attributed are exposure to wet and cold; drinking cold water when the body is heated; abuse of alcohol, sugar, and starchy substances; powerful emotional disturbance, or excessive mental work; injuries to the head, spine, and various other parts, or general concussion of the body; and organic diseases affecting certain parts of the nerve-centres or the sympathetic trunk. In some cases the disease seems to have followed the prolonged action of depressing mental

causes, combined with influences which tend to impair the health, as, for instance, grief and anxiety connected with long-continued watching over the sick; or worry and close confinement in business. Occasionally diabetes has appeared to be the sequela of some acute febrile disease; and in some instances it is associated with gout.

**Predisposing causes.**—As regards predisposing causes, diabetes is most common in adults, from 25 to 65 years of age, especially during the period of development and activity of the sexual functions; in males, after the period of puberty; and in persons residing in cities and manufacturing districts. In some instances there unquestionably appears to be some hereditary predisposition; or the disease tends to run in families.

**ANATOMICAL CHARACTERS.**—There are no morbid appearances at present recognized as peculiar to diabetes, but the most important lesions which have been observed are those associated with the *nervous system*. In some cases there is obvious organic disease, such as a tumour involving the medulla or pons, or pressing upon the sympathetic trunk. In others minute microscopic examination is necessary in order to detect the pathological changes. Dr. Dickinson has described peculiar alterations in various parts of the nerve-centres, especially about the medulla and pons, in the way of dilatation of the arteries, followed by degeneration and destruction of the nerve-elements around these vessels, leading to the formation of excavations, which may be of some size. Other observers have, however, failed to detect any such changes after careful and minute examination in cases of diabetes of the most typical character, and Drs. Frederick Taylor and Goodhart have especially disputed the validity of Dr. Dickinson's conclusions. Most authorities deny that there is any special morbid condition of the liver, though some observers have described peculiar changes in its general or microscopic characters. The kidneys are often diseased, but this is a secondary result of the diabetes, the most frequent morbid state being some form of Bright's disease. The lungs are also frequently affected, the lesion being usually some form of phthisis; but occasionally acute pneumonia of a low type, or gangrene supervenes. The heart is generally small, and wanting in tone. There is a tendency to serous inflammations of a low character; and also to inflammation of other structures, ending in suppuration or gangrene. Some observers have recognized an important relation between the condition of the pancreas and diabetes. Thus it has been found atrophied and shrunken, small, hard, and bloodless; the seat of fatty change; or hypertrophied and enlarged. The stomach is generally dilated, its mucous coat being thickened and softened, and its muscular coat sometimes hypertrophied.

**SYMPTOMS.**—Clinically cases of diabetes differ remarkably in their severity, one class presenting but slight symptoms; another group being accompanied with marked local and constitutional disturbance. In a typical example of the disease the symptoms may be arranged under the following heads:—

1. **Urinary organs and Urine.**—Micturition becomes more and more frequent, and the urine is increased in quantity. This fluid is also irritating in quality, and hence often causes in the male a sense of heat or burning along the urethra, or sets up slight inflammation, excoriation, or even ulceration about its orifice; while in the female the vulva is frequently much irritated, and may be the seat of troublesome itching, or of erythema or eczema. These conditions may cause the



patient to indulge in masturbation. Pain and tenderness are often felt over the region of the kidneys. The quantity of urine may amount to 8, 12, 20, or even 30 pints in the 24 hours. It is usually very pale, clear, and watery, the more so in proportion to its quantity; possesses a sweet taste, and occasionally a sweetish odour; has a high specific gravity, this being generally from 1030 to 1040, but it may range from 1015 to 1060 or more; ferments rapidly if kept in a warm place, with the formation of torulæ, at the same time becoming opalescent or depositing a sediment; while it yields more or less sugar to the usual tests. (See EXAMINATION OF URINE.) Many different statements have been made as to the proportion of urea and uric acid present in diabetic urine; probably these constituents are as a rule absolutely increased, but relatively to the water they are diminished. The quantity of water is generally about equal to that taken into the system. The amount of sugar discharged is greater after food has been taken, especially after such articles as contain much sugar or starch, being considerably less when the diet is restricted to animal food. In any pyrexial condition it becomes greatly diminished, or may even disappear altogether, and no glycogen is formed in the liver under these circumstances. The proportion of sugar usually present ranges from 8 to 12 per cent., and from 15 to 25 ounces are discharged daily on the average; but the quantity may vary from less than an ounce to two pounds or more. The urine may contain albumen, or occasionally a little blood; it is also stated to yield fat sometimes, or to resemble chylous urine in its characters.

**2. Digestive organs.**—A very constant, though not invariable symptom of diabetes is insatiable thirst, attended with a dry, parched, and clammy condition of the mouth and throat, due to the presence of sugar in the blood, which creates a demand for much liquid. In many cases also there is excessive appetite, but disinclination for food is not uncommonly observed. The tongue generally presents a peculiar irritable, red, clean, cracked, and dry appearance; it may, however, be moist and furred. Sponginess of the gums, with a tendency to bleeding, and rapid destruction of the teeth are frequently noticed. The saliva contains sugar, and is said to be very acid sometimes, owing to the conversion of this sugar into lactic acid. The breath has in some cases a distinctly sweet or ale-like odour; in most cases which end fatally with nervous symptoms it yields a smell resembling that of stale vinegar or stale beer (Balthazar Foster). Dyspeptic symptoms are of common occurrence, such as epigastric fulness or sense of sinking, flatulence, and gaseous or acid eructations. As a rule the bowels are constipated, with pale, dry, and spongy stools; but there may be diarrhœa or dysenteric symptoms, especially towards the close of the case.

**3. General symptoms.**—The aspect of the patient is in many instances strikingly characteristic of diabetes, the prominent features being emaciation, often extreme, involving not only the fat but also the muscles, which feel flabby and soft; a peculiar dry, harsh, scurfy condition of the skin; and a distressed, worn, and suffering expression of countenance. The patient feels very weak and languid, is often chilly, and is indisposed for any bodily or mental effort, at the same time complaining of pains and soreness or aching in the limbs, and these sensations are sometimes very prominent. Slight œdema of the legs is frequently observed, and occasionally dropsy affects other parts. Sometimes the temperature is markedly reduced, and in any pyrexial condi-



tion it does not become nearly so much elevated as it would otherwise be. Sexual inclination and power are commonly greatly diminished or lost. The mental condition and disposition become usually much altered in established cases of diabetes, as evidenced by decline of mental vigour; disposition to lassitude or drowsiness; lowness of spirits; petulance and irritability; or decline in firmness of character and moral tone. Temporary dimness of vision is not an infrequent phenomenon. The blood contains sugar, which is also found in the various secretions.

**4. Complications.**—Most of the complications of diabetes have been already alluded to in the account of its morbid anatomy, the most frequent symptoms coming under this head being those indicative of pulmonary phthisis. Here may also be mentioned the not uncommon occurrence of boils and carbuncles; of chronic skin-affections, such as psoriasis; of gradual permanent blindness, from atrophy of the retina; and of cataract, the last being almost always of the soft kind, and attributed to imbibition of sugar, which, it is said, has been detected in the lens.

**COURSE AND TERMINATIONS.**—The precise clinical history of diabetes varies much in different cases, as regards the intensity and exact combination of the symptoms just described, and the rapidity of the progress of the disease. Ordinarily the course is essentially chronic, the symptoms setting in very insidiously, and becoming gradually but progressively worse. It may happen that for a considerable time the advent of diabetes is only indicated by slight general symptoms, such as debility, languor, and some loss of flesh. Occasionally the disease runs an acute course; or it may exhibit remissions from time to time, and may certainly be often materially influenced in its course by treatment, in some cases a cure being effected. It is frequently observed that the symptoms are more intense at the early stage than subsequently. Most cases ultimately prove fatal, and towards the close the symptoms often change considerably in their characters, the urine and sugar diminishing in quantity; albuminuria setting in; there being complete disgust for food; and hectic or colliquative diarrhœa supervening. The fatal result usually arises either from gradual exhaustion; from blood-poisoning, leading to stupor ending in complete coma, or occasionally to delirium or convulsions; or from complications. Now and then death takes place quite suddenly, or with great rapidity. The cause of this rapidly fatal termination in cases of diabetes is not positively known. It has been attributed to uræmia; to some affection of the nervous or respiratory systems; to thickening of the blood, so that it cannot circulate; to lipæmia—fatty or milky blood, and fat emboli in the brain; and to the chemical transformation of sugar within the body, leading to the production of poisonous compounds. Dr. Balthazar Foster thinks that acetonæmia is the actual cause of the comatose condition. He is of opinion that all patients who are seriously diabetic have probably a small quantity of acetone formed in their economy, and under certain circumstances this rapidly undergoes great increase, and gives rise to the acute symptoms which lead to sudden death. He thinks it highly probable that alcohol is also formed in the system. Two cases terminating in this manner have come under my notice, and in one case the result was clearly traceable to suddenly cutting off the supply of water.

**DIAGNOSIS.**—When diabetes is well-established, there ought to be no difficulty in recognizing the disease. The urinary symptoms and characters of the urine; the symptoms referable to the alimentary canal;

and the general condition are highly characteristic. The rule of always examining the urine carefully when the health is persistently out of order, and especially if there are the slightest symptoms suggestive of this complaint, will often lead to a diagnosis at an early period. If a patient complains of languor and debility, or of pains in the limbs, or is losing flesh without any obvious cause; if much irritation is experienced about the external genital organs, or if these parts are affected with erythema or eczema; or if a child is detected masturbating, diabetes should always be borne in mind. The mere finding of a trace of sugar in the urine is not, however, evidence of the presence of diabetes. It must be in some quantity, persistent, and attended with polyuria. Seldom can any definite diagnosis be made of the exact morbid condition upon which diabetes depends. The possibility of a comatose condition being due to diabetes must be borne in mind in any case of unconsciousness of which the cause is not evident. The smell of the breath, and examination of the urine ought to clear up any difficulty in the diagnosis under such circumstances.

**PROGNOSIS.**—Confirmed diabetes is a very serious disease, a large proportion of cases ending fatally, and their average duration is stated to be about from one to three years. In many instances, however, much improvement may be effected; and in some cases recovery can be brought about. The chief circumstances which influence the prognosis are age, the prospect being worse in very young than in old persons; the general condition of the patient, the disease being much less serious in stout individuals; the cause of the complaint; the amount of sugar and urine passed; the severity of the general symptoms; the presence or absence of complications, as well as their nature; the duration and progress of the case; and the results of treatment, as well as whether this is properly carried out by the patient. Any person suffering from diabetes should be particularly cautioned against exposure to wet and cold, and other recognized causes of disease.

**TREATMENT.**—Cases of diabetes must necessarily call for much diversity in their management, and no case ought to be subjected to treatment until its nature and the conditions present have been ascertained as fully as is practicable; there are, however, certain general principles to be followed, to which attention will now be directed. At the outset it is most important to impress upon patients that they must be prepared to place themselves under strict discipline and guidance, and that much of the success of treatment will depend upon their own conduct.

1. The first indication almost universally recognized in the treatment of diabetes is **regulation of the diet**. The object aimed at is to prohibit, or to restrict within proper limits, the consumption of such articles as contain sugar or starch, especially ordinary bread or flour; sugar in any form; honey; vegetables and fruits containing starch or sugar, namely, potatoes, peas, beans, carrots, turnips, parsnips, strawberries, raspberries, plums, gooseberries, currants, apples, pears, &c.; rice; prepared varieties of starch, such as arrowroot, sago, macaroni, tapioca, and vermicelli; shell-fish, and the soft parts of crabs and lobsters. Animal food, including meat, poultry, game, and fish, should be the main diet, with the exception of liver. Dr. Lauder Brunton has suggested the use of raw meat, finely chopped, and mixed with pepper and salt. The chief substitutes for bread which are employed include bran-cake or biscuits, gluten bread, almond rusks and



biscuits, or very thin slices of bread toasted until they are almost black. Eggs, butter, cheese, broths, good soups, and jellies are admissible; also vegetables not containing sugar or starch, such as cabbage, Brussels sprouts, broccoli, cauliflower, lettuce, cress, mustard, and celery.

The question of *drink* is one of much moment. Milk is theoretically contra-indicated in diabetes, because it contains a quantity of sugar, but it has been found in some instances that when given in moderate quantities milk is not injurious, and may even prove highly beneficial. Therefore it is allowable to try the effects of a regulated amount of this article of diet in any individual case, being guided accordingly in its subsequent administration. The milk may be mixed with lime-water or soda-water. Cream may be given in abundance, if it agrees with the patient. Dr. Donkin has advocated the treatment of diabetes entirely by skimmed milk, given in quantities of from six to eight or even twelve pints daily, and continued for several weeks if necessary, no other food or medicine being allowed. My experience of this treatment is anything but favourable, but I have found much benefit result from giving a considerable quantity of skimmed milk daily, as much as three or four pints, along with other food. Dr. Lauder Brunton has recommended the use of butter-milk. The balance of evidence is decidedly against the consumption of *alcoholic stimulants* to any considerable extent in diabetes. A small quantity is frequently serviceable, those forms of stimulant being employed which are most free from sugar, namely, dry sherry, bitter ale, brandy or whisky well-diluted, claret, and burgundy. Tea and coffee without sugar may be allowed; and also cocoa made from the nibs, provided it agrees. It is not desirable to restrict the quantity of liquid too much, but it must be moderated so far as the feelings of the patient will permit. Most injurious is it to cut off the supply of liquids suddenly, and I have known a rapidly fatal issue result from this cause. Thirst may be relieved by iced water; by acid drinks, of which a solution of phosphoric acid has been much recommended; or by a solution of cream of tartar. Prout affirmed that tepid liquids relieve thirst better than cold. The Bristol Hot-wells, Carlsbad, and Vichy waters are said to have some direct influence upon diabetes, in addition to being serviceable as a drink, whilst the Carlsbad waters have also an aperient action. A mineral water named the Bethesda water has obtained considerable reputation in the treatment of diabetes, but on no adequate grounds.

It is highly important to attend to the following points in regulating the diet in diabetes:—1. The change should be brought about gradually and not suddenly. 2. Frequent variations in the food should be made amongst those articles which are permissible. 3. In many cases it is necessary to watch carefully that the regimen laid down is strictly adhered to, especially during the early period of treatment, and in dealing with ignorant patients. 4. Every individual case must be studied for itself, and the advisability of persevering in the restricted diet or not must be judged by the results. In some instances, where there is much loathing of food, a little bread is often of great service. Again, if a fair trial of the recognized diet does not seem to lead to any improvement, or if the general condition is becoming worse, as may especially happen when the disease is far advanced, it may be desirable to let the patient follow his own inclinations, guided by intelligence and common sense; sometimes also patients cannot possibly take the prescribed food, and then a mixed diet must be permitted.



Allusion may be here made to the *saccharine* treatment of diabetes, in which sugar and honey are administered in considerable quantities, any diet being allowed. This has been proved to be decidedly injurious in the majority of cases.

2. **General hygienic management** is highly important in diabetes. The patient should be completely clad in flannel or other warm material; and should have two or three warm baths every week, or an occasional Turkish bath. Change of air, especially to the sea-side, with sea-bathing, is useful in some cases. Regular and sustained active exercise in moderation is often of great service.

3. **Therapeutic treatment.**—Numerous medicines have been brought forward, which are supposed to have a direct curative influence upon diabetes, especially in limiting the amount of urine and sugar discharged. The principal of these include opium, given in gradually increasing doses up to gr. vi-xx daily, which certainly seems to be useful in some cases; codeia, in doses of from gr.  $\frac{1}{2}$  to gr. iij; morphia; alkaline bicarbonates; pepsine; rennet; arsenic, in the form of Fowler's solution; iodine or iodide of potassium; bromide of potassium; conia; cannabis indica; lactic acid or lactate of soda; glycerine; quinine; ergot; ether; valerian; permanganate of potash; and peroxide of hydrogen. These are supposed to act either through their influence upon the nervous system; or by promoting the decomposition and combustion of sugar; or by supplying a readily combustible substance in its place. The evidence in favour of the efficacy of most of these drugs, however, is by no means satisfactory.

4. **Symptomatic treatment** often calls for attention in diabetes, this being especially directed to the digestive organs; to the general condition and state of the blood; to nervous disturbance, in the way of sleeplessness and restlessness; and to the various complications. These conditions must be managed on ordinary principles. Iron, especially in the form of tincture of perchloride, as well as other *tonics*, are often of much service. Cod-liver oil is also valuable in many cases. It must be borne in mind that complications may considerably modify the treatment of cases of diabetes. For diabetic coma the chief measures which have been tried are transfusion; inhalation of oxygen; and the administration of medicines to check the fermentative process which develops the poison, such as carbolic acid, salicylic acid or its salts, and thymol.

## II. DIABETES INSIPIDUS—POLYURIA—POLYDIPSIA.

**PATHOLOGY AND ÆTIOLOGY.**—The ætiology of diabetes insipidus is very obscure, but the complaint seems to be allied to diabetes mellitus. The probable immediate cause of the excessive flow of urine, which is one of the chief characteristics of the complaint, consists in dilatation of the renal vessels, due to paralysis of their muscular coat, resulting from deranged innervation. The condition can be induced experimentally by irritating a spot in the floor of the fourth ventricle immediately above the auditory nuclei; by section of the great splanchnic nerve; by section of the sympathetic trunk in the chest above the origin of the splanchnic nerves; and by section of the vagus nerve and electrization of its peripheral end. It has also been attributed to injury of the nervous centres, especially resulting from blows inflicted on the

front of the skull, by which the posterior part of the brain was injured; to injuries affecting the sympathetic trunk; to organic diseases of the brain and cord, such as grey degeneration of the floor of the fourth ventricle, meningitis, and growths in the brain; to pressure upon the splanchnic nerves, the solar plexus, or the pneumogastric nerves, by tumours or aneurisms; to depressing emotions; and to hysteria, neuralgia, and other nervous disorders. The lesions in the brain have nearly always been found in the cerebello-medullary region. Among other alleged causes of polyuria should be mentioned exposure to cold; drinking cold water when the body is heated; abuse of alcoholic stimulants; violent effort and muscular strain; and previous febrile or inflammatory attacks. The complaint is much more common in children than adults. It is occasionally hereditary, or diabetes mellitus may have existed in the parent.

**SYMPTOMS.**—Diabetes insipidus is characterized by great thirst; with an increased flow of urine, which is watery and usually of low specific gravity, but does not contain any sugar or other abnormal ingredient. The quantity of urine discharged may be enormous, sometimes exceeding considerably the amount of fluid taken into the system; and the proportion of solids discharged in the twenty-four hours may be normal, excessive, or below par. Usually they are in excess, especially the urea, and occasionally a condition of so-called *azoturia* is present. Frequent micturition is often observed. Patients suffering from diabetes insipidus have been sometimes known to drink their own urine, if their drink was restricted. Occasionally they enjoy excellent health, but more commonly present more or less of the symptoms which are noticed in diabetes mellitus, especially a dry and harsh skin, loss of flesh, weakness, sensitiveness to cold, and dryness of the mouth. In most cases the appetite is not excessive, but sometimes it is voracious. Epigastric pains and constipation are often complained of. As a rule diabetes insipidus is chronic in its onset and course; occasionally it sets in suddenly. Recovery is extremely exceptional, but death usually results from some organic complication. Occasionally the fatal termination is preceded by progressive wasting and exhaustion, total anorexia, diarrhoea, and vomiting.

**TREATMENT.**—Opium, valerian, camphor, nitrate of potash, iron, ergot, iodide of potassium, arsenic, belladonna, bromide of potassium, dilute nitric acid, and pilocarpin by subcutaneous injection, are the chief medicines which have been recommended in the treatment of diabetes insipidus. Withdrawal of liquids from the diet has not proved successful. The use of the constant galvanic current, applied over the hypochondrium, or to the neck and spinal column, has been advocated. The general health and digestive functions must be attended to; and symptoms treated as they arise.

## II. LOCAL DISEASES.

IN treating of *local diseases* in the following pages, the plan is adopted of giving a summary of the general clinical characters which belong to the several organs and systems, and of the methods to be employed in their clinical investigation, before entering upon the consideration of the individual disorders to which they are liable. It may be stated once for all, however, that it is always of essential importance to study the *constitutional condition* of the patient, as this materially affects the diagnosis, prognosis, and treatment of local affections. Some of the more important symptoms will be discussed as fully as the limits of this work permit.

---

### CHAPTER I.

#### DISEASES OF THE MOUTH, TONGUE, AND SALIVARY GLANDS.

CLINICAL CHARACTERS.—Important indications are often afforded by the mouth and tongue with regard to the state of the system generally, and of the alimentary canal, but these parts may also be the seat of *local* affections, to which attention will now be directed. Their presence may be revealed by the following symptoms and signs:—

1. **Altered sensations**, such as pain, soreness, or a feeling of heat or dryness; as well as various derangements of the sense of **taste**.
2. More or less interference with the **actions** carried on in the mouth, namely, mastication, sucking, the first stage of deglutition, and articulation. These acts are also often attended with pain.
3. Changes in the **quantity or quality of the saliva**; or the escape of **unusual discharges**, such as pus, blood, &c.
4. A disagreeable odour of the **breath**, which may amount to extreme fœtor.
5. Interference with the **act of breathing** occasionally, owing to mechanical obstruction to the passage of air.
6. A change in colour, or the existence of any swelling, deposit, ulceration, or other morbid condition, as disclosed on **objective examination** of the mouth, aided by a good light. At the same time the absorbent glands in the neighbourhood should be examined.

#### I. INFLAMMATION OF THE MOUTH—STOMATITIS.

Stomatitis is a very common affection, and occurs under several forms. Its varieties are:—1. **Catarrhal**. 2. **Follicular**. 3. **Aphthous**. 4. **Ulcerative**. 5. **Parasitic**. 6. **Gangrenous**. 7. **Mercurial**.

ÆTIOLOGY.—The chief *predisposing causes* of stomatitis are:—1. Age, the different forms being far most common in infants and young



children. 2. Improper hygienic conditions, such as want of cleanliness, impure air, or an unhealthy residence. 3. Errors in diet, or an insufficient supply of food. 4. Certain unhealthy conditions of the system; or the presence of certain diseases. The different forms of stomatitis are exceedingly rife among the children of the poor, especially those living in large towns. This applies particularly to the more severe varieties of the disease, the *gangrenous* form being rarely met with except among this class of patients. Infants who are brought up by hand, or fed on artificial food, are very commonly affected; as well as those who have been suckled for too long a time, or have been nursed by an unhealthy mother. Children who are debilitated from any cause, and those prematurely born, are also very prone to this class of diseases. They are common in cases of congenital syphilis; and as complications or sequelæ of some of the exanthemata. *Thrush* is frequently associated with typhoid fever; or, in adults, with chronic wasting diseases, especially phthisis. *Gangrenous* stomatitis rarely occurs except after some acute illness, particularly severe measles.

**Exciting causes.**—1. *Local irritation* is one of the most frequent causes of stomatitis. This may arise from want of cleanliness, dentition, decayed teeth, sucking imperfectly-formed or inflamed nipples, or for too long a time; as well as from all forms of mechanical or chemical irritation, undue heat or cold, excessive smoking, wounds, ulcers, and other local morbid conditions. 2. The milder varieties may be dependent upon *disorder of the alimentary canal*. Repeated *follicular* stomatitis in adults generally indicates some gastric derangement. 3. The presence of some *poison in the blood* frequently excites inflammation in the mouth. This partly explains its occurrence in the acute specific fevers. Certain *metallic* poisons, however, are those which ordinarily act in this way, especially mercury. 4. Catarrh of the mouth may be due to *extension of inflammation* from neighbouring parts. Hence it may be associated with erysipelas of the face; or with throat-affections. 5. *Contagion* originates some forms of stomatitis. *Thrush* can be propagated by direct transplantation of the fungus which causes it, though it does not usually spread in this way, being probably due to the presence of the spores in the air, which in the mouth find favourable conditions for their development in the decomposing food and epithelium, their growth being aided by the parts being kept at rest, and by want of cleanliness. Some authorities believe that *ulcerative* stomatitis is contagious. Dr. Sansom has described minute translucent bodies in active movement in the blood and excretions during life in a case of gangrenous stomatitis, which he considers to be of the nature of organisms. Inoculation with the infected blood induced septicæmia, with the manifestation of similar characteristic motile particles.

**SYMPTOMS.**—The clinical history of each variety of stomatitis will need a brief description.

1. **Simple or Catarrhal.**—When acute, this form of stomatitis begins as small bright red patches on the inside of the cheeks, or at the angles of the mouth. Ultimately by extension and coalescence of the patches the whole mucous lining may be involved. There is more or less swelling of the affected parts. At first the surface is dry, but soon excessive secretion forms, containing many imperfect cells. Superficial erosions or ulcerations are often produced. The subjective sensations are pain or soreness, heat, a slimy feeling in the mouth, and impaired or unpleasant taste. The breath is often disagreeable. Generally the

alimentary canal is out of order, as evidenced by a furred tongue, loss of appetite, and, in children, by disordered bowels and flatulence. Children are also irritable and sleepless. Catarrh of the mouth often occurs as a chronic affection.

2. **Follicular or Papillary.**—At first little red raised spots are seen, which feel hard; these are due to enlarged and obstructed mucous follicles. As a rule they soften and burst, discharging their contents, and leaving small, circular, well-defined ulcers, with some surrounding redness. A good deal of soreness is complained of.

3. **Aphthous or Croupous.**—Much confusion has existed with regard to what is meant by *aphthæ*, but it seems best to restrict the term to certain small ulcerations, which have a special mode of origin. They commence as little whitish or whitish-yellow spots on the lips, cheeks, palate, or tongue, which are often in considerable number, and may become confluent. More or less redness surrounds each spot. They are generally considered to be vesicular, and to contain a fluid, which usually becomes opaque, while the vesicles ultimately rupture. Some authorities, however, regard them as solid exudations under the epithelium, of a croupous nature, which become detached from the circumference towards the centre, leaving superficial ulcerations. Aphthous stomatitis is usually attended with much pain, rendering sucking, mastication, deglutition, or even speaking difficult to perform. The buccal secretion is increased, and there may be much salivation. The breath has often a very disagreeable smell. Infants are usually feverish and restless, even for some days before the *aphthæ* appear. They refuse nourishment, but are thirsty. The tongue is furred, and diarrhœa or vomiting may be present.

4. **Ulcerative or Diphtheritic—Gyngivitis Ulcerosa.**—This is a form of inflammation which usually ends in extensive and unhealthy ulceration, and it may assume an epidemic character. It is regarded by some as being of a diphtheritic nature. As a rule it begins on the margin of the lower gums in front, but may extend backwards, or to the lips, cheeks, or tongue. The gums appear much congested, swollen, and spongy; bleed very readily; and seem to be separated from the teeth. Soon a deposit is observed, in the form of membranous-looking patches, at first whitish, but speedily becoming grey or even black. Tolerably firm and adherent at the outset, and leaving a bleeding surface when detached, the substance shortly becomes soft and pulpy. It has been stated that the mucous membrane itself is involved, a diphtheritic slough being formed. The patches usually separate, leaving irregular ulcers, which may spread and run together so as to give rise to an extensive ulcerated line or surface. The margins of the ulcers are raised, the surrounding membrane being congested, swollen, and oedematous. They are usually not deep, and their surface is covered with a pulpy yellowish substance. If properly treated, they generally heal quickly, but in some cases serious results follow, the teeth dropping out, and the jaws becoming carious or necrosed.

The subjective symptoms are generally severe. There is a great deal of pain, increased by movement of the jaws or other local irritation; hence there is much difficulty in chewing or swallowing. The saliva is very abundant, and is frequently mixed with blood and other matters. The breath is very fœtid. Often the glands in the neighbourhood are enlarged and tender. In most cases the constitutional symptoms are but slight.



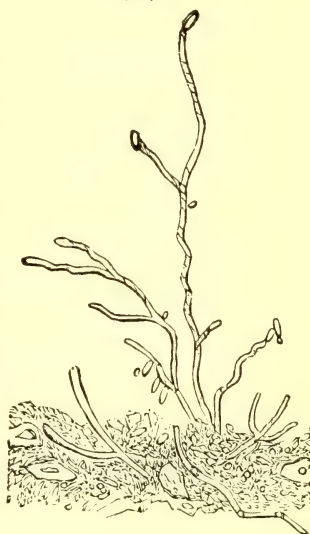
5. **Parasitic or Fungous—Thrush—White Mouth.**—By these and other names a variety of stomatitis is described, which depends upon the presence of a parasitic fungus, the *oidium albicans* (Fig. 19). Gravit states that this fungus is a yeast, probably the *mycoderma vini*. At first red patches form, on which whitish points appear, which may extend and coalesce into considerable patches of variable thickness. They look like curdled milk, being of a soft consistence, and soon becoming easily detached. They consist of epithelium and fat, in which are imbedded the sporules and filaments of the fungus. The deposit first appears generally about the angles of the mouth, but may be noticed on any part of this cavity, and even extends occasionally to the pharynx, larynx, œsophagus, or, very rarely, to the stomach. There is necessarily a good deal of pain and soreness about the mouth, which is hot and dry, the saliva being diminished in quantity at first.

Very young infants are subject to thrush as a distinct affection, being preceded by some slight febrile disturbance, and attended with digestive disorders, evidenced by vomiting, diarrhœa, pain, tenderness, and swelling of the abdomen, and irritation about the anus. In most cases, however, the condition is associated with some pre-existing disease, especially certain acute specific fevers, and chronic exhausting diseases, such as phthisis. Under these circumstances there may be no symptoms. In connection with the acute specifics, the presence of thrush does not add to the danger, but in the chronic affections it is commonly a sign of approaching death.

6. **Gangrenous — Cancrum Oris — Noma—Water Canker.**—This is a very

rare, but exceedingly dangerous form of stomatitis. It begins insidiously, and almost invariably first affects one of the cheeks, attacking its inner surface. When the patient comes under observation, there is usually a circumscribed hard swelling in the cheek, with surrounding œdema. The skin covering it is tense, shining, and hot, generally red, the colour shading off from the centre, which is bright; sometimes the surface is pale or mottled. The mucous membrane is merely reddened at the outset, but soon becomes discoloured and gangrenous, and a vesicle often rises upon it. Then a small irregular ulcer forms, with jagged, red or livid edges, and a sloughy surface. After a time the central spot of bright redness becomes livid, and finally black, being converted into a dry slough, which extends rapidly. At the same time the gangrene is spreading internally, so that finally the entire cheek may be affected, or even one-half of the face or more, while the gums, lips, and tongue are also frequently involved to a variable extent. When the sloughs separate the mouth is opened up, the teeth often drop out, and the bones may be exposed and necrosed. A hideous excavation with ragged gangrenous edges is left, which may still go on spreading. If the destructive process is checked, the surface may clean, granulate, and cicatrize, but great deformity often results, with adhesion of various structures.

FIG. 19.

*Oidium albicans.* (Fairlie Clarke.)



The gangrene does not necessarily spread to the extent just described. It may only produce a hole in the cheek, which ultimately closes up, or remains as a fistulous opening. The glands and tissues around are always infiltrated, swollen, and hard.

One of the most striking features of this disease consists in the fact that pain and tenderness may be either very slight, or altogether absent. A large quantity of saliva flows from the mouth, which is extremely foetid, and mixed with blood and gangrenous discharges. Serious hæmorrhage does not occur, because the vessels are blocked up by coagula. The breath has an excessively foul, gangrenous odour.

The *general* symptoms vary considerably, but it is often observed that even when the disease is extensive, they are by no means severe. Much will depend upon the previous condition of the patient. There is not much fever as a rule, the skin being cool; and the strength may be fairly maintained for a while, at the same time food being taken eagerly. The pulse at first is rather frequent. As the disease progresses there is a tendency to prostration, the patient ultimately becoming extremely low and asthenic, with a very feeble and small pulse. Food may be taken to the last, and there is much thirst. Diarrhœa often sets in. The patient frequently becomes delirious or drowsy in fatal cases. Death may result from septicæmia or asthenia.

**7. Mercurial.**—The first effect of mercury is to cause redness and tumefaction of the gums, which feel tender and bleed readily; while the patient experiences a peculiar metallic taste; the saliva is increased; and the breath has a characteristic unpleasant odour. Afterwards superficial greyish sloughs and ulcerations form along the margins of the teeth, and the gums become detached, the teeth loosening or even falling out. There may be extensive inflammation of the mouth and tongue, ending in ulceration, suppuration, or actual gangrene. Salivation becomes very profuse, various discharges being mixed with the saliva. The salivary and lymphatic glands and other neighbouring structures are swollen and painful; while much pain is experienced in the mouth and face, with difficulty in moving the jaws, in swallowing, and in speaking. Only slight constitutional symptoms are usually observed.

Although the subject has already been noticed under syphilis, it is necessary to refer here to the effects of stomatitis, especially mercurial, upon the teeth. This complaint leads chiefly to defective formation of enamel, and the teeth become of bad colour, eroded, pitted or "honeycombed," and often show a transverse furrow crossing them at the same level. Mr. Hutchinson regards the first permanent molars as the test teeth of infantile stomatitis, and next to these the incisors. M. Magitot maintains, however, that the enamel defects are due, not so much to stomatitis, as to interference with development brought about through the nervous system, in connection either with infantile convulsions or with severe illness during early life.

**DIAGNOSIS.**—The different forms of stomatitis are readily recognized when the mouth is properly examined. In infants it is highly important to bear these complaints in mind, and to look to the mouth should these subjects appear to be ailing. It must also be mentioned that the *ulcerative* and *gangrenous* varieties sometimes set in and extend very insidiously, giving rise to little or no evident disturbance. Fœtor of the breath may lead to their discovery.

**PROGNOSIS.**—Most cases of stomatitis can be readily cured, if appropriate treatment is carried out. The *ulcerative* and *gangrenous* forms of the disease may prove extremely serious or even fatal. In chronic wasting diseases, especially phthisis, the occurrence of *thrush* may indicate a speedily fatal termination.

**TREATMENT.**—The indications for the treatment of the various forms of stomatitis may be summed up as follows:—

1. **Hygienic conditions** must be properly observed, and duly regulated in every respect. 2. Particular attention is required with regard to **diet**, especially in the case of infants. If they are suckling, care must be taken that they are not fed too frequently or excessively; and that the nipple is properly cleansed, and in other respects satisfactory. At the same time the mother's health must be looked to, and she must be prevented from using irritating articles of food. If an infant is brought up by hand, good milk should be given, while the feeding-bottle must be kept scrupulously clean, and used only at regular intervals. Inquiry should also be made as to any artificial diet employed, as this is often of a very irritating nature. 3. The state of the **alimentary canal** frequently needs correction. *Aperients* are called for in many cases, such as castor-oil, a dose of jalap with calomel, or rhubarb with magnesia or grey powder. *Antacids* are also valuable, especially lime-water with milk, carbonate of soda or magnesia, or chalk. 4. A most important indication is to remove or avoid all sources of **local irritation**, such as dentition, bad teeth, and excessive smoking. In the case of infants the mouth must be properly cleansed. In the treatment of *mercurial* stomatitis of course the withdrawal of the drug is essential.
5. **Local applications** are often of great value. In *simple* stomatitis nothing is required, unless there should be much mucus on the surface, when it is desirable to wash the mouth out with a weak solution of carbonate of soda, chlorate of potash, or glycerine of borax. In the *aphthous*, *follicular*, and *ulcerative* forms, a solution of chlorate of potash is very valuable, either employed as a mouth-wash, or directly applied with a camel's hair brush. If there is much irritation *demulcent* washes afford relief, such as thin mucilage. A weak solution of Condyl's fluid is serviceable for the purpose of checking fœtor. Subsequently *astringent* applications are often called for, especially alum in the form of solution or powder. The direct application of nitrate of silver to ulcers may be requisite, either in the solid form or as a solution. In the treatment of *thrush* various local remedies are useful, namely, a solution of sulphite of soda (3 i ad 3 i); borax and honey or glycerine; vinegar and water; creosote; bromide of sodium with glycerine and water; or solution of chlorate of potash. *Cancrum oris* calls for energetic local treatment. The gangrenous surface must be at once freely and effectually destroyed by strong nitric or hydrochloric acid, the former being preferable, and it may be necessary to repeat the application. *Antiseptic* mouth-washes should be freely used, such as one containing Condyl's fluid, chlorine, carbolic acid, or carbolate of glycerine, which may also be employed as dressings. Chlorate of potash solution is likewise very valuable in this form of disease. Poultices externally are also required, these being changed frequently, and sprinkled with some antiseptic. 6. **General treatment** is called for in some instances. In the majority of cases of ordinary stomatitis no general treatment is required, but if the health is lowered from any cause, it is important to use measures for its improvement. When there is extensive ulceration, *tonics* and tincture of steel, along with nutritious



diet, are often of essential service; and the internal administration of chlorate of potash also leads to most satisfactory results. In *gangrenous stomatitis* one of the most necessary parts of the treatment consists in keeping up the patient's strength in every possible way, by means of nourishing soups, wine or brandy, ammonia and decoction of bark, mineral acids, or quinine with tincture of iron. Chlorate of potash solution should be employed as a drink at the same time. The internal administration of sulpho-carbolate of sodium, sulphite of sodium, or other *antiseptics* is recommended in this form of the disease.

## II. GLOSSITIS.—PARENCHYMATOUS INFLAMMATION OF THE TONGUE.

Glossitis may occur either as an *acute* or a *chronic* affection, and each form needs to be briefly considered.

**A. Acute glossitis.**—This is an inflammation of the actual substance of the tongue, leading to an exudation in the midst of its muscular fibres, and in exceptional instances involving the latter also. It is a rare, but very acute and dangerous affection.

**ÆTIOLOGY.**—1. Some *direct irritation* is almost always the cause of glossitis, which may result from mechanical injury; swallowing boiling liquids; the action of acrid or corrosive substances; or a sting of the tongue by some insect, especially the bee and wasp. 2. It is very rarely due to some *poison* in the system, either mineral (mercury), animal, or vegetable. 3. Occasionally glossitis occurs as a *complication* or *sequela* of one of the exanthemata. 4. In extremely rare cases it originates from *extension of inflammation* from neighbouring parts, such as the tonsil.

**SYMPTOMS.**—The entire tongue is usually implicated, and presents the following characters:—The organ is enlarged, so that the mouth cannot contain it, and it may even project some distance beyond the teeth, being indented at the sides, and the pressure exerted upon it may lead to its ulceration. The surface is dark red, generally smooth, shining, and tense, but it may be fissured. The dorsum is covered with fur, which tends to be brownish. Owing to its protrusion and exposure, the tongue soon becomes dry. If resolution does not speedily take place, small abscesses are liable to form in the substance of the organ, which coalesce, and finally burst if they are not opened. Rarely gangrene occurs. The tongue may remain large for a considerable time. Acute glossitis is attended with marked pain and tenderness, a feeling of heat, and other uncomfortable sensations. All the functions of the tongue are necessarily greatly interfered with. Occasionally, by causing mechanical obstruction, or by pressing upon or setting up œdema of the larynx, the enlarged organ seriously impedes respiration, and may even threaten asphyxia. There is much salivation; while the breath is very offensive. The glands and tissues around are usually inflamed; and the face may appear tumid and congested, in consequence of pressure upon the jugular veins.

The *general* symptoms are severe in most cases of glossitis, there being much inflammatory fever, with marked restlessness. Nutrition becomes greatly impaired, and signs of imperfect aëration of the blood may supervene.

**DIAGNOSIS.**—The appearances presented by the tongue, as just described, and the accompanying local symptoms, are sufficiently characteristic of acute glossitis.



**PROGNOSIS.**—Acute glossitis is at all times a serious affection, but especially when it results from severe local irritation. It may lead to speedy suffocation. The formation of abscesses increases the gravity of the prognosis.

**TREATMENT.**—The first thing to be done in treating glossitis is to neutralize any cause of irritation, if possible, as, for instance, the sting of a wasp, which should be immediately touched with solution of ammonia. For severe glossitis the best treatment seems to be to make free and deep incisions along the upper surface of the tongue. In milder cases the application of a few leeches about the angles of the jaw is recommended. Ice should be constantly given to suck, and the parts must be kept moist. *Saline aperients* may be administered, if required. Ammonia and other stimulants are often of great service. When food cannot be swallowed, it may be necessary to administer nutrient enemata at regular intervals. If asphyxia is threatened, it is sometimes requisite to perform laryngotomy or tracheotomy. Incisions must be made as soon as there are signs of abscesses having formed.

**B. Chronic glossitis** is occasionally met with, either as a sequel of the acute form, or as the result of some chronic irritation. It may affect the entire tongue, but is usually partial, the organ presenting indurated fibrous patches, especially on its margins. A peculiar form is described under the term *glossitis dissecans*, in which the tongue presents deep furrows upon its surface, which tend to ulcerate.

**TREATMENT.**—As regards the treatment of chronic glossitis, compression of the tongue, ligature of the lingual arteries, or excision of the organ may be necessary, if the affection should become serious.

### III. ULCERS OF THE MOUTH AND TONGUE.

In addition to the forms of ulceration in the mouth already considered, the following may occur:—1. **Herpetic**, due to an eruption of herpes in the mouth. 2. **Variolous**, resulting from small-pox pustules. 3. **Syphilitic**. These are very frequent on the tongue. 4. **Scorbutic**, chiefly seen about the gums. 5. **Cancerous**. 6. **Irritative**, the ulcers being due to local irritation. These are also common on the tongue, often beginning as small vesicles about the tip and sides of the organ. They are generally due to irritation set up by the teeth, and may become very hard, so as to simulate syphilitic or malignant ulcers.

**TREATMENT.**—This consists:—1. In the removal of all sources of local irritation. 2. In attending to the constitutional condition. 3. In the use of local mouth-washes and direct applications, *disinfectant*, *astringent*, or *demulcent*, according to circumstances. The immediate application of nitrate of silver is often most useful. Chlorate of potash is also very valuable as a local remedy in ulceration affecting the mouth.

### IV. SYMPTOMATIC PAROTITIS—PAROTID BUBO.

**ÆTIOLOGY.**—The form of parotitis characteristic of mumps has already been described, but a brief account is required of a variety of the disease which is liable to attend certain acute affections, either as a

*complication* or as a *sequela*. It is frequent during severe epidemics of typhus fever, but may be met with also in connection with small-pox, measles, scarlatina, cholera, pneumonia, and other complaints. Sometimes it arises by *direct extension*, as when it follows erysipelas of the face.

**ANATOMICAL CHARACTERS.**—Symptomatic parotitis differs from the idiopathic form in its great tendency to end in suppuration, though this is not an invariable result, for resolution may take place. After a period of congestion and tumefaction, a substance collects in the ducts, which soon changes into pus. The lobules break down in the centre, and either form a number of distinct abscesses, or run into one large accumulation of matter, the cellular tissue which separates them being destroyed. The parts around may be extensively involved, cellular tissue, muscles, periosteum and bones; and the inflammation may either extend to the cerebral meninges or to the brain itself, as well as to the ear. Thrombi sometimes form in the neighbouring veins, and these may lead to embolism and septicæmia. Gangrene occasionally occurs.

**SYMPTOMS.**—These are usually by no means marked at the commencement of parotid bubo, and the inflammation often advances very insidiously. When it ends in suppuration, the skin covering the parotid region becomes red, and prominent fluctuating points appear. If the pus is not evacuated externally, it may be discharged into the external meatus, pharynx, or mouth; or may find its way to the lower part of the neck, or even into the thorax. The general symptoms are usually of a low adynamic type.

**TREATMENT.**—The *local* treatment should consist of frequent poulticing, and the repeated use of fomentations; as soon as signs of suppuration appear, proper incisions should at once be made. Internally *stimulants* and *tonics* are generally required.

## V. CHRONIC ENLARGEMENT OF THE PAROTID.

Occasionally the parotid is chronically enlarged, either as the result of previous inflammation; or from the formation of cancerous or other growths. This may give rise to marked local pressure-symptoms. The enlarged organ usually needs surgical interference.

## VI. DISORDERS OF SALIVA.—SALIVATION.

**VARIETIES.**—The chief disorder affecting the saliva is *an increase in its quantity*, constituting, when considerable, the symptom named *salivation* or *ptyalism*. The secretion is often *diminished in quantity*, giving rise to dryness of the mouth, or more or less thirst, as in the febrile state, diabetes, certain morbid conditions of the digestive organs, or from the effects of opium, belladonna, and other drugs. Deficiency of saliva unquestionably helps in the production of dyspeptic symptoms in some cases. With regard to the *quality* of the saliva, observations were made by Dr. Samuel Fenwick, who brought the results before the Medico-Chirurgical Society. He found a yellow colouring matter in the saliva in every case of jaundice which he examined; and in one case where

there was an intense bitter taste without jaundice, he discovered traces of the biliary salts by the ordinary tests. This observer also investigated as to the proportion of sulpho-cyanide of potassium in different diseases. He found that its amount was not affected by decayed teeth or by smoking. It was almost always deficient in cases of obstructive jaundice; and Dr. Fenwick considers that the quantity of sulpho-cyanide depends on the amount of bile which reaches the intestine. The quantity of food taken affected and regulated the amount of sulpho-cyanide. It was always deficient in œsophageal stricture and cancer of the stomach; in persistent vomiting, diarrhœa, and dysentery; in cases of severe atonic dyspepsia; and in all cases of chronic disease where the appetite was very bad. It was in excess in fat persons, and in those who were gaining flesh; deficient in those who were thin or rapidly losing weight. The sulpho-cyanide was greatly in excess in all cases of acute rheumatism, reaching the maximum in the second week; also in excess in acute gout, and in most persons liable to "bilious headaches." An excess was found in the early stages of all inflammatory disorders, as gastric catarrh, acute pleurisy, renal disease, and phthisis, but much below the average in the later stages of these diseases.

**Salivation.**—Increased flow of saliva is a frequent and troublesome symptom, and calls for special consideration. It may be brought on by a variety of causes. In some cases the secretion is not actually formed in excess, but is allowed to flow from the mouth instead of being swallowed, so that it appears to be produced in abnormal quantity. The amount discharged varies greatly, but it may be exceedingly large, either continually running from the mouth, or causing the patient to be perpetually spitting or swallowing, or to saturate several handkerchiefs daily. The fluid is not quite identical in composition with healthy saliva, and may be mixed with various morbid materials. It contains some mucus, with numerous epithelium cells. Its reaction is alkaline, and it yields a good proportion of fat, but after a time little or no ptyaline or sulpho-cyanide of potassium can be detected. Albumen is sometimes present. Digestion is generally impaired; and marked emaciation may be produced. Sometimes the fluid is ejected from the stomach in considerable quantity, after having been swallowed.

**ÆTIOLOGY.**—1. More or less salivation accompanies the various sources of *local irritation* in connection with the mouth already considered, such as stomatitis or ulcers; being also produced by irritating substances taken into the mouth. These act by causing reflex excitation. 2. *Reflex irritation* through other nerves often induces ptyalism. Thus it may be associated with throat-inflammations, many diseases of the stomach and pancreas, or worms in the intestines; while it is a symptom very commonly observed in cases of pregnancy. 3. In certain *nervous diseases* salivation is not uncommon, as in various forms of insanity, hydrophobia, hysteria, paralysis, and neuralgia of the face. In some of these affections it is produced in a reflex manner; in others as the result of some direct cerebral influence. 4. Certain *metallic* and *vegetable* substances, when taken for some time, induce ptyalism, by causing local irritation, and also by directly influencing the secretion of saliva. Of these the most important is mercury, but iodine and other substances have sometimes a similar action. 5. *Critical* salivation is observed in some cases of fever, but salivation in febrile diseases is not always connected with a crisis. 6. *Infants* and *old people* are liable to an excessive flow of saliva. In the former this is often associated with *dentition*. In both



classes of subjects there is frequently no excess of secretion formed, but the saliva is allowed to escape from the mouth instead of being swallowed. 7. *Idiopathic* salivation is that form which occurs without any obvious cause.

TREATMENT.—1. In treating salivation the cause must be sought out, and if possible removed. In many cases this is all that is required. 2. *Astringent* mouth-washes are useful, such as solution of alum, tannic acid, oak-bark, weak mineral acids, or chlorate of potash; or alum may be sucked. 3. Opium and tincture of belladonna are the most useful internal remedies in obstinate cases of ptyalism.

## CHAPTER II.

### DISEASES OF THE THROAT.

#### A. CLINICAL CHARACTERS.

THROAT-AFFECTIONS are of very common occurrence, and may be indicated by more or less of the following clinical phenomena:—

1. The presence of soreness, pain, or other **morbid sensations**. These vary greatly, both in degree and kind; but among the most common are a sense of dryness, burning, tightness, or as if a foreign body were present, which induces a constant desire to hawk or swallow. There may also be external tenderness. 2. **Disorders of deglutition**. This act may be painful or difficult, or even quite impossible, and sometimes substances tend to pass in wrong directions, such as into the larynx or posterior nares. The physical conditions of the materials which are being swallowed,—such as whether they are solid or liquid, hot or cold,—often influence the degree of dysphagia. 3. **Alteration of the voice**, which is either somewhat hoarse or husky, or even completely altered in its quality, becoming thick and guttural or nasal. The act of speaking may cause pain. 4. **Hawking and cough**. These are very common and troublesome symptoms attending throat-affections, especially those of a chronic nature. Even in pulmonary affections, particularly phthisis, the state of the throat often aggravates cough considerably; and the same is true of the so-called *stomach-cough* observed in dyspeptic cases. Frequently the act is attended with the discharge of abundant mucus or other materials. 5. **Disturbance of breathing**. There is not as a rule any difficulty of breathing in mere throat-affections, but in certain cases considerable obstructive dyspnoea may be felt, especially on lying down; while patients often breathe with the mouth wide open, and snore loudly. 6. **An unpleasant or foul smell of the breath**. 7. **Deafness** occasionally, due to obstruction about the openings of the Eustachian tubes. 8. Signs revealed on **physical examination**. A careful examination of the whole of the fauces is requisite, whenever symptoms point to this part as being the seat of mischief. A good light is needed, and it is sometimes useful to employ artificial light, with the aid of the reflector of the laryngoscope. The tongue must be kept down by means of the handle of a spoon or a tongue-depressor, or with

the finger. In some cases it is also desirable to use the finger for the purpose of feeling the structures in the throat, especially when examining children. By the physical examination of the throat a knowledge is gained :—*a*, of the appearance of the general surface of the fauces, as regards colour, degree of moisture, smoothness or roughness, and other characters; *b*, of the presence of any deposit or accumulation of secretion; *c*, of the general form and dimensions of the pharynx and its openings; as well as of the size, shape, and other characters of the soft palate and its arches, the uvula, and the tonsils; *d*, of the existence of any enlarged follicles, abscesses, ulcers, eruptions, gangrene, old cicatrizations, polypi, or cancerous or other tumours. When the throat is affected, the external structures of the neck should likewise be examined, particularly those about the angles of the jaw, special attention being paid to the lymphatic glands. It will be well also to notice the state of the mouth and lips.

## B. SPECIAL DISEASES.

### I. ACUTE INFLAMMATORY AFFECTIONS OF THE THROAT.

The throat is the seat of acute inflammation of a special character in diphtheria and scarlatina; thrush may also extend from the mouth to this part; or it may be inflamed in connection with eruptions on its mucous surface, especially herpetic and variolous, or from the extension of erysipelas from the face. At present, however, attention will be confined to *local* inflammatory affections, which are of considerable importance.

**ÆTIOLOGY.—Predisposing causes.**—Throat-inflammations may occur at any age, but are most common on the whole in adults, probably because they are more exposed to the exciting causes. Tonsillitis is, however, chiefly met with among young persons. Former attacks seem to increase the liability to inflammatory throat-affections, many individuals suffering from the slightest cause, or being liable to periodic attacks, especially if the tonsils are enlarged. Anything that lowers the health is stated to be a predisposing cause of sore-throat, and certainly this is true with regard to living in a hospital for some time. Persons who are constitutionally weak appear to suffer with unusual frequency; and syphilitic individuals are decidedly more liable than others to this class of affections. The opinion seems to be gaining ground that many cases of sore-throat, and especially of acute tonsillitis, are manifestations of the rheumatic diathesis. Intemperance acts as a predisposing cause in some instances. Tonsillitis appears sometimes to run in families. Most cases occur during spring and autumn.

**Exciting causes.**—1. Inflammatory throat-affections generally result from *taking cold* in some way or other, especially from exposure to cold and wet, to sudden changes of temperature, or to damp cold winds. In many cases they seem to be but a part of a general catarrh from this cause. Some forms are believed to be associated with the rheumatic condition. 2. Occasionally the milder forms of sore-throat appear to be due to some *derangement of the alimentary canal*; follicular pharyngitis is often associated with stomach disorders. 3. *Local irritants* excite more or less inflammation, which may then be of a very serious

character, as from swallowing hot water or chemical irritants. 4. Certain forms of throat-inflammation may be due to some *atmospheric poison* acting on the system. Many cases of hospital sore-throat seem to arise in this way; and some writers regard tonsillitis as having this mode of origin. 5. Pharyngeal catarrh is often a *complication* of the exanthemata. 6. *Excessive use of the voice* is liable to cause sore-throat, especially the follicular variety, which constitutes a part of *clergyman's sore-throat*.

**SYMPTOMS.**—For clinical description, cases of acute inflammation affecting the different structures of the throat may be conveniently arranged under the following groups:—

**1. Acute Pharyngeal Catarrh—Catarrhal Pharyngitis—Relaxed Sore-throat—Cynanche Pharyngea—Angina Simplex.**—A large proportion of sore-throats may be included in this general group, being due to *catarrhal inflammation* of the fauces and pharynx, differing much in its extent and severity in different cases. Occasionally the inflammation is of a severe character, and extends more or less deeply. Uneasiness, soreness, or pain is experienced in the throat, in proportion to the degree of inflammation, often accompanied with a sense of heat and dryness. Swallowing is always attended with discomfort, and is often painful, though the patient may be constantly inclined to perform the act, being prompted thereto by a feeling as if there were something in the throat, especially when the uvula is involved. There is also a frequent tendency to cough and hawk, in order to remove the secretion formed, or to get rid of the feeling of obstruction. The voice is often thick or husky, and the act of speaking may cause pain; but there is no dyspnoea. The symptoms are generally worse during the night, and after sleep. Occasionally deafness is complained of, the Eustachian tubes being blocked up.

Examination reveals more or less general redness of the throat, which is usually bright, but may tend to lividity. The surface appears dry and glistening. Considerable cedema may be observed where the submucous tissue is loose, giving rise to swelling and a watery translucent appearance, particularly in connection with the uvula. Secretion often collects in patches and points over the back of the fauces and on the tonsils; these sometimes look remarkably like diphtheritic patches, but they are easily detached without causing bleeding or excoriation. Not uncommonly superficial erosion is produced, and the more intense forms of inflammation are liable to terminate in ulceration, or in more or less deep suppuration; the latter may constitute a *pharyngeal abscess*.

In slight cases of sore-throat there are no *general* symptoms. The more severe forms of pharyngeal inflammation may be ushered in with chilliness, headache, and pains in the limbs; and are attended with pyrexia. The pulse may rise to 100 or 120; and the temperature to 102° or even higher. A bright blush occasionally suffuses the face and upper part of the body, where there is no reason to suspect scarlatina. In some cases of apparently simple pharyngeal catarrh I have met with albuminuria, which entirely disappeared after a time.

Sore-throat may come on very rapidly, attaining considerable intensity in a few hours, and this particularly applies to a form of *hospital sore-throat*, which appears to be of an erysipelatous nature. This generally sets in during the night, and by the morning the symptoms are very prominent. There is much cedema and swelling, but no particular redness; while deglutition is very uncomfortable and difficult.



**2. Acute Follicular Pharyngitis.**—This variety is characterized by the follicles of the throat being chiefly implicated, which become enlarged, hard, and red. It is attended with a good deal of local soreness or pain, and abundant secretion forms, causing continuous hawking. Sometimes the follicles suppurate and ulcerate.

**3. Acute Tonsillitis — Amygdalitis — Cynanche Tonsillaris — Quinsy.**—This affection consists in a parenchymatous inflammation of one or both tonsils. Usually some general febrile disturbance is observed before throat-symptoms occur. These are soon manifested, however, and they may appear simultaneously with the fever. At first uneasiness is felt over one or both tonsils, which soon increases to considerable pain, of a dull, aching character, with much tenderness. The throat feels dry, and a most uncomfortable sensation is experienced, as if a foreign body were present. External tenderness is felt behind the angles of the jaw, which may be considerable. Deglutition is difficult and causes much distress, the pain during the act often shooting towards the ear, while in severe cases fluids tend to return through the nose. After a time a quantity of sticky mucus forms, entailing frequent efforts to swallow, or continuous hawking. The voice is characteristically altered, having a thick, muffled, guttural or nasal character, which cannot be mistaken when once heard; occasionally it is altogether lost. Breathing is not interfered with as a rule, but if both tonsils are greatly enlarged, dyspnoea may be felt, especially on lying down. The patient snores loudly during sleep, and the symptoms are always worse on waking. The breath is very unpleasant. Salivation may be present. Deafness and noises in the ears are often complained of.

Examination of the throat is not always easily carried out in tonsillitis, but if it can be effected, the appearances observed are:—general redness of the fauces, but more particularly of one or both tonsils; enlargement of these structures, sometimes so considerable that they meet in the middle line, and almost completely block up the passage, while they look like balls of flesh, and may actually ulcerate from mutual pressure; white or yellowish opaque spots or patches on the surface, being the products of follicular secretion. The palate and uvula are also swollen and cedematous, the latter being almost always observed to adhere to one of the tonsils. When the parts cannot be inspected, the finger must be made use of in order to feel the tonsils, and this is especially required in children, or in the later stages of the complaint, in order to ascertain whether suppuration is taking place. Very often the salivary glands are swollen, and also the structures about the neck, especially the lymphatic glands, which feel firm and tender; a sensation of stiffness and uneasiness being likewise experienced by the patient.

Tonsillitis is usually attended with considerable fever, and the patient feels decidedly ill, being often much prostrated. The temperature frequently rises to 102° or more, and may reach 104°; the pulse usually ranging from 100 to 120. There is often severe headache, with much restlessness, and occasionally slight delirium at night. The tongue is covered with a thick creamy fur; appetite is lost, but there is much thirst; and the bowels are constipated. A red rash on the skin is now and then observed. The urine is markedly febrile; while chlorides are deficient, or sometimes almost entirely absent.

Cases of tonsillitis vary greatly in their severity. Frequently only one tonsil is involved, but in many cases both are attacked, generally in succession, but occasionally simultaneously. The inflammation usually

attains its height in five or six days, and the entire duration of most cases of tonsillitis is under ten days. Pathologically the disease may terminate in :—*a. Resolution*, the symptoms gradually subsiding. *b. Suppuration*. This is very common, being indicated by the pain becoming more pulsating or throbbing, and shooting towards the ear; by the occurrence of rigors; and by the tonsil feeling soft and fluctuating, or the colour of the pus being actually evident through the redness. The abscess often bursts suddenly, either spontaneously or from some mechanical irritation, or it is opened; and in either case rapid improvement follows the escape of the pus, which is often very offensive. Only one tonsil suppurates as a rule. *c. Gangrene*, of very rare occurrence, and only met with in those who are low and debilitated. *d. Chronic enlargement*, with a granular or irregular appearance of the tonsil, especially after repeated attacks ending in suppuration, and in weak individuals. Clinically cases of tonsillitis almost invariably terminate in recovery. Death is an exceedingly rare event, but may result from hæmorrhage or from extension of inflammation to the larynx.

**DIAGNOSIS.**—The fact of the existence of an acute inflammation of the throat is usually readily recognized, and a proper examination will generally reveal its nature. Difficulty may, however, be experienced in determining whether the complaint is *local*, or merely a part of some *general* disease, especially diphtheria or scarlatina. A careful inquiry with regard to the ætiology of the case; the mode of invasion; the collateral symptoms; and the degree of pyrexia, ought generally to clear up any doubt. Sometimes it is requisite to wait for a short time before giving a definite opinion; and it must be remembered that sore-throat may be almost the only symptom of scarlatina.

From *laryngeal* inflammations, those involving the throat are distinguished by the absence or slight degree of dyspnoea as a rule, or its different character when present; by deglutition being more interfered with; by the voice being less altered, or affected in a different way; by cough being a much less marked symptom, and not having laryngeal characters; and, above all, by the results of examination. It must be remembered that the inflammation may spread to the larynx; or the parts may be involved simultaneously, each then giving rise to its own special symptoms.

**PROGNOSIS.**—Local throat-inflammations are rarely dangerous, but they may become so by occasioning considerable œdema of the tissues, or by spreading to the larynx, being thus liable to cause suffocation. Death from hæmorrhage has in rare instances occurred in connection with an abscess in the tonsil. It is often difficult, and may be impossible, to get rid of the liability to attacks of sore-throat.

**TREATMENT.**—In slight cases of sore-throat the application of a wet rag round the throat at night, covered with a piece of flannel, is all that is necessary. Even when the affection threatens to be severe, it may probably be checked not infrequently by the assiduous application of cold water outside the throat, and the frequent sucking of ice.

In many cases, however, of acute inflammation in connection with the throat, more active treatment is required, and there are certain general rules which should always be carried out. The patient should be kept quiet, in a comfortably warm room, and not allowed to talk. A *saline aperient* may be given at the outset, and the bowels should be kept well-opened throughout. If there is pyrexia, and if the patient is not very weak, it is useful to administer a *saline* mixture for two or three days.



It is not desirable to keep the patient low, therefore a good quantity of beef-tea, milk, and other liquid nutritious food should be given in moderate quantities, at frequent and regular intervals. Mucilaginous drinks afford relief; and the frequent sucking of ice is very grateful, as well as beneficial. Stimulants are often indicated, good port-wine being the best form, this being particularly useful during convalescence, and in cases of tonsillitis; in the latter complaint from four to eight ounces of wine may be given during the twenty-four hours.

It is affirmed that, by the employment of certain drugs, acute inflammatory affections of the throat can be very rapidly subdued, and their progress stopped. Dr. Ringer and others advocate the use of tincture of aconite in small doses frequently repeated. Guaiacum has also been supposed to exert a specific influence upon tonsillitis; and salicylate of soda has likewise been specially recommended. An emetic of ipecacuanha with tartar emetic at the outset has been considered efficacious in checking the course of this disease, but this is more than doubtful.

The remedies which I have found of most service in this class of complaints are quinine (gr. i-ij); tincture of iron (℥ xv-xxx); or dilute nitric acid with decoction of bark. Either of these may be given at intervals of three or four hours, or three times a day, according to the severity of the case, and they are often beneficial from the very commencement. Quinine and iron may in many cases be advantageously combined. A mixture containing dilute hydrochloric acid and chlorate of potash has been much commended in acute catarrh of the throat; or chlorate of potash may be given with tincture of iron. It is often desirable to order some *saline* drink along with the tonics, such as a solution of citrate of potash, so as to maintain a free action on the part of the skin and kidneys. In cases of tonsillitis it is not infrequently requisite to administer some narcotic at night, such as Dover's powder or hydrate of chloral, but sleep should not be too prolonged. The patient should rest with the head well-raised.

*Local treatment* is always most important. In the various forms of pharyngeal catarrh steam-inhalations, tepid milk-and-water gargles, with poultices and fomentations over the front of the neck, give most relief at first. Afterwards *astringent* gargles are useful, such as one containing alum, tannin, dilute mineral acids, or port-wine. Gargles of chlorate of potash are often serviceable. It may be desirable to apply nitrate of silver or its solution, especially in the *follicular* variety. If there is dangerous oedema, it is necessary to scarify the surface; and should suppuration occur, the pus must be let out by incision.

For *hospital sore-throat* the treatment which I have always found rapidly effectual is to persevere with steam-inhalations, frequent gargling, and the external application of heat and moisture; and to give large quantities of beef-tea, a glass of port-wine every three or four hours, and quinine in two or three grain doses every four hours.

For *acute tonsillitis* the best local treatment consists in the continuous application of heat and moisture to the throat, by means of steam-inhalations, and the use of gargles of lukewarm water or milk and water, to which a little Condyl's fluid may often be added with advantage, in order to diminish the unpleasant taste and odour of the breath. At the same time very hot and well-made linseed-meal poultices should be applied round the front of the neck, being changed at frequent intervals; or spongio-piline wrung out of hot water answers very well. When these applications are removed, the part should be well fomented. These



measures tend to subdue the inflammation, if resolution is going to take place; or they will hasten the process of suppuration. When indications appear that pus has formed, it is desirable to let this out, and thus give immediate relief; or it may be requisite to puncture the tonsils, even though there is no distinct evidence of the presence of pus, especially if the breathing should be greatly impeded. Some practitioners advocate the use of irritating applications to the neck externally, such as sinapisms, liniments, and blisters, but to me these appear decidedly objectionable. Possibly, should there be very severe local symptoms, accompanied with great swelling, it might be advisable to apply two or three leeches behind the angle of the jaw.

During convalescence after tonsillitis *tonics* are needed for some time, with good nourishing food and a little wine. A change of air often does much good. Locally *astringent* gargles or other forms of application are necessary, the most serviceable being glycerine of tannin, tincture of steel with glycerine, or nitrate of silver solution. Should the tonsils remain permanently much enlarged, it may be requisite to excise them.

As *prophylactic* measures in the case of those who are subject to sore-throat or quinsy, frequent cold douching of the throat externally, and the daily use of cold water or of some mild astringent as a gargle, should be recommended. The general health must be attended to, and all injurious habits checked. A change of air, and a course of tonic medicines frequently lead to good results.

## II. ULCERATION OF THE THROAT.

Ulcers are very common in connection with the various structures of the throat, being either *acute* or *chronic*. They may be enumerated as follows:—1. **Catarrhal**, which are slight and superficial, being very frequently observed, especially at the back of the pharynx, and often associated with chronic catarrh. 2. **Follicular**. These ulcers are generally small, and circular or oval, corresponding to the follicles, but by their union they may become irregular and of some size. 3. **Syphilitic**, either secondary or tertiary. 4. **Scarlatinal**. 5. **Diphtheritic**. 6. Ulcers following **eruptions**, such as herpes. 7. **Gangrenous** or **Sloughing sore-throat**—**Cynanche** or **Angina maligna**. This form of ulceration is generally associated with syphilis or scarlatina, but may be independent of these affections. Thus it may follow severe catarrhal inflammation, if the patient is in a very low state of health from any cause; and occasionally it occurs as a complication of typhus, enteric fever, or other exanthemata. It spreads more or less extensively, but not as a rule deeply; the mucous membrane is dusky; while there is much oedema around. 8. Ulcers on the **tonsils**, simulating syphilitic ulcers, but probably originating in blocking-up and subsequent inflammation of their follicles. 9. **Cancerous** ulceration, which is extremely rare.

**SYMPTOMS.**—Ulceration of the throat may be unattended with any symptoms, even when of considerable extent. Usually, however, *local* symptoms are present to a greater or less degree. There may be merely uneasiness or pain and difficulty in swallowing, but when certain parts are destroyed, most unpleasant and dangerous symptoms are liable to arise. Food, especially of a liquid kind, may tend to pass into the pos-

terior nares or down the larynx instead of into the cesophagus. The voice is often completely altered, being thick, guttural, and indistinct; or the patient may scarcely be able to articulate at all. Offensive matters are hawked or coughed up; and the breath is in many cases very foul, sometimes peculiarly so. It is important to notice that dyspncea is not uncommonly present, being attended with very noisy breathing; and that there may be a liability to sudden death from suffocation, in consequence of the ulceration involving the upper opening of the larynx. In some cases there is also a danger of hæmorrhage.

Ulceration of the throat is often attended with a low condition of the general health, and there may be much emaciation and debility, owing to inability to swallow food. In *gangrenous* ulceration there is a danger of septicæmic symptoms setting in. Of course when the ulceration is a part of some special disease, such as scarlatina, the general symptoms will be modified accordingly.

The ultimate local consequences of ulceration are also liable to be very unpleasant, or may even prove dangerous, in the way of permanent destruction of tissues, adhesions, and contractions after cicatrization. I have seen a case in which the throat was one large chasm, with thickened bands extending along its walls, every trace of its various parts having disappeared. Of course under these circumstances swallowing becomes very difficult, and the voice is permanently altered.

DIAGNOSIS.—It must be borne in mind that the throat may be ulcerated without any complaint of local symptoms being made by the patient. The smell of the breath has not in a few instances led me to the discovery of unsuspected ulceration in this part, and when this is foetid, the throat should always be carefully examined. In conducting the examination it is necessary to raise the uvula, in order to see the upper part of the back of the pharynx, as ulcers are not uncommon here, and may otherwise be overlooked. It is important to determine the nature of any ulceration of the throat, and especially whether it is of a syphilitic character.

PROGNOSIS.—Ulceration of the throat may prove immediately dangerous, in consequence of interfering with deglutition, and thus affecting nutrition; spreading to the larynx; giving rise to hæmorrhage; or inducing septicæmia. Some forms are difficult to cure. The destructive effects of ulceration may lead to serious permanent mischief.

TREATMENT.—1. **Local.** For most ulcerations of the throat nothing answers better than the frequent use of chlorate of potash as a gargle (3ij-iiij to Oj); or in the form of lozenges or spray. *Follicular* ulcers, as well as other chronic forms, often require to be freely touched with nitrate of silver or its solution. When the surface is sloughy, *antiseptic* gargles or sprays must be abundantly employed, such as those containing Condry's fluid, carbolic acid, creosote, or chlorine, and they may be used alternately with the chlorate of potash gargle. In *gangrenous* forms of ulceration exhibiting a tendency to spread it is advisable to start by brushing the surface over carefully with strong nitric or hydrochloric acid, then proceeding with the other applications. Inhalations containing carbolic acid, creosote, or other *antiseptics* are also very valuable.

2. **General.** It is very important before commencing treatment to determine the nature of any throat-ulceration, and especially whether it is due to syphilis. If such be the case, iodide of potassium with decoc-

tion of cinchona bark or quinine generally produces the best results. Sometimes a course of mercury is required, but it must be administered with care. It will often be found, even in syphilitic cases where there is much sloughing, that dilute nitric acid with decoction of bark brings about rapid improvement, and this mixture is very useful in other forms of gangrenous ulceration. Tincture of steel, in doses of  $\text{mxx}$ -xl every four or six hours, is also exceedingly valuable, especially if there is much debility, and it may be combined with quinine. The internal administration of chlorate of potash is regarded as almost a specific remedy in throat-ulcerations. It should be freely employed locally, but may be given at the same time as a drink or with tincture of iron. Dr. Sansom advocates the use of the sulpho-carbolates.

Not uncommonly one of the most important matters requiring attention in throat-ulcerations is the feeding of the patient. In many cases, owing to great difficulty or pain being experienced in swallowing, very little or no nourishment is taken, and hence the system becomes greatly lowered, so that healthy action cannot take place, and the ulceration will not heal. Under these circumstances the patient must be compelled to take small quantities of beef-tea and milk at frequent intervals, and in this way a considerable amount of nutriment may be administered. If this is persevered in for a short time, the patient generally becomes able to swallow easily, and there is a marked effect for good produced on the ulceration. At the same time a good quantity of port-wine should be given in similar small doses. If deglutition is really impossible, nutrient enemata must be employed.

When there is much dyspnœa accompanying throat-ulceration, the patient must be carefully watched, as remarkably sudden death may occur from suffocation, and laryngotomy or tracheotomy may be called for at a moment's notice. Indeed, in cases attended with great danger, it is decidedly advisable to open the larynx as a precautionary measure, so that there may be no fear of sudden death, while at the same time the ulcerated structures are left in a state of rest, and therefore in a more favourable condition for undergoing the healing process.

### III. CHRONIC AFFECTIONS OF THE THROAT.

1. **Chronic Pharyngeal Catarrh.**—This is a very common condition, the symptoms being uneasiness or soreness of the throat, increased by irritating substances; roughness or huskiness of the voice; a frequent desire to clear the throat; hawking and cough, especially in the mornings, there being much difficulty in clearing away the secretion which forms. Examination reveals redness, frequently with permanent enlargement of the vessels; a rough and granular appearance—**granular sore-throat**, or numerous enlarged follicles—**follicular sore-throat**, or raised hard papules of considerable size; generally much thick sticky secretion; and superficial erosions or ulcerations, or follicular ulcers. Chronic pharyngeal catarrh is often associated with disorders of the stomach; phthisis; chronic alcoholism; excessive smoking; too much talking or singing; or a relaxed uvula.

2. **Relaxed Mucous Membrane** is frequently the cause of unpleasant throat-symptoms, and it either follows pharyngeal catarrh, or is associated



with general debility. A quantity of secretion forms on the surface of the fauces, which excites cough. A *relaxed and elongated uvula* gives rise to very uncomfortable sensations, with a tickling cough, which comes on particularly when the patient lies down at night, owing to the uvula falling back and causing irritation at the top of the larynx. It may excite nausea and vomiting.

**3. Chronic Enlargement of the Tonsils.**—Enlargement of the tonsils beginning during very early life is liable to lead to more or less serious results, and, therefore, when examining children it is always desirable to ascertain the condition of their tonsils, especially if they are rickety, tubercular, or strumous. Its pathological causes are:—1. *Chronic inflammation*, either following acute tonsillitis, especially after several attacks; or coming on gradually. 2. Persistent or frequently-repeated *congestion*. 3. Repeated *ulceration*. 4. *Albuminoid disease*.

The chief symptoms of enlarged tonsils are difficulty of deglutition; and alteration of the voice. Breathing is not uncommonly interfered with, and the report frequently given is “that the child makes a great noise when asleep,” snoring loudly. In course of time deformity of the chest may result in very young subjects, from the obstruction to the entrance of air into the lungs. Deafness is not an unusual symptom. On inspection the tonsils are seen to be more or less enlarged, and they may meet in the middle line. There is no particular redness, but the surface appears granular or irregular, and white opaque accumulations of secretion are often observed upon it. The tonsils feel unusually firm and hard. The general health is in most cases below par, and nutrition may be interfered with, so that growth and development do not proceed properly. Chronically enlarged tonsils are not uncommonly liable to acute exacerbations from time to time.

**4. Polypi** and various other **morbid growths**, benign or malignant, are in rare instances met with in the throat. They cause more or less unpleasant local sensations, with obstruction to deglutition or breathing, and abundant secretion may be hawked or coughed up. Sometimes hæmorrhage takes place. The nature of any growth is revealed on examination; and it may sometimes be felt when it cannot be seen.

**DIAGNOSIS.**—All that need be said here is that chronic affections of the throat may be simulated in nervous people when there is nothing really wrong; and that certain symptoms which are attributed to other diseases are not uncommonly due to some abnormal condition of this part, which has been overlooked. This remark applies particularly to cough. The habit of always examining the throat when such symptoms are present will guard against this error.

**TREATMENT.**—In treating any chronic throat-affection, it is requisite first of all to find out its cause, and to remove this if possible. Intemperance in drink; excessive smoking; the habitual use of hot spices and condiments in excess; or too much speaking in public or singing, must be put a stop to. At the same time it is often important to improve the general health, by careful attention to hygienic conditions and diet, especially in the case of children suffering from enlarged tonsils, for whom a change to the seaside often proves highly beneficial. If the alimentary canal is out of order, it must be attended to. The internal administration of quinine and iron, acids with bitter infusions, or nuxvomica or strychnia, frequently does much good. Steel-wine, or syrup of phosphate of iron, and cod-liver oil are very useful for children whose tonsils are chronically enlarged.

*Local treatment* is often essential. The regular and efficient employment of *astringent* applications is generally called for, in the form of gargles, glycerines or solutions applied with a brush, spray, lozenges, or powders. The most serviceable topical remedies are alum, tannin, dilute mineral acids, tincture of capsicum, catechu, tincture of steel, sulphate of zinc, or nitrate of silver. Infusion of roses with dilute sulphuric acid and tincture of capsicum, or alum with honey, constitute agreeable and efficient gargles. Glycerine of tannin is a very useful application in many chronic conditions of the throat. It may be necessary to puncture follicles, and then touch them with nitrate of silver; or to snip off an elongated uvula. When the tonsils are enlarged, the regular application of strong glycerine of tannin, nitrate of silver, or tincture of iodine may be tried, but generally these are quite ineffectual, and the tonsils have to be excised. After excision, it is necessary to see that the tonsils heal properly, as they are apt to remain in a painful state for some time. If the chest is becoming deformed, there should be no delay in removing the tonsils. Morbid growths may also need excision. Galvano-puncture, the wire being at a white heat, has been advocated instead of excision in cases of chronic enlargement of the tonsil.

#### IV. RETRO-PHARYNGEAL ABSCESS.

*ÆTIOLOGY.*—This is a very rare affection, which may be either *acute* or *chronic*, and occurs under the following circumstances:—1. As a *complication* or *sequela* of the acute specific fevers. 2. In *pyæmia*. 3. As the result of *local injury* or *disease*, for example, caries of the cervical vertebræ, or disease of the laryngeal cartilages. 4. Extremely rarely as the termination of a *primary local inflammation*.

*SYMPTOMS.*—The symptoms of retro-pharyngeal abscess are pain at the back of the pharynx; much difficulty in swallowing food, while drink returns by the nose; alteration in the voice; cough; and often great dyspnoea, with a sense of suffocation. The abscess may be seen or felt in the pharynx, or it may form an external enlargement; and after a time fluctuation can be detected in most instances.

*TREATMENT.*—This consists in at once letting out the pus by careful incision; and keeping up the strength of the patient by nutritious diet, stimulants, and tonics.

### CHAPTER III.

#### DISEASES OF THE ŒSOPHAGUS.

##### A. CLINICAL CHARACTERS.

THE clinical phenomena which are to be looked for as indicative of œsophageal affections include:—

1. **Morbid sensations**, namely pain, which generally seems to lie deep in the chest, as if between the shoulders, and fixed in some particular spot; as well as other abnormal sensations, such as fulness, tightness, oppression, burning, or a feeling of obstruction by a foreign body.

2. **Pain or difficulty during deglutition.**—When this symptom is noticed, inquiry must be made with regard to its degree; whether the difficulty can be overcome by repeated efforts; if it can be localized in any particular spot; whether it has come on gradually and steadily increased, or suddenly; if it is constant or only paroxysmal, associated or not with the taking of food; if it is influenced by the liquid or solid character of things swallowed, the size of solids, hot or cold substances, or special articles of diet; and whether the act is aided by any particular position.

3. **Rejection** of various substances, such as food, mucus, blood, exudation, pus, &c., either by mere regurgitation, by spasmodic action, or by vomiting. This may occur immediately after taking food; or only after an interval, a large quantity being then discharged, as if the materials had been accumulating for some time. Food which has remained in the œsophagus instead of entering the stomach has an alkaline reaction, and is macerated and decomposed, instead of having undergone the digestive process.

4. Signs revealed on **physical examination.** Physical examination often aids materially in the investigation of œsophageal affections. This includes:—*a.* A full *inspection of the throat.* *b.* The passage of an *œsophageal bougie*, which must be done cautiously, the instrument having been previously softened and then lubricated with oil, glycerine, or white of egg. The tube of the stomach-pump answers very well for this purpose. Before either of these instruments is used, it must be ascertained that no aneurism exists in the chest. By their employment important information may be obtained. (i.) The practitioner can thus ascertain by mediate palpation if there is any actual obstruction, as well as its seat and degree; whether such obstruction is constant and increasing, or only present from time to time; and if it can be overcome by continuous moderate pressure, yielding more or less suddenly, or when the patient is under chloroform. (ii.) Should there be obstruction, the actual shape of the obstructed part may be determined by making the bougie warm and soft, and thus getting a mould of the passage. (iii.) Frequently the bougie brings up materials on its surface, such as blood, pus, or cancer-cells, which should then be examined microscopically. (iv.) Occasionally the bougie may be felt to pass round something, such as a polypus; or into a diverticulum. (v.) The patient's feelings must not be forgotten. Pain always felt at one particular spot when the bougie is passed may be indicative of the precise seat of a disease. *c. Auscultation.* This is sometimes useful, the stethoscope being applied in the course of the œsophagus behind, a little to the left of the spine in the cervical or dorsal region. In health, when a person swallows water, a short, clear, gurgling sound is heard; if there is obstruction, the sound is prolonged, and altered in character below the seat of the obstruction. *d. External examination:*—(i.) For any swelling in the neck or elsewhere in the course of the gullet, which might indicate dilatation or sacculation of this tube; at the same time noticing whether any such enlargement is influenced by taking food or drink, or by the act of vomiting or retching. (ii.) For any tumour in the neck or chest likely to affect the functions of the œsophagus, by exerting pressure upon this tube or upon its nerves.



## B. SPECIAL DISEASES.

The individual diseases of the œsophagus may be conveniently described under the two main divisions:—I. ACUTE CÆSOPHAGITIS. II. CHRONIC DISEASES.

## I. ACUTE CÆSOPHAGITIS.

ÆTIOLOGY.—Cœsophagitis may arise:—1. As a simple *catarrhal* inflammation, along with catarrh of other mucous membranes. 2. From *direct injury* by foreign bodies. 3. From *irritation* or *corrosion* by chemical substances, such as acids, alkalies, or corrosive sublimate; also by very hot or cold articles. 4. By *extension* of thrush or diphtheria, when the characteristic deposits are met with. 5. As a *complication* of the specific fevers, cholera, pyæmia, and other acute affections. 6. In connection with local *organic diseases*, such as ulceration or stricture.

ANATOMICAL CHARACTERS.—Acute cœsophagitis presents the usual signs of inflammation, namely, redness and swelling of the mucous membrane; diminution in consistence; and the presence of various secretions or deposits on the surface, differing according to the nature of the inflammation. Occasionally ulceration is seen, and superficial erosions are common; while, if the inflammation is caused by corrosives, there may be much destruction of tissue. As a rare event pus forms beneath the mucous membrane of the œsophagus.

SYMPTOMS.—More or less pain is felt, which lies deep in the chest, along the course of the œsophagus, and which may extend to the epigastrium. If ulceration occurs the pain is very severe at the corresponding spot. Deglutition is difficult and painful, and the food and drink may be ejected, either immediately by spasm, or subsequently by vomiting, along with much mucus, or sometimes with blood, pus, or membranous shreds or casts. In severe cases there may be great distress, with an urgent sense of oppression about the chest. The *general* symptoms are of a febrile character, and there is much thirst. Should the œsophagus be corroded or ulcerated, perforation may possibly take place, indicated by its special symptoms.

TREATMENT.—In mild cases no special treatment is required. In severe forms of cœsophagitis all that can be done is to let the patient suck ice freely; to give only liquid and mucilaginous diet in small quantities, or, if there is corrosion, to employ nutrient enemata, so that the œsophagus may be left entirely at rest; to apply hot fomentations externally; and to administer opium in order to relieve pain and procure rest.

## II. CHRONIC DISEASES OF THE CÆSOPHAGUS.

These are of much importance, and may be described under:—(1.) FUNCTIONAL DERANGEMENTS. (2.) ORGANIC DISEASES.

## (1.) FUNCTIONAL DERANGEMENTS.

1. **Paralysis.**—This extremely rare condition of the Œsophagus is met with only in certain nervous affections, namely, in general paralysis of the insane; progressive muscular atrophy; some cases of brain-disease; the paralysis which follows diphtheria; hysteria; or as a part of glosso-pharyngeal paralysis.

**SYMPTOMS.**—Dysphagia is the only symptom of Œsophageal paralysis, which is particularly felt with regard to liquids, these tending to pass into the larynx. Solids, especially large pieces, are more easily swallowed; and the erect posture facilitates the act of deglutition. The bougie can be passed quite readily.

2. **Œsophagismus or Spasm.**—A slight degree of Œsophageal spasm is not at all infrequent; but sometimes this condition gives rise to much distress, inducing complete obstruction.

**ÆTIOLOGY.**—The causes of Œsophagismus are:—1. Most commonly some nervous condition, especially hysteria and hypochondriasis. 2. Brain-disease very rarely. 3. Local irritation, especially that of an ulcer. 4. Occasionally dyspepsia with flatulence. 5. Irritation of the nerves supplying the Œsophagus. 6. Abuse of alcohol. 7. Swallowing unmasticated lumps of food, or very hot or cold things.

**SYMPTOMS.**—There may be a constant feeling of obstruction in some fixed point, as if a foreign body were impacted in the gullet, but pain is absent. When food is taken, sudden dysphagia comes on, and a stoppage is felt at a certain spot, either absolute and complete, or yielding after several efforts have been made to swallow, this event often occurring equally suddenly. The attacks are not constant in all cases, for at times deglutition may be effected comfortably. The act is often influenced by the nature and temperature of the food. Usually much discomfort is felt during the attempts at swallowing, with a sense of oppression or suffocation, and sometimes spasmodic movements of the muscles of the neck are excited. On attempting to pass a bougie its progress is stopped, but after careful continued pressure the spasm gives way, sometimes suddenly, and the instrument passes on. In the cases of Œsophagismus which have fallen under my notice the upper part of the canal was usually affected, but the lower end may be involved. Dyspeptic symptoms are frequently complained of, especially flatulence and eructations, which may bring on the spasm. There are no signs of wasting or of serious organic disease as a rule, but generally the patient is distinctly hysterical or hypochondriacal. Should Œsophagismus be due to a tumour pressing on the nerves of the Œsophagus, this will probably be revealed by physical signs.

## (2.) ORGANIC DISEASES.

1. **Chronic Ulceration.**—An ulcer is occasionally seen in the Œsophagus, presenting characters similar to those observed in gastric ulcer, which will be hereafter described. It is liable to give way and to cause perforation.

**SYMPTOMS.**—Usually considerable localized pain is experienced, which is of a burning character. Deglutition is very painful and difficult, or

may even be impracticable, chiefly on account of spasm, and this is sometimes the only symptom observed. Blood and mucus may be expelled, or brought away on the bougie. Great care must be exercised in using this instrument if there is any reason to suspect ulceration.

**2. Stricture and Obstruction.**—The conditions which may cause narrowing or complete closure of the canal of the œsophagus, including the presence of foreign bodies, may be thus enumerated:—

(i.) *Organic changes in its walls*, namely:—*a.* Cancerous infiltration, *b.* Cicatricial thickening and contraction after wounds, ulcers, or corrosion. *c.* Hypertrophy of its coats, especially of the submucous cellular tissue; or exudation and thickening, as the result of chronic inflammation. *d.* Syphilitic disease. *e.* Ulceration, with induration and swelling of the margins of the ulcer.

(ii.) *External pressure* from:—*a.* An enlarged thyroid gland. *b.* Enlarged lymphatic glands in the neck or chest. *c.* Various tumours or growths in the neighbourhood, such as cancer, fibrous tumours, exostoses; as well as aneurisms and abscesses. *d.* Great distension of the pericardium by fluid.

(iii.) *Growths projecting inwards from the walls of the œsophagus, or from without*, these being generally either cancerous, fibrous, or of the nature of polypi.

**ANATOMICAL CHARACTERS.**—The morbid appearances will necessarily vary according to the cause of the obstruction. After a time the gullet becomes dilated and hypertrophied above the seat of any obstruction, and in this distended portion food is apt to accumulate, causing irritation, ulceration, and sometimes perforation. The mucous membrane is also liable to ulcerate at the point of stricture. Below this point the tube usually becomes narrowed and atrophied.

**SYMPTOMS.**—Dysphagia is the chief symptom of organic obstruction of the œsophagus, and a feeling is experienced as if the food always stops at a certain spot, which in the great majority of cases lies behind the upper piece of the sternum. At first the difficulty is only slight, but it increases, usually gradually and slowly, until at last nothing whatever will pass. Liquids and soft pulpy substances are far more easily swallowed than solids, especially when these are in large pieces; hence patients suffering from œsophageal obstruction learn to chew their food thoroughly. If a good-sized lump can be got through the narrowed part, what follows will then pass more readily for a time. Food, if it cannot get through the obstruction, is either immediately regurgitated or spasmodically rejected; or it is retained in a dilatation for some time, and then discharged in large quantities, being almost always alkaline in reaction, as well as sodden and decomposed. A good deal of mucus is brought up, and sometimes blood or pus, should there be ulceration. Pain is frequently absent or very slight, but if cancer or an ulcer is present, it is commonly severe. A sense of pressure and weight in the chest is often complained of. If the obstruction is seated near the lower end of the œsophagus, the symptoms may appear to be associated with the stomach.

In proportion to the difficulty experienced in taking nourishment, there will be more or less emaciation and weakness, these symptoms being in many cases extreme, the abdomen becoming greatly retracted, and the patient exhibiting all the signs of starvation, at the same time being often very hungry and thirsty.

The use of the bougie gives accurate information as to the actual



existence, position, degree, shape, and rate of progress of any stricture or obstruction. It may also reveal its cause, by the peculiar sensation it yields, or by the substances which it brings up. The sensation afforded in syphilitic or cicatricial stricture is very firm and resistant. Auscultation may afford some aid in diagnosis. A tumour usually gives rise to symptoms of pressure on surrounding structures; as well as to external physical signs.

**3. Cancer or Malignant Disease.**—It is requisite to make a few additional remarks with regard to this extremely rare disease. Males and persons of advanced years are most frequently the subjects of œsophageal cancer, but I have met with it in females.

**ANATOMICAL CHARACTERS.**—The upper end of the œsophagus is involved in most cases, the lower portion much less frequently, and only very exceptionally the middle part. All forms of cancer are met with, the *epithelial* variety being most frequent above, and *scirrhous* near the cardiac orifice; the growth may infiltrate the coats and extend throughout the entire circumference of the tube, or it may form a distinct tumour. The affected part is thickened, constricted, hard, and irregular, and ulceration of the mucous surface is liable to occur. The sub-mucous tissue is that in which the formation is first observed. The glands in the neighbourhood are generally cancerous, but other organs are not often implicated.

**SYMPTOMS.**—In addition to symptoms pointing to œsophageal obstruction, there is much pain, generally localized, but also shooting along the sides, or upwards, or backwards between the scapulæ. The bougie may bring up cancer-cells, or these elements may be discovered in the materials rejected. Signs of pressure are usually evident, especially dyspnœa. Dyspeptic symptoms, with pyrosis and eructations, are common, and may be present for some time before any local signs of the disease appear. Great wasting and debility are generally observed, with œdema of the legs; and the cancerous cachexia may be well-marked. Œsophageal cancer almost always runs a rapid course, the average duration being about thirteen months.

**4. Dilatation or Pouching.**—The œsophagus may be dilated in its whole circumference; or a hernia or sacculation may form on one side. This condition arises from:—(i.) Some obstruction most commonly. (ii.) Paralysis from chronic catarrh. (iii.) No evident cause. An œsophageal pouch may form a tumour in the neck, influenced as to its size and other characters by taking food or drink, or by the act of vomiting. Substances collect in the dilatation, and are afterwards discharged at variable intervals.

**5. Perforation.**—The œsophagus may be perforated from within as the result of ulceration, cancer, corrosive destruction, or injury by a foreign body; or from without, by an aortic aneurism, an abscess, or a glandular or other tumour. The perforation may take place into various parts, and the symptoms will vary accordingly. It is generally accompanied with signs of collapse.

### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT OF CHRONIC ŒSOPHAGEAL DISEASES.

**DIAGNOSIS.**—When a case occurs in which symptoms point to the œsophagus, the diagnosis has to determine:—1. Whether this tube is itself organically or functionally affected; or is interfered with by some neighbouring tumour or other morbid condition. 2. The nature and origin of the disease—if organic, whether cancerous or other; if functional, whether of the nature of paralysis or spasm. 3. The seat of the morbid condition, as regards the part of the tube involved.

It is often very difficult to determine the actual conditions present, and in order to arrive at a correct diagnosis the following points must be carefully taken into account:—1. The general and family history of the patient; the conditions as regards age and other circumstances; as well as the history of the complaint with respect to its cause, previous duration, and course. 2. The general state of the patient, especially as to the degree of emaciation and debility, and the presence of any diathesis. 3. The degree, characters, and situation of the local symptoms, particular attention being paid to deglutition, from which important information may be gained. 4. The knowledge conveyed by the use of the bougie, which is often very accurate and decisive; as well as by the other modes of physical examination employed, especially with the view of determining whether any tumour exists in the vicinity of the œsophagus. 5. Whether any signs of adjacent pressure are present. 6. The state of the nervous system. 7. The condition of the stomach. 8. The progress of the case. The descriptions already given will sufficiently indicate how the several diseases of the œsophagus differ in these particulars.

**PROGNOSIS.**—Spasmodic affections of the œsophagus are not dangerous as a rule, but they are often exceedingly difficult to cure. Paralysis is very serious in most cases, being a part of some grave nervous disease. In all organic diseases of the gullet prognosis is highly unfavourable. In ulceration the chief dangers to be feared are from perforation, or from stricture after healing. Every form of obstruction involves an unfavourable prognosis; and when cancer exists, a speedy termination may be foretold. In some of the conditions which cause obstruction the prognosis depends partly on the effects of treatment.

**TREATMENT.**—1. The **general condition** of the patient must be attended to. If a hysterical state is evident, assafoetida, aloetics, iron, and valerianate of zinc are indicated. In cases of serious nervous disease, strychnine and electricity are of most service, but generally little or no good can be done. Attention to *diet* is most essential if there is any actual obstruction; liquid or pulpy nourishing food must be administered in sufficient quantities, and the patient may need to be fed at intervals by means of the stomach-pump. At last nutrient enemata have generally to be employed, and should there be ulceration, they are decidedly indicated at an early period, so that the ulcer may not be irritated, but may be allowed to heal. Any special diathesis, such as syphilis, must have its appropriate remedies. Dyspeptic symptoms frequently require attention. The strength must be kept up by *tonics*, cod-liver oil, and *stimulants*, as well as by food.

2. External **local applications** sometimes do good in œsophagismus, such as a blister over the sternum, a belladonna plaster, or friction with belladonna liniment. Probably these act beneficially chiefly by exerting a mental influence upon the patient. No local treatment has any effect whatever upon other conditions of the œsophagus.

3. The **bougie** is most valuable in treatment as well as in diagnosis. The threat of its use sometimes does good in spasmodic cases; and its regular employment often leads to much improvement in such cases. By its aid œsophageal strictures can often be dilated, but great care must be exercised in passing the instrument, especially if there should be ulceration.

4. Various **symptoms**, such as pain, sleeplessness, or vomiting, frequently call for special treatment, for instance, the administration of opium, morphia, or other *narcotics*; the application of local heat or of the ice-bag; and other appropriate remedial measures.

5. In some instances the advisability of performing **œsophagotomy** or **gastrotomy** has to be considered, and unquestionably under certain circumstances one or other of these operations ought to be performed, with the view of prolonging life, and relieving distressing symptoms.

---

#### CHAPTER IV.

### DISEASES OF THE RESPIRATORY ORGANS.

#### I. CLINICAL CHARACTERS.

THE clinical phenomena associated with the respiratory organs vary considerably in their precise characters, according to the part affected, and the nature of the disease; but the following outline will serve to indicate their general features.

1. **Morbid sensations** are very commonly complained of. When the larynx or trachea is involved, these sensations are referred to this portion of the respiratory tract, and they include chiefly mere uneasiness, soreness, or actual pain of variable characters; a sense of burning; a feeling of irritation; or a sensation as if a foreign body were present. They are often increased by acts causing local disturbance, such as coughing, speaking, or singing. There may be external tenderness over the larynx, especially on making pressure directly backwards. In affections of the bronchi, lungs, or pleuræ, pain or some other morbid sensation is generally experienced over some part of the chest, upon which it is always important to observe the effects of cough and of deep inspiration.

2. The act of **breathing** is frequently disturbed in some way or other, there being some form of **dyspnœa**. Any obstruction in the main air-tube causes serious interference with respiration, and hence in laryngeal and tracheal diseases breathing is frequently noisy, hissing, whistling, or stridulous; at the same time the act being prolonged and laboured; while there may be signs of urgent dyspnœa, with indications that little or no air enters the lungs during inspiration, especially



in the case of young children. In certain conditions expiration is undisturbed. Laryngeal dyspnoea may be constant or only paroxysmal, sometimes coming on very suddenly, and being always liable to exacerbations. As regards other portions of the respiratory apparatus, the characters of the disorder of breathing will vary with the nature of the disease.

3. Certain **expulsive actions** are often excited, which have for their object the removal of some source of irritation in connection with the respiratory apparatus. The most important of these is **cough**, but **sneezing** and **hawking** also come under this head, the former being excited by some irritation in the nasal passages, the latter by a similar condition in the larynx or throat. Cough differs in its characters according to its cause. Laryngeal cough is peculiar and characteristic, being irritable and liable to come on in distressing fits; difficult to repress; hard, hoarse, cracked, croupous, metallic, or barking in quality; or occasionally aphonic. In many cases of laryngeal disease there is a constant desire to cough or hawk, in consequence of a feeling of persistent irritation or obstruction being experienced.

4. The expulsive acts just alluded to are frequently attended with the discharge of various substances, technically termed **expectoration** or **sputa**. These may consist of mucus; muco-purulent matter; actual pus; croupous or diphtheritic exudation; portions of morbid structures, or of the tissues of the air-passages or lungs; calcareous particles; or various other materials.

5. **Hæmoptysis** or **spitting of blood** demands notice as a special form of expectoration, this being a symptom of considerable importance.

6. The **voice** is liable to more or less alteration in its characters when the larynx is implicated. It may be weak to complete aphonia; altered in quality, being rough, harsh, hoarse, croupy, or cracked; or changed in pitch or range.

7. Occasionally the **expired air** presents abnormal characters.

8. In diseases affecting the larynx, **deglutition** may be somewhat uneasy or painful, and if the epiglottis is destroyed, the act of swallowing becomes difficult, objects being apt to pass into the air-passage. In exceptional cases of certain lung-affections the patient may also experience some difficulty in deglutition.

9. The **general aspect** and **posture** of the patient often afford important indications in connection with diseases of the respiratory organs, and therefore should always receive particular attention.

## II. PHYSICAL EXAMINATION.

**Physical examination** constitutes an essential and most important part of clinical investigation directed to the respiratory organs, and this subject needs therefore to be discussed as fully and completely as the limits of this work permit, though it will be impossible to enter into lengthy details.

### A. EXAMINATION OF THE LARYNX AND TRACHEA.

Physical examination directed to the investigation of morbid conditions affecting the larynx or trachea includes:—

1. **External examination of the neck.**—This may reveal, for example, the presence of any tumour interfering with the main air-tube, or a fistulous communication with its interior. Direct examination over the larynx and trachea, by means of *palpation* and *auscultation* more especially, may also be useful, the latter method detecting alterations in the breath-sounds, or revealing local rhonchi. It is said that the *vocal fremitus* on the two sides of the larynx may differ in intensity, if one vocal cord is paralyzed.

2. **Examination of the throat internally.**—Much information is often gained by inspecting this part with a good light, as in many cases the throat and larynx are affected with similar morbid conditions. The state of the epiglottis can also be frequently made out in this manner. The finger may likewise be employed sometimes with advantage, to feel the epiglottis, œdema of the glottis, a foreign body, or a growth.

3. **Examination of the chest.**—This will show whether there is any obstruction to the entrance of air into the lungs; or if there is any tumour in the thorax, disturbing the functions of the air-tubes.

4. **Examination with the laryngoscope.**—The laryngoscope is the special instrument employed for determining the state of the upper part of the windpipe, and although it is possible in many instances to come to a tolerably accurate conclusion on this matter without its aid, yet the exact conditions present can only positively be ascertained by the use of this apparatus. It is also valuable for the purpose of applying local remedies, and in the performance of operations. Much practice is required before the laryngoscope can be effectually employed.

The *laryngoscope* consists of an apparatus for illuminating the back of the throat; and a small mirror, which is introduced into this region in such a way as to reflect an image of the interior of the larynx. Illumination is usually accomplished by *reflection*, a mirror being attached to the observer's head in various ways, which is made to reflect either solar light, or, more commonly, light derived from some artificial source. Numerous lamps have been invented, but any lamp which yields a clear, strong, and steady light will suffice for ordinary purposes. Some laryngoscopists employ *direct illumination*, by means of a lamp placed on a narrow table between the operator and the patient, having a powerful lens directed towards the latter, and being screened towards the former. The oxy-hydrogen light answers best for this method of illumination. The *throat-mirrors* ordinarily used are circular, varying in diameter from half-an-inch to an inch; but if the tonsils are enlarged, oval or ovoid mirrors may be employed. They should be made of glass backed with a coating of silver; mounted in German silver; and fixed at an angle of about  $120^{\circ}$  to a slender shank, which is inserted into a handle.

*Mode of examination.* The patient sits opposite the observer, with the head inclined very slightly backwards, an interval of about a foot being left between their faces. The lamp is placed at the side of the patient, the flame being on a level with the eyes. The mouth being opened as widely as possible, and the tongue protruded, this is held gently between the thumb and forefinger of the left hand, covered with a handkerchief or soft napkin. By means of the reflector the light is then directed to the back of the throat, so that the centre of the disc corresponds to the base of the uvula. The throat-mirror having been properly warmed over the lamp, is now introduced, the handle being held between the thumb and fingers of the right hand; its reflecting surface is directed

more or less obliquely downwards, while the opposite surface touches the base of the uvula, which should be pushed gently upwards and backwards. In order to facilitate the introduction of the mirror, it is often necessary to make the patient take a deep breath, or repeat the sound "ah." Some persons can bear examination for any length of time, if it is properly conducted, but in most cases it is better to introduce the mirror several times in succession, for a few seconds each time.

It is requisite to become familiar with the appearances observed with the laryngoscope in the healthy larynx, before this instrument can be of any service in investigating diseased conditions. The morbid conditions which may be detected are:—1. Changes in colour, due to congestion, inflammation, or other causes. 2. Alterations in the size, shape, and position of the epiglottis. 3. Thickening of tissues, with irregularity, resulting from chronic inflammation. 4. Oedematous swelling. 5. Various deposits, especially croupous or diphtheritic. 6. Ulcerations. 7. Growths and tumours. 8. Changes in the shape and size of different parts, particularly of the opening of the glottis. 9. Derangement of the action of the muscles of the glottis, as observed during the act of breathing or vocalization. The laryngoscope may also be used for the purpose of revealing the presence of a foreign body in the air-tube.

#### B. EXAMINATION OF THE CHEST.

Physical examination of the chest includes that of its walls; and of its several contents. The present section will be mainly devoted to a consideration of the subject so far as it applies to the respiratory apparatus; other structures must, however, be alluded to more or less, but the examination of the heart and vessels will be treated of separately.

There are some general points of practical importance to which it is desirable to call attention at the outset. 1. A knowledge of the anatomy and physiology of the thorax and its contents is essential before physical examination can be applied to the investigation of their morbid conditions. It is also requisite to be thoroughly familiar with the normal *physical signs*, and hence students should first practise the examination of healthy individuals. 2. When investigating for disease the chest should be, as a rule, exposed to the full extent that any individual case may require. It is often necessary to strip the chest and upper part of the abdomen completely, and in the case of males and children there need be no hesitation about doing this; the examination of females must of course be conducted with due regard to decency. To make a practice of examining the thorax through garments is most objectionable. This remark, however, only applies to those cases in which there is no danger in exposing the chest. In some instances this cannot be done at all, or only a very small portion should be uncovered at a time. 3. The patient should assume an appropriate position, the objects aimed at being to place the superficial structures under such conditions that they will not interfere with the production or perception of the physical signs; and to enable the operator to conduct the examination in a comfortable and unrestrained attitude. Ordinarily, when the front of the chest is being examined, the patient should sit or stand with the hands hanging down by the sides. A slightly stooping posture, with the head bent forward and the arms well folded across the chest, so that



the scapulæ are drawn away from the spine, and the muscles are put on the stretch, answers best for the examination of the back. The sides are most conveniently reached by having the arms raised vertically above the head. Of course the position has in many cases to be modified on account of the patient being confined to bed, or being even unable to be moved. 4. The investigation ought to be carried out thoroughly and completely, whenever circumstances seem to require this, every part of the chest being explored. Certain regions, however, demand special attention, namely, those corresponding to the apices of the lungs, both in front and behind; to their bases posteriorly and laterally; and to the heart and great vessels. 5. It is most desirable that the examination should be conducted in a systematic and orderly manner. The different methods should be gone through in regular succession, the lungs being first attended to, and then the heart and vessels. In many cases it is advisable to complete the examination of a particular region before proceeding to another part of the chest. It must be remembered that the various structures within the thorax mutually affect the physical signs which they severally present; and that examination of one organ may aid in detecting some morbid condition in another. 6. Repeated examination may be needed before a satisfactory diagnosis can be arrived at. Moreover, in many acute cases the physical signs should be noted at frequent intervals, in order to observe their progress; while in the course of affections which are known to have a tendency to implicate the thoracic organs, these organs must be investigated as often as the nature of the case seems to require. 7. Certain conditions which influence the physical signs both in health and disease must always be taken into account. These are the state of the chest-walls, as regards the amount of fat and muscle, and the conditions of the ribs and cartilages; the form of the thorax; the part over which the examination is made; the age and sex of the individual examined; the state of the nervous system, nervousness and hysteria materially affecting the respiratory and cardiac actions; the manner in which the breathing is carried on, many persons needing instruction as to how to perform this act; and the amount of air contained in the lungs, according to the stage of the respiratory act as regards inspiration and expiration. 8. When examining opposite sides of the chest, with the view of comparing them, care must be taken that the examination is made over corresponding regions, and in precisely the same manner. 9. A preliminary acquaintance with the principle or theory of physical examination is highly desirable, but the student must remember that by long-continued experience alone can he become thoroughly grounded in the practical application of these principles. 10. Physical signs are but indications of certain physical conditions; and, therefore, in order to appreciate the significance of the signs elicited in any particular disease, the abnormal physical conditions associated with such disease must be clearly understood.

#### DIVISIONS OF THE THORAX.

For the purpose of describing the situation and limits of physical signs elicited in connection with the chest, certain imaginary lines are drawn, and regions marked off, the chief of which are as follows:—

1. **Lines.**—These are drawn vertically from the top to the bottom of the chest, and are thus named according to their position:—1. *Mid-*

*sternal*. 2. Right and left *lateral sternal*. 3. *Nipple- or mammary line*. 4. *Acromial*, extending from the acromion process. 5. *Mid-axillary*. 6. *Scapular*, along the vertebral border of the scapula. 7. *Mid-spinal*.

II. **Regions**.—These may be arranged in the following manner:—

1. *Median*, included within the width of the sternum:—*a. Supra-sternal*, corresponding to the depression above the sternum. *b. Upper sternal*, extending to the lower border of the third cartilages. *c. Lower sternal*, from the third cartilages to the lower end of the sternum.

2. *Antero-lateral*, bounded internally by the margin of the sternum; externally by the acromial line on each side:—*a. Supra-clavicular*, including the space above the clavicle, and bounded superiorly by a line from the outer third of this bone to the trachea. *b. Clavicular*, corresponding to the inner half or two-thirds of the clavicle. *c. Infra-clavicular*, limited below by the lower margin of the third rib. *d. Mammary*, from the third rib to the lower border of the sixth. *e. Infra-mammary*, from the sixth rib to the lower margin of the thorax.

3. *Lateral*, bounded in front by the acromial line; behind by the axillary border of the scapula:—*a. Axillary*, from the apex of the axilla down to a line continuous with the lower boundary of the mammary region. *b. Infra-axillary*, from the above to the lower margin of the thorax.

4. *Posterior*, from the axillary edge of the scapula to the middle line behind:—*a. Supra-spinous* or *superior scapular*, corresponding to the supra-spinous fossa of the scapula. *b. Infra-spinous* or *inferior scapular*, opposite the infra-spinous fossa. *c. Infra-scapular*, below the scapula to the margin of the thorax, and extending internally to the spine. *d. Inter-scapular*, including the space between the base of the scapula and the spinous processes of the corresponding dorsal vertebrae.

## METHODS AND OBJECTS OF PHYSICAL EXAMINATION.

It is necessary to have a clear comprehension of the different methods of examination employed in connection with the chest, with regard to their nature; the manner in which they are severally performed; and what each is capable of teaching; therefore a brief outline of this part of the subject will now be given.

1. **Inspection**.—This merely signifies “the act of looking,” and all that need be said about the mode of conducting it is, that different views of the chest must be taken, from the sides and behind as well as from the front; and that a good light should be obtained. Inspection reveals:—1. The state of the *superficial parts*, as regards colour, oedema, amount of fat, fulness of the veins, and other conditions. 2. The *shape and size* of the thorax, along with which may be noted the state of the supra-sternal and supra-clavicular depressions; the direction of the ribs; the characters of the intercostal spaces; the size of the costal angles (*i.e.*, the angle between the ensiform cartilage and the contiguous rib-cartilages on each side); and the relative height of the shoulders. 3. The frequency, extent, and characters of the *respiratory movements*.

II. **Palpation, Manipulation, or Application of the Hand**.—The palmar surface of the hand and fingers is applied to the chest, in order to appreciate certain impressions which are capable of being con-



veyed by the sense of touch. In some cases it may be necessary to grasp the sides, particularly in the examination of children; in others only the tips of the fingers need be used, especially when it is desired to localize the signs; but generally it is best to feel with as much of the hand as can be applied. The objects of palpation are:—1. To give more accurate information with regard to what is revealed by inspection. 2. To determine the existence and characters of various kinds of *fremitus*. This term comprehends certain tactile sensations conveyed to the surface of the chest, and which are classed as:—(i.) *Vocal fremitus*, produced by the act of speaking or crying. (ii.) *Tussive fremitus*, originated by coughing. (iii.) *Rhonchal fremitus*, due to the passage of air during the act of breathing through the air-tubes or into cavities, when certain physical conditions are present. (iv.) *Friction fremitus*, elicited by the rubbing together of roughened surfaces of the pleura. 3. To detect the presence of any *fluctuation*; or of *succussion-movement*.

III. **Mensuration or Measurement.**—In some cases it is important to obtain accurate information as to the size and shape of the chest; and the extent of the respiratory movements. For this purpose certain measurements are taken, either while the chest is at rest, or in different states as regards expiration and inspiration. The only measurements likely to be required are:—1. *Circular* or *circumferential*, in different parts of the chest. 2. *Semi-circular*, so as to compare the two sides. 3. *Antero-posterior*, in the middle line and on either side, especially under the clavicles; and *transverse* or *lateral*; also noticing the relations of these two diameters. 4. *Vertical*, from the middle of the clavicle to the lower margin of the thorax. 5. *Local*, particularly from the nipple to the mid-sternal line; and from the same point to the clavicle on each side. The requisite instruments include an ordinary tape-measure; a double tape-measure, made by uniting two tapes in such a manner that they start in opposite directions from the same point, which is useful for comparing the two sides, the point of junction being applied to the middle line behind, and the tapes drawn round, one on each side of the chest, until they meet in the mid-sternal line; and different movable calipers, by which the diameters are taken. Several ingenious instruments have been invented for the purpose of indicating the respiratory movements very precisely, such as the *stethometer*, *thoracometer*, and *stethograph*, but in my opinion they are not of much clinical use. The same remark applies to the *spirometer*, by which the vital capacity of the lungs is determined; and to the *pneumatometer*, which measures the force of inspiration and respiration.

In some instances it is essential to get an outline of the shape as well as of the size of the two sides of the chest in different parts, in order to determine the exact capacity of each half, as this depends much on the form, the measurement being sometimes actually less on the side which has the larger sectional area and volume. This is accomplished by means of the *cyrtometer*, and a convenient form of this instrument consists of two portions of flexible lead-tubing of small calibre, united by a short piece of india-rubber tubing. The latter is fixed over the spine, and the two parts of lead-tubing are brought round the sides until they meet in the middle line in front. When the apparatus is removed it indicates the shape as well as the size of each lateral half of the chest, and the outline may then be traced on paper.



**IV. Percussion.**—By this term is meant “the act of striking,” and it affords signs of the highest value in diagnosis. There are two ways of performing percussion, namely, first, by striking the part under examination immediately, which is called *immediate* or *direct* percussion; secondly, by placing something on the surface, technically named a *pleximeter*, and percussing over this, which is termed *mediate* or *indirect* percussion. As a rule the latter mode should be employed, but the former is useful sometimes, especially when percussing over a bone, such as the clavicle. A great deal of discussion has been carried on as to the relative value of instruments or of the fingers in percussion. Some practitioners use small plates made of ivory or other materials to place on the chest; and a light flexible hammer or *plessor* to strike with. Other instruments are also employed, but without entering into any discussion on the subject, I venture to express the opinion that the use of the fingers is much preferable, and answers every necessary purpose. The following description may give some notion as to how this mode of examination is to be conducted:—One of the fingers of the left hand should be used as a pleximeter, especially the fore- or middle-finger, but the little-finger is conveniently applied when percussing above the clavicle. It must be laid on evenly and firmly, with the palmar aspect next the surface of the chest. Percussion must then be made with the ends of the fingers of the right hand, the nails being appropriately shortened. Some percuss with all the fingers, either arranged in a line, or gathered into a cone with the thumb; others employ only three, two, or one. It is well to practise all methods, but ordinarily the fore- and middle-fingers together answer best, or the middle-finger alone. The force employed must vary according to circumstances, but usually it should only be moderate. The stroke should be made perpendicularly to the surface; from the wrist; quickly and sharply; the fingers not being allowed to remain on for too long a time.

The objects of percussion are:—1. To elicit certain *sounds*. 2. To bring out different *sensations*, such as the amount of resistance, elasticity, fluctuation, and other objective feelings. These physical signs will be considered in detail further on.

**V. Auscultation.**—This signifies the “act of listening,” which may also be performed in two ways. The ear may be applied to the chest either directly, or only with the intervention of a handkerchief, or towel, or part of the clothing, which is termed *immediate* or *direct* auscultation; or the *stethoscope* is employed as a medium of communication between the part to be examined and the ear of the operator, this mode of examination constituting *mediate* or *indirect* auscultation. For many reasons the latter method should be adopted as a rule; but the former is often practised with advantage, especially in the examination of children, and when auscultation is performed over the back. It is impossible here to enter into any discussion with regard to the numerous stethoscopes which have been recommended. A great deal more depends upon the ability to auscult, and a knowledge of what is to be heard, than on the kind of instrument employed; and any simple ordinary stethoscope answers perfectly well, if the auscultator is competent. A convenient instrument is one made of a single piece of wood, such as deal or cedar, with a moderate-sized hollow stem, a well-fitting and slightly-hollowed ear-piece, and a rather small chest-extremity, which will fit into the intercostal spaces. In using the stethoscope, care must be

taken that the chest-end is applied in its whole circumference, without undue pressure; and also that the ear is properly placed upon the ear-piece. During auscultation the instrument must not be held by the fingers; and care must be taken that it is not touched by clothing or any other article which might occasion abnormal sounds. Special forms of binaural stethoscopes are now much employed.

So far as the *respiratory organs* are concerned, the objects of auscultation are to investigate *sounds*, namely:—1. The *respiratory* or *breath-sounds*. 2. *Râles* or *rhonchi*, which include adventitious sounds originating in the lungs or air-tubes from certain abnormal physical conditions. 3. *Friction-sounds*, due to the rubbing together of roughened surfaces of the pleura. 4. Certain peculiar sounds, namely, *metallic tinkling*, *amphoric echo*, and the *bell-sound*, which are met with in rare conditions of the lungs or pleuræ. 5. *Vocal resonance*, or the sound produced during the act of speaking or crying. 6. *Tussive resonance*, or the sound elicited by the act of coughing.

**VI. Hippocratic Succussion.**—This mode of examination is rarely required, and it simply means “shaking the patient,” when a *splashing sensation* is felt, or a *splashing sound* is heard in exceptional cases, where a mixture of air and fluid exists in the pleural cavity or a large vomica.

**VII. Determination of the Displacement of Organs** is ordinarily ranked as a special method of examination, but in reality it is merely a conclusion founded on the information derived from some of the other modes already considered. At the same time displacement of organs, both thoracic and abdominal, often gives most valuable indications in the diagnosis of abnormal conditions.

**VIII. The Use of the Aspirateur or Exploratory Trochar** may be classed as a mode of examination, and these instruments occasionally afford most important assistance in the diagnosis of morbid conditions within the chest.

Other modes of examination have been described, but they are not of sufficient practical value to call for notice in this work.

### SPECIAL PHYSICAL SIGNS.

Having given this outline of the modes of examination, the physical signs will now be considered under the following headings:—I. SHAPE AND SIZE OF THE CHEST. II. MOVEMENTS OF RESPIRATION. III. VARIOUS KINDS OF FREMITUS. IV. PERCUSSION-SIGNS. V. AUSCULTATION-SIGNS. VI. SUCCUSSION-SIGNS.

#### I. SHAPE AND SIZE OF THE CHEST.

The size and shape of the chest are ascertained by *inspection*, *palpation*, and *measurement*. They may be considered together, as they generally bear a close relation to each other, the thorax being larger in proportion as it approaches the circular form; while both its capacity and its shape depend considerably on the direction of the ribs, and on their relative position to each other. In healthy children the thorax is comparatively large, and nearly circular in form; in adults it is usually more or less elliptical during ordinary breathing, the long diameter being transverse. Many diversities of form may, however, be observed within the range of health, and though there ought to be no obvious want of symmetry between the two sides, slight differences can generally be detected.

## A. DEVIATIONS FROM THE NORMAL NOT DUE TO EXISTING DISEASE.

1. The thorax may be **small** and **contracted** in its capacity, this condition being either congenital or acquired. Two forms of small chest are met with, namely:—*a.* That in which the ribs are very oblique and the intercostal spaces wide; the chest is long and narrow in all its diameters; the costal angles are very acute; while the scapulæ are often tilted up behind, so as to resemble wings, this form of chest having on this account being termed *alar* or *winged*. *b.* That in which the front of the chest is flattened, so that the antero-posterior diameter is very small.

2. Some important **deformities** of the chest are originated during the period of infancy and childhood, in consequence of an insufficient amount of air entering the lungs during the act of breathing. This may be due either to some obstruction in the air-passage; to a weak condition of the muscles which expand the chest; or to both these causes combined; and the deformities are more liable to occur in proportion to the yielding nature of the thoracic walls. When these conditions exist, sufficient air does not reach the lungs during inspiration to enable them to fill up the vacuum produced by the descent of the diaphragm, and hence the chest is driven in to a greater or less degree by external atmospheric pressure, being also partly drawn in by the action of the diaphragm and possibly of other muscles. The chief diseases originating the obstruction which leads to deformity of the chest are bronchitis; whooping-cough; laryngismus stridulus; croup; and chronic enlargement of the tonsils.

Four special deformities of the chest have to be described, namely:—

*a. Transversely-constricted.* This is a very frequent deviation from the normal, the lower part of the chest presenting in front a more or less deep groove or depression, passing obliquely outwards and downwards from the ensiform cartilage.

*b. Pigeon-breast.* Here there is a falling-in of the true ribs on each side, so that they become more or less straight in front of their angles, while the sternum is actually or seems to be projected forwards. Hence a transverse section of the chest would be triangular, with the base behind and the apex in front. The ensiform cartilage is also frequently bent sharply backwards at its junction with the sternum, where there is consequently an angular ridge or prominence. More or less transverse constriction is always observed along with the pigeon-breast.

*c. Anteriorly-depressed.* Occasionally the part of the sternum below the third cartilages is considerably depressed, so as to present a concavity of variable depth, carrying with it the contiguous portions of the rib-cartilages. In some instances this deformity is congenital, but this is by no means always the case.

*d. Rickety.* Many will not recognize a special form of rickety thorax, but, while it may be merely an ordinary pigeon-breast, undoubtedly the form of chest in rickets is not uncommonly very peculiar and characteristic. It is more or less flattened posteriorly as far as the angles of the ribs, where there is a marked bend; on each side a groove, varying in breadth and depth, runs obliquely downwards and outwards, which may extend from the first to the ninth or tenth ribs, but is most marked



about opposite the fifth, sixth, and seventh; this corresponds mainly to the line of junction of the ribs with their cartilages, which is indicated by a series of nodular swellings, but the bottom of the groove is formed more by the ribs than by the cartilages. In front of this groove the cartilages are more or less curved, and the sternum is somewhat prominent, so that the antero-posterior diameter is increased. The greatest lateral diameter is opposite the angle of junction between the dorsal and lateral regions; and the shortest corresponds with the bottom of the lateral depressions. The solid organs underneath will influence the form of the rickety chest.

3. The thorax may be deformed as the result of occupation; of the pressure of stays or belts; or of previous injury to, or disease of, the ribs or spinal column.

#### B. CHANGES IN SHAPE AND SIZE DUE TO EXISTING DISEASE.

1. **General enlargement.**—In this condition the chest is more or less expanded, approaching to the form and size which it presents after a deep inspiration, or even going beyond this, so as to become “barrel-shaped.” The enlargement may involve the whole length of the thorax, or only its upper or lower part. *Causes.* *a.* Emphysema usually.

*b.* Double pleuritic effusion very rarely.

2. **General diminution.**—This is the opposite of enlargement, and the thorax may assume either the alar or flattened form. It often results from phthisis, but the two sides are rarely contracted to an equal extent, while local depressions are usually observed in this disease.

3. **Unilateral enlargement.**—An enlarged side is usually more rounded than the opposite one, and appears to be short, having a comparatively long antero-posterior diameter, and a large costal angle. The corresponding shoulder is raised; and the spine tends to be curved towards the opposite side. *Causes.* *a. Conditions of the pleura.* (i.) Pleuritic effusion in the great majority of cases. (ii.) Occasionally pneumothorax or hydro-pneumothorax. (iii.) Very rarely hæmothorax. *b. Conditions of the lung.* (i.) Hypertrophy or distension of one lung. (ii.) Secondary cancer.

4. **Unilateral diminution or Retraction.**—The characters are the reverse of those observed in enlargement, the entire side being small and cramped, while the ribs are aggregated together to a variable degree. *Causes.* *a.* In most instances pleuritic adhesions, binding down the lung so that it cannot expand. In all cases of retracted side pleuritic adhesions are formed after a while. *b.* Collapse of the lung from any cause. *c.* Changes in the lung-structure diminishing its volume and power of expansion, namely, phthisis; chronic or interstitial pneumonia; primary cancer.

5. **Local enlargement—Bulging.**—This alteration necessarily varies much in its extent and form, and careful measurement may be required in order to determine it accurately. *Causes.* The most frequent and important causes of bulging of the chest are enlargement of the heart; pericardial effusion; and aneurism of one of the great vessels. The remaining causes include:—*a. Conditions of the pleura.* (i.) Empyæma pointing on the surface. (ii.) Localized pleuritic effusion. (iii.) Localized pneumothorax. *b. Conditions of the lung.* (i.) Pneumonia at the

base or apex. (ii.) A large phthisical cavity at the apex. (iii.) Localized emphysema. (iv.) Very rarely a hernial protrusion. *c.* Mediastinal tumours or enlarged glands. *d.* Enlargements of the liver or spleen. *e.* Disease of the sternum or ribs, or of their investing periosteum. *f.* Superficial abscesses and growths.

6. **Local diminution—Depression or Flattening.**—*Causes.* (i.) Phthisis, the depression being due to the local changes in the lung and the accompanying pleuritic adhesions, and especially affecting the supra- and infra-clavicular regions. (ii.) Localized pleuritic adhesions.

7. The **costal angles** and **intercostal spaces** are often altered in size; and the latter are frequently abnormally bulged or depressed, as the result of morbid conditions of the pleura. Anything that affects the size of the chest, either generally or unilaterally, will necessarily influence that of the costal angle and intercostal spaces; while the latter will also be altered locally along with any local bulging or depression.

## II. MOVEMENTS OF RESPIRATION.

The respiratory movements are investigated by *inspection*, *palpation*, and *mensuration*; some employ special instruments. When examining for disease, it is necessary to observe these movements during ordinary and forced respiration.

The following facts bearing upon the physiology of the respiratory movements must be borne in mind:—1. They are partly *costal* or *thoracic*: partly *diaphragmatic* or *abdominal*. The thoracic movements are made up during inspiration of *elevation* and *expansion*; during expiration of *depression* and *retraction*. 2. In health there is no obvious difference in the movements of the two sides. 3. In males and children the diaphragm and lower ribs chiefly act during ordinary breathing, the movements being mainly abdominal; while in females the upper part of the chest moves most, and breathing is upper costal. During forced respiration the movements are chiefly upper costal in all individuals. 4. The ordinary number of respirations ranges from 16 to 20 per minute. 5. Different observers have given different statements as to the relative length of inspiration and expiration, but it may be affirmed that there is no marked difference between them. Walshe states that if the whole act of respiration is taken as 10, inspiration may be taken as 5, expiration as 4, and the pause between expiration and inspiration as 1. 6. The intercostal spaces in most parts become rather more hollow during inspiration; as well as the supra-clavicular fossæ. This is especially noticed when a deep breath is drawn, and the sinking of the spaces is best observed towards the lower and lateral part of the chest. 7. Inspiration is almost entirely effected by muscular action; expiration chiefly by the elasticity of the lungs and chest-walls, aided somewhat by muscular force, which is called into play to a much greater degree during forced respiration.

## ABNORMAL RESPIRATORY MOVEMENTS.

The deviations from the normal which the respiratory movements present may be arranged in the following manner:—

## A. ALTERATIONS AFFECTING THE GENERAL MOVEMENTS.

**1. Alterations in frequency.**—The respirations may be counted by watching the movements; or by applying the hand over the epigastrium. Their frequency may be:—(i.) *Increased. Causes.* *a.* Most conditions which interfere with the action of the lungs in any way, and which give rise to the different forms of dyspnoea. *b.* Many cardiac affections. *c.* Certain nervous disorders, such as hysteria. *d.* An unhealthy state of the blood, for example, that present in anæmia or fevers. (ii.) *Diminished.* A slow rate of breathing is often noticed in apoplexy, narcotic poisoning, and some nervous derangements, for example, trance.

**2. General movements in excess.**—The patient breathes deeply and with unusual force, the extraordinary muscles being brought into play; the range of movement is greater; and more air is changed during each respiration. *Causes.* *a.* Anything that interferes with the functions of the lower part of the lungs, such as diseases in these organs themselves, for example, pneumonia, congestion, œdema, bronchitis; accumulations in the pleuræ; or abdominal enlargements (ascites, enlarged liver). *b.* Cardiac diseases which impede the circulation of the blood, and hence interfere with its proper aëration. *c.* Certain abnormal conditions of the blood itself, such as anæmia.

**3. General movements deficient.**—This deviation may be associated either with increased, normal, or diminished frequency. *Causes.* *a.* Anything that extensively obstructs the functions of the lungs, such as capillary bronchitis, double pneumonia, or effusion into both pleuræ. *b.* Painful chest-affections, for example, acute pleurisy or pneumonia, pleurodynia, intercostal neuralgia. *c.* Rarely interference with the action of the respiratory muscles, from spasm or paralysis. *d.* Certain conditions of the central nervous system, for instance, narcotic poisoning and trance. *e.* Rigidity of the chest-walls; or, very rarely, infiltration of their structures with cancer.

**4. Altered relation between the thoracic and abdominal movements.**—(i.) *Thoracic movements in excess, from diminished action of the diaphragm. Causes.* *a.* Usually some accumulation in the abdomen, mechanically interfering with the descent of the diaphragm, such as ascites, flatus, or a large tumour. *b.* Conditions which render movement of the diaphragm or abdominal walls painful, especially peritonitis, but also diaphragmatic pleurisy, muscular rheumatism, and inflammation of the diaphragm or abdominal walls. *c.* Extreme pericardial effusion. *d.* Paralysis of the diaphragm from any cause. (ii.) *Diaphragmatic and abdominal movements in excess. Causes.* *a.* Any condition which is attended with pain on bringing the chest-walls into play, such as pleurisy, pleurodynia. *b.* Paralysis of the thoracic muscles. *c.* Obstruction in connection with the air-passages, preventing the entrance of a sufficient amount of air.

**5. Alteration in the ratio between the expansion- and elevation-movements of the ribs.**—The only important deviation in this respect is a diminution in the expansion movement, which may amount to its complete absence. It is especially observed during forced breathing, when there may appear to be considerable movement of the chest, but none of the expansile kind. *Causes.* *a.* General emphysema, the lungs being already distended, and the chest expanded to a greater or less extent. *b.* Rigidity of the chest-walls. *c.* Anything within or



external to the lungs which either prevents them from acting, or interferes with the entrance of the air, for example, pulmonary consolidations, pleuritic accumulations or adhesions, or pressure on the air-tubes.

**6. Alterations in the rhythm of the respiratory act.**—Unequal or *jerky* breathing is often noticed in certain nervous disorders, such as chorea and hysteria. The most important change in rhythm, however, is that in which the *relative length of inspiration and expiration becomes disturbed*, the former being more or less short and quick; and the latter prolonged, slowly performed, and often laboured—**Expiratory dyspnœa.** *Causes.* *a.* Diminution in the elasticity of the lung-tissue and chest-walls, which is particularly observed in emphysema with rigid chest. Expiration becomes then either entirely a muscular act; or far more so than in the normal condition. *b.* Some obstruction to the escape of air through the principal air-passages; or narrowing of a large number of bronchi.

**7. Depression of the chest-walls during inspiration.**—**Inspiratory dyspnœa.**—Instead of expanding during inspiration, the chest may fall in to a greater or less extent, especially at its lower part, producing either temporarily or permanently one of the forms of deformed thorax already described. This deviation is chiefly observed in children. *Causes.* *a.* Almost invariably some obstruction to the entrance of air into the lungs. The conditions to which such obstruction is mainly due are:—(i.) Bronchitis. (ii.) Hooping-cough. (iii.) Anything tending to occlude the larynx or trachea, such as croup, œdema glottidis, laryngismus stridulus, or the pressure of a tumour or aneurism. (iv.) Enlarged tonsils or other impediment connected with the pharynx. *b.* Occasionally, it is said, very rapid œdema of the lung, or hydrothorax.

#### B. ALTERATIONS AFFECTING UNILATERAL MOVEMENTS.

**1. Inequality of the respiratory movements on opposite sides.**—This is usually due to *deficient or complete want of expansion* on one side. *Causes.* *a.* Some accumulation in one of the pleural cavities, or adhesions binding down the lung, and preventing its expansion more or less. *b.* Changes in the lung-tissue on one side, interfering with its inflation; for example, acute or chronic pneumonia, phthisis, cancer. *c.* Pressure on either chief bronchus by a tumour, or obstruction of its canal, air being thus prevented from passing into the lung. *d.* Painful affections of one side. *e.* Unilateral paralysis of the muscles rarely. When the movements of one side are interfered with, those of the opposite side frequently become excessive, owing to the corresponding lung having to perform extra work.

**2. Altered relation of the abdominal to the thoracic movements on one side.**—This is certainly observed occasionally, but it is not of much consequence as a rule.

**3. Unilateral inspiratory dyspnœa.**—The chest-wall may fall in on one side during inspiration, owing to obstruction of a main bronchus.

#### C. LOCAL CHANGES IN MOVEMENT.

**1.** The common deviation met with is a **local deficiency**, in which both expansion and elevation are involved, but especially the former.

The usual causes of this change are phthisis; and localized pleuritic adhesions.

2. Occasionally a **limited falling-in** of the chest during inspiration is observed, due to obstruction of one of the smaller bronchial divisions.

#### D. ABNORMAL MOVEMENTS OF THE INTERCOSTAL SPACES.

In pleuritic effusion, pneumonia, and other conditions which affect the movements of the chest, the intercostal spaces frequently do not exhibit their usual changes of form during the act of breathing, and in some instances of extreme pleuritic effusion an undulatory motion is perceptible. These spaces are often quite motionless over the seat of local deficiency in movement.

### III. EXAMINATION OF VARIOUS KINDS OF FREMITUS.

A. VOCAL AND CRY-FREMITUS.—Vocal fremitus is investigated by applying the hand to the surface, while the patient repeats the words “ninety-nine,” or counts from one to ten, or answers a question. As already remarked, the cry answers the same purpose in children. The normal variations due to the quality of the voice; the age and sex of the individual; the state of the chest-walls; and the part of the chest over which the examination is made, must be borne in mind. As a rule vocal fremitus is more marked on the right than on the left side, especially over the upper part of the chest in front.

The changes affecting vocal fremitus which may be observed in disease are:—

1. **Changes in area.**—This may be—(i.) *Increased*, in distension of the lungs from emphysema or hypertrophy. (ii.) *Diminished*, when the lung is retracted by adhesions; or pushed aside by some solid mass, such as an enlarged heart, or a tumour.

2. **Changes in intensity.**—(i.) *Increased*. *Causes.* *a.* Consolidation of the lung from any cause, provided that the consolidating material is not too abundant, or too dense or pulpy; and that it encloses tubes or spaces containing air. Vocal fremitus is especially marked if at the same time the bronchial tubes are dilated; or if cavities of certain characters exist in the lungs. Hence increased vocal fremitus is an important sign of pneumonia, phthisis, chronic pneumonia with dilated bronchi, some cases of cancer, and similar conditions. *b.* Condensation of the lung from compression or collapse. *c.* Bronchitis, congestion or cedema of the lungs, and pulmonary apoplexy, but the fremitus is by no means constantly increased in these affections, and is of little importance. (ii.) *Diminished* or *suppressed*. *Causes.* *a.* Separation of the lung from the chest-wall by some intervening imperfectly-conducting material, for example, fluid or air in a pleural cavity; or enlarged organs or morbid growths encroaching upon the cavity of the thorax. *b.* Very extensive, as well as dense or pulpy consolidation of the lungs, with obliteration of the tubes, so that no air can enter, as in extensive soft cancer, certain cases of phthisis, and pneumonia with rapid and abundant exudation. *c.* Distension of the lungs in emphysema.

These alterations in the intensity of the vocal fremitus may be observed over a very limited region, or over a considerable part of the chest. It is at the base and apex of the lungs that they are chiefly important, especially in distinguishing between fluid effusion and pneumonic consolidation at the base, and in aiding in the diagnosis of consolidation at the apex, mainly in phthisis. Increase and deficiency may be noticed in different parts of the thorax on the same side; thus, in cases of pleuritic effusion the fremitus is often absent below, but in excess above, on account of the compressed state of the lung.

**B. TUSSIVE FREMITUS.**—This is affected in much the same way as vocal fremitus, but it is of little importance, except when the voice is very weak, and hence cannot be made use of.

**C. RHONCHAL FREMITUS.**—Due to the passage of air through bronchial tubes containing thick mucus or some other fluid, rhonchal fremitus becomes an important sign of bronchitis or œdema, especially in the case of young children. It may also be felt occasionally over cavities in the lungs, especially with cough.

**D. PLEURITIC FRICTION-FREMITUS.**—Not often met with, this fremitus indicates the presence of much firm material in connection with the pleura. It is most frequently observed during the later stages of acute pleurisy; but may be very marked in cases of chronic dry pleurisy.

#### IV. PHYSICAL SIGNS OBTAINED BY PERCUSSION.

##### A. PERCUSSION-SOUNDS.

**SOUNDS IN HEALTH.**—Five distinct sounds may be obtained by percussion in a healthy subject, which differ in their degree of resonance, length, fulness, pitch, and clearness.

**1. Tympanitic or Drum-like.**—In this country the term *tympanitic* is applied to the sound which is elicited by percussing over the abdomen, being dependent upon accumulation of gas within the stomach and intestines. It has considerable resonance; is of prolonged duration, low in pitch, and full; being either more or less muffled or clear, according to the degree of distension.

**2. Pulmonary or Sub-tympanitic.**—This is the sound brought out by percussing over healthy lungs, and which is therefore elicited over the greater part of the chest. Possessing a fair amount of resonance, it is shorter, less full, and higher-pitched than the tympanitic sound, and ordinarily is muffled. It has been likened to the sound elicited by striking over a "muffled drum."

**3. Laryngeal, Tracheal, or Tubular.**—As these names indicate, this sound is produced over the main wind-pipe. Much less resonant than those already described, it is also considerably shorter and of higher pitch, and has a tubular quality; as a rule it is not clear, on account of the structures which cover the air-tube.

**4. Bony or Osteal.**—It is not always possible to obtain the osteal sound distinctly over the healthy chest, but it imparts its characters to other sounds, especially over the sternum and clavicles. It can be observed over any of the bony prominences of the body. With hardly any resonance, this sound is very short, high-pitched, and tolerably clear.



5. **Dull or Non-resonant.**—When percussion is made over organs and other structures which are solid, the sound elicited is more or less dull, short, and abrupt; it is supposed that each organ gives rise to a sound having a peculiar pitch of its own, but certainly few persons are able to appreciate this difference.

#### CHANGES AFFECTING THE PERCUSSION-SOUNDS.

When percussing the chest with the view of obtaining evidence of disease by the aid of the sounds elicited, the points to be noticed are:—  
1. Whether there is any actual change in the characters of the sound elicited, either over the chest as a whole, or over any part of it. 2. If there is any increase or diminution in the extent over which the normal pulmonary sound is heard. 3. Whether the proper difference is noticed between the sounds produced after a full inspiration and after a deep expiration respectively, either generally or locally, both as regards characters and area. 4. If superficial and deep percussion yield different results.

##### (A.) Changes in the characters of the Pulmonary sound.—

1. *The percussion-sound may become hyper-resonant or actually tympanitic.*—*Causes.* (i.) Pneumothorax, provided the amount of air in the pleura is not so great as to stretch the chest-walls to an extreme degree, when it is found that the sound becomes muffled or dull. (ii.) Certain states of the lungs, in which they contain excess of air in proportion to the solid tissues, this being often combined with more or less distension of the air-vesicles, namely, emphysema, hypertrophy, atrophy, extreme bloodlessness. In these conditions the sound is more or less hyper-resonant, but rarely actually tympanitic.

2. Without any marked alteration in quality, *the pulmonary percussion-sound may become unusually clear*, as in some cases of bronchitis, congestion, œdema of the lungs, or the early period of pneumonia. This is due to “liquid or solid being mixed intimately with air-containing tissues,” and the sound under these conditions may even assume a tubular quality.

3. *Resonance may be more or less diminished, to absolute dulness.* When this deviation exists, it is necessary to note the degree of the change, which may vary from a slight deficiency in resonance to the most complete dulness; its situation and extent; the form of the dulness; the pitch of the sound elicited; and, in some cases, whether alteration in posture has any effect upon it. There are two dull percussion-sounds which call for special mention, namely:—1. The *hard wooden* sound, which is very short and abrupt; almost non-resonant; exceedingly high-pitched; and accompanied with a sensation of much resistance. 2. The *putty-like* sound, this being, as its name suggests, an absolutely non-resonant, dull, heavy, and dead sound. *Causes.* It may be useful to enumerate here all the causes which are capable of giving rise to abnormal dulness in various parts of the chest. They include—(i.) Certain affections of the chest-walls, such as infiltrated cancer, diseased or inflamed bone and its consequences, periostitis. (ii.) Fluid accumulations in the pleural sac, whether of serum, pus, or blood; or its extreme distension with air. (iii.) Consolidation of the lungs, as in pneumonia, phthisis, thickening of the bronchi, cancer. (iv.) Excessive collection of fluid in the bronchi, air-vesicles, or lung-tissues, due to

extensive bronchitis, congestion, or œdema; or a local collection of fluid, such as an abscess or hydatid-cyst. (v.) Considerable collapse or compression of the lungs; or, on the other hand, extreme distension of these organs. (vi.) Enlargements in connection with the heart; or solid or fluid accumulations in the pericardium. (vii.) Mediastinal enlargements of all kinds, such as various tumours, enlarged glands, abscess, aneurism. (viii.) Enlarged or displaced abdominal organs, especially the liver or spleen; or, rarely, tumours extending upwards from the abdomen.

4. *Peculiar sounds.* *a. Tubular.* This sound is not uncommonly met with in some parts of the chest, but never over any great extent. It resembles that produced over the trachea, varying somewhat in its pitch, and usually being quite clear. *Causes.* (i.) Cavities in the lungs, not too large, either superficial or having some firm, well-conducting tissue between them and the chest-walls, and containing little or no fluid. Such cavities are generally associated with phthisis, but may possibly be due to enlarged bronchi. (ii.) The presence of some solid mass in the chest, not of too large a size, and intervening between the trachea or one of the main bronchi and the surface, forming a well-conducting medium, for example, any mediastinal tumour, but especially enlarged glands in the posterior mediastinum, the tubular sound being then heard in the interscapular regions, particularly in children. (iii.) Certain conditions in which the lower part of the chest is invaded upon, so that the lung is pushed or floated upwards, when it is partially in a relaxed, partly in a condensed condition; a tubular sound may then be perceptible under the clavicle. This is often observed in cases of pleuritic effusion; and occasionally in connection with tumours in the chest or enlarged abdominal organs, or consolidation of the lung itself, for example, basic pneumonia.

*b. Amphoric.* This is a peculiar resonant sound of very rare occurrence, having a markedly hollow and metallic character. *Causes.* (i.) A very large phthisical cavity in the lungs, situated near the surface; usually with adherent pleura; having smooth, thin, but firm walls; and containing chiefly air, with but little fluid. (ii.) Pneumothorax occasionally.

*c. Metallic.* This is a high-pitched sound, of distinctly metallic quality, not unfrequently tinkling or splashing. It is not always easy to distinguish it from the *crack-pot* sound, and both may be elicited in the same case, according to the force used in percussion, as they depend upon similar physical conditions, namely, a cavity containing air.

*b. Crack-pot or cracked-metal sound.*—"Bruit de pot fêlé." Also of metallic quality, this has in addition a cracked character, as its name indicates, and gives the impression of air being driven out through a chink or small aperture. It may be imitated somewhat by claspings the hands loosely together, and striking the back of either of them over the knee, so as to drive out the enclosed air. *Causes.* In the chest the crack-pot sound is caused by air enclosed between two surfaces, the anterior of which is yielding, being suddenly expelled through an orifice, and it may be met with under the following conditions:—(i.) It is most important as a sign of a cavity in the lung, which must be of good size, tolerably superficial, containing air, having one or more bronchi opening into it, and its front wall being more or less yielding. These conditions are rarely fulfilled except by phthisical cavities at the apices of the lungs, and hence the sign is most commonly observed in one or other infra-clavicular region. In order to elicit the crack-pot sound, the patient should open his mouth and turn towards the operator, and per-

cussion must be made firmly, but rapidly and abruptly, during the act of expiration. (ii.) In children affected with bronchitis, or even when very young infants merely cry, a sound resembling a crack-pot sound may be brought out in many parts of the chest, but there ought to be no difficulty in distinguishing this sound from that due to a cavity. It must be mentioned, however, that a similar sound may occasionally be elicited in subjects 10 or 12 years old, who have very soft and yielding chest-walls. (iii.) In rare instances this sign is observed over the front of the upper part of the chest in cases of pleuritic effusion, or of consolidation involving the posterior part of the lung.

(B.) **Change in the extent of pulmonary resonance.**—The area of pulmonary resonance may be:—1. *Increased*, when the lungs are distended with air, especially from emphysema, but also as the result of hypertrophy, or of temporary inflation. 2. *Diminished*. It is not easy to separate this deviation from dulness, still it not infrequently becomes an important sign of a lung being contracted within its usual limits.

(C.) **Effects of inspiration and expiration upon the percussion-sound.**—1. *There may be no extension in area, or increase in amount of resonance, after a full inspiration, either generally or on one side; and no diminution in the same after a deep expiration.* *Causes.* (i.) Extreme distension of the lungs, with loss of elasticity, as in emphysema. (ii.) Some obstruction to the passage of air through the air-tubes, for instance, from pressure on a bronchus, considerable bronchitis, or spasmodic asthma. (iii.) Interference with the expansion of the lung, either from external pressure upon it, such as that of pleuritic effusion or adhesions; or from extensive disease in the organ itself. This only affects the results of inspiration. (iv.) Air in the pleural cavity, which cannot be increased or diminished in quantity by the act of breathing.

2. In cases of suspected consolidation at the apex of a lung, where the signs are not marked, it is very important to notice whether there is the normal difference in the percussion-sounds after a deep inspiration and a forced expiration respectively, as regards the amount and area of resonance, and alteration in pitch.

(D.) **Difference between superficial and deep percussion.**—This may be of much use in determining the precise physical conditions present in a phthisical lung. For instance, superficial percussion may give rise to hard dulness, showing the presence of much fibroid consolidation; while on deep percussion a crack-pot sound may be elicited, indicating the existence of a cavity beneath the consolidation.

## B. SENSE OF RESISTANCE OR ELASTICITY.

During the act of percussion the sensation which is conveyed to the fingers should always be noted. The information thus obtained in connection with the thorax is mainly useful:—1. In making out the state of the chest-walls, as to the degree of rigidity, elasticity, distension by air, and other conditions. 2. In distinguishing between dulness due to fluid or to some solid material. 3. In determining the amount and actual density of any solid accumulation.



## V. PHYSICAL SIGNS OBTAINED BY AUSCULTATION.

## A. RESPIRATORY OR BREATH-SOUNDS.

**BREATH-SOUNDS IN HEALTH.**—In health three typical sounds may be heard during the act of breathing, on listening over different parts of the respiratory apparatus.

1. **Tracheal or Laryngeal.**—Heard on applying the stethoscope immediately over the wind-pipe in the front of the neck, this sound has the following characters:—It is very loud, more or less hollow, and high-pitched; begins simultaneously with the act of inspiration, and continues of the same intensity throughout; presents a marked interval between its inspiratory and expiratory portions; and the latter is not only well heard, but is rather the longer, louder, and higher pitched. This sound is laryngeal in its origin.

2. **Bronchial.**—This differs from the former in the following particulars:—It is not at all hollow, nor is it so loud or high-pitched; has a harsh quality; is not quite so rapidly evolved; does not present such a distinct interval between inspiration and expiration; and the latter is shorter than in the tracheal sound. Bronchial breathing may be heard normally in some individuals in the interscapular regions; as well as over the upper part of the sternum and contiguous end of the clavicles. This sound is laryngeal in origin, modified by conduction along the larger bronchi.

3. **Pulmonary or Vesicular.**—On listening over the greater part of the chest, a soft breezy sound is usually heard during inspiration, which is gradually developed, but continuous; no interval can be observed between it and the expiratory sound; and the latter, when present, is very much shorter and more feeble than the inspiratory sound, though rather harsher and lower pitched, but not infrequently it is quite inaudible. Some authorities maintain that the pulmonary breath-sound is actually produced in connection with the air-vesicles; others that it is either entirely or in part the laryngeal sound conducted and modified; probably it is both vesicular and laryngeal in its origin.

Several conditions influence the breath-sounds in health, but only *age* and *sex* can be here alluded to. In children they are very loud, and expiration is prolonged, this breathing being termed *puerile*. In aged persons the sounds are weak, but expiration is usually lengthened, owing to degeneration of the lung-tissue. In females they are frequently loud, and may be of a jerky character.

## CHANGES IN THE BREATH-SOUNDS OVER THE CHEST DUE TO DISEASE.

(A.) **Changes in Intensity.**—*The breath-sounds may be weakened in various degrees, or completely annulled, either over a limited region; over one side; or over the greater part or the whole of the chest. In some conditions they seem to be deep and distant. Causes. a.* Anything interfering with the entrance of air through the air-passages in o the lungs, whether due to spasmodic or other form of constriction, internal obstruction, or external pressure. *b.* Imperfect respiratory movements

on account of pain, paralysis or spasm of the muscles, or any other cause. *c.* Permanent distension of the lungs, so that little or no air can enter into the vesicles, as in extensive emphysema. *d.* Any condition which by pressing upon the lungs prevents them from expanding, or which interferes with the transmission of sound, for example, pleuritic accumulations of all kinds, extensive adhesions, abdominal enlargements encroaching upon the chest, or intra-thoracic tumours. *e.* Very extensive or dense consolidation of the lungs, as in extensive cancer, and certain cases of phthisical or pneumonic consolidation. *f.* Conditions in which the respiratory sounds are obscured by râles, for instance, capillary bronchitis or pulmonary oedema.

2. *The respiratory sounds may be puerile, being increased in intensity, the expiratory portion becoming then unusually distinct. Causes.* *a.* If one lung, or any portion of one or both lungs is called upon to do extra work, in consequence of interference with the functions of the other lung or parts, the respiratory sounds become puerile over the corresponding regions. This is observed, for instance, in cases of pleuritic effusion or adhesions, consolidation of portions of the lungs, or obstruction of a bronchus. *b.* When a bronchial division is suddenly relieved of spasm or obstruction, the breathing becomes exaggerated over the portion of lung to which its ramifications pass.

(*B.*) **Changes in Rhythm.**—Many deviations in the rhythm of the breath-sound are described, but only two can be readily appreciated, so as to become practically useful.

1. *The breath-sounds may become more or less jerky or wavy, so as sometimes to have a cogged-wheel rhythm, especially the inspiratory sound.* This is by no means a reliable sign of disease, especially in females, in whom it is often met with if they are at all nervous or hysterical, while it frequently depends upon excited cardiac action. *Causes.* Jerky breathing may be observed:—*a.* In painful affections of the chest, such as early pleurisy or pleurodynia, when the patient often breathes in an interrupted manner. *b.* In the early stage of phthisis. *c.* In connection with pleuritic adhesions.

2.—*The most important change in rhythm is a prolongation of the expiratory sound, so that it may become twice or even three or four times the length of that of inspiration, which is often actually shortened.* Prolonged expiration generally attends certain alterations in the breath-sounds to be immediately noticed, but it may be the main or the only alteration observed. *Causes.* *a.* Emphysema, the elasticity of the lungs being more or less diminished. *b.* Obstruction to the exit of air through the respiratory passages.

(*C.*) **Change in the area over which the breath-sounds are heard.**—This may be:—1. *Increased*, from distension of the lungs. 2. *Diminished*, owing to their retraction.

(*D.*) **Changes in quality, along with other characters.**—Some very important abnormal breath-sounds are met with, which differ entirely from those ordinarily heard over the chest, in their quality, pitch, rhythm, and other particulars.

1. *Harsh or rough breathing.*—As its name indicates, this merely implies a harshness of the breath-sound, its soft and breezy character being wanting, which is specially marked during expiration, this portion being unduly lengthened. Harsh breathing is not very reliable as evidence of disease, but is frequently observed in connection with

slight consolidations, bronchial catarrh, the early period of pneumonia, and various other morbid conditions.

2. *Bronchial*.—In its characters bronchial breathing corresponds with the normal sound thus named, but it is heard in unusual regions, or is unusually marked. *Causes*. *a*. Consolidation of the lung-tissue, if moderate in amount and contiguous to the surface, for example, in phthisis, cancer, chronic pneumonia, and some cases of acute pneumonia. Even when the lungs enclose small cavities or dilated bronchi, the breathing is often merely of a bronchial character. *b*. Condensation of the lung from compression or collapse.

3. *Blowing*.—Although approaching on the one hand to bronchial breathing, and on the other to tubular, this sound has sufficiently distinctive characters to merit recognition. It differs from the former in its markedly “blowing” quality, being much clearer and higher-pitched; from the latter in being diffused, and not hollow, or as if it originated in a localized tube. *Causes*. *a*. Diffused pulmonary consolidation, not too abundant, especially in certain parts, as in some cases of phthisis and pneumonia. *b*. Small cavities or dilated bronchi surrounded by solid material. *c*. Occasionally conduction of the sound from a large bronchus to the surface by a solid medium.

4. *Tubular*.—A high-pitched, concentrated, somewhat hollow and metallic sound, this much resembles the normal tubular breathing heard over the trachea, and gives the impression of being directly conveyed from a tube. *Causes*. *a*. Acute pneumonia, in which disease the sound often presents the most typical characters. *b*. Certain cavities in the lungs. *c*. A solid mass of moderate size intervening between the trachea or a main bronchus and the chest-wall, so as merely to form a medium of communication between them.

5. *Cavernous*.—This is a clear and distinctly hollow sound, varying in its pitch, which tends to be low, especially during expiration. It is usually only perceived over a limited area, and strikes the ear as being produced in a hollow space, its exact characters varying according to the dimensions and other characters of this space. *Causes*. *a*. A cavity in the lung, of some size, tolerably superficial, and not containing much fluid. *b*. In rare instances, it is said, consolidation around an ordinary-sized bronchus.

6. *Amphoric*.—A still more hollow sound, and of peculiar metallic quality, amphoric breathing resembles the sound heard on blowing into a large empty glass bottle or metallic vessel. *Causes*. For its production it is necessary to have an extensive empty cavity, with firm and smooth walls, into which air enters more or less freely. These conditions are found in:—*a*. Pneumothorax most frequently, air entering the pleura through a communication opening from the lung. *b*. Phthisical excavation in the lung very rarely.

The morbid sounds just described may be heard in different parts of the same chest; or they may be observed in succession over the same region as the physical conditions change, there being no marked boundary-line between them, but a gradual transition from one to another. For instance, in phthisis the breathing, which is at first harsh or bronchial, may become blowing, especially in certain regions, and then in succession tubular, cavernous, or even amphoric, as cavities form and increase in size. The hollow sounds, though usually present during inspiration and expiration, may only be heard during the former



act. A deep breath usually causes them to become much louder; and frequently a sharp cough enables them to be heard where they did not exist before, owing to the displacement of some obstructing secretion, or the discharge of fluid from a cavity. These sounds may appear to be superficial and strong; or more or less deep and feeble. Care must be taken not to mistake a conducted pharyngeal or laryngeal sound for cavernous respiration.

7. In exceptional instances peculiar respiratory sounds are met with. In connection with some cavities, they may have a *sucking* or *hissing* character. Sometimes the air appears to be drawn away during inspiration, and puffed back during expiration; this is termed the *souffle* or *veiled puff*.

### B. RÂLES OR RHONCHI.

These terms are applied to certain adventitious sounds which are originated within the lungs or the air-tubes. Before proceeding to their consideration it is necessary to mention that they may be simulated by sounds induced by the act of breathing, and resulting from contraction of the muscles of the chest-walls; subcutaneous cedema or emphysema; hairs on the surface of the thorax; fluid in the mediastinal cellular tissue; or the opening-up of healthy lung-tissue during a deep inspiration.

Râles or rhonchi are generally produced by the passage of air during the act of breathing:—(i.) Through bronchial tubes narrowed by thickening of the mucous membrane; by various deposits upon the surface of this membrane, such as thick secretion or exudation; by organic changes in the walls of the tubes; or by spasm of their muscular fibres. (ii.) Through fluids of variable consistence, contained in normal or enlarged air-tubes or vesicles. (iii.) Through fluid contained in cavities in the lungs. (iv.) Through substances originally solid, which are undergoing a process of softening. (v.) Into air-vesicles, either of normal size or enlarged, the walls of which are collapsed or stuck together, thus causing them to open up. In rare instances the action of the heart originates rhonchal sounds, when there is much fluid in contiguous tubes or cavities.

The following points must be noticed with regard to these adventitious sounds:—1. Their characters, namely, whether dry or liquid; large or small; peculiar quality, such as musical, crackling, bubbling, gurgling, whistling, &c.; pitch; whether at all hollow or metallic, and the degree in which this character is present. 2. If they are heard both during inspiration and expiration, or only during one of these acts. 3. Their situation and extent. 4. Their amount. 5. Whether they are constant or only heard at intervals; and if they are affected by a full inspiration or cough.

### CLASSIFICATION AND SPECIAL CAUSES OF RÂLES OR RHONCHI.

So many different classifications of râles or rhonchi have been invented, and such confusion has prevailed with regard to the meaning of the various terms employed, that it is by no means easy to fix upon any classification. I have, however, adopted the following arrangement:—

(A.) **Vibratory or Dry Musical Rhonchi.**—These sounds are produced by air traversing air-tubes narrowed in some of the ways already mentioned. They vary in their precise characters according to the size of the tube affected, and the immediate cause of the narrowing. They are divided as follows :—1. *Sonorous*. This is a deep-toned, low-pitched sound, varying much in its exact quality, which may be snoring, growling, humming, cooing, &c. ; it appears to be superficial, is often extensively heard, and generally accompanies both inspiration and expiration, but may be limited to either act, especially the latter. 2. *Sibilant*. This is of much higher pitch, and is often whistling, hissing, or musical ; it is not so extensively heard as the sonorous rhonchus ; and as a rule accompanies both inspiration and expiration. Both these classes of rhonchi are liable to much irregularity, disappearing from time to time, especially after a cough, and they often occur together. *Causes*. *a*. Bronchitis, especially chronic, but also the acute form in its early stage, and the plastic or fibrinous variety. *b*. Spasmodic contraction of the bronchial tubes in cases of asthma.

(B.) **Crepitant Râles.**—1. *True crepitant râle*. This is a sound met with in the early stage of acute pneumonia, and therefore usually observed towards the base of one lung, but it may be heard over any part of the organ which is the seat of the inflammatory process. It consists of a great number of extremely minute, sharp, crepitant sounds ; equal in size ; perfectly dry ; heard in short puffs during inspiration alone in most cases, and often only towards the termination of this act ; and increased in amount by a deep breath. It has been aptly compared to the sound produced by rubbing a lock of hair firmly between the finger and thumb close to the ear ; or to the burning of salt in the fire. The theories as to the production of the *true crepitant râle*, presuming that it originates within the lung, are that it is due to the opening-up of air-vesicles stuck together, to air passing through thick exudation in these vesicles, or to minute lacerations of lung-tissue. It must, however, be mentioned that some authorities regard this adventitious sound as pleuritic in its origin, being in reality a friction-sound, and I am bound to say that my own observations in cases of pneumonia which have come under my notice incline me to this view. 2. *Redux crepitant râle*. Heard in the advanced stage of acute pneumonia, when resolution is taking place, this differs from the râle just considered in that the crepitant sounds are much less abundant ; larger and of unequal size ; less dry ; and perceived during both inspiration and expiration. It gives the impression of air passing through a thick material, which is the pneumonic exudation undergoing a process of softening. A sound of similar characters is sometimes noticed in phthisis. 3. *A large dry crepitant râle* is said to be observed in some cases of emphysema, not abundant, and resembling the sound produced by inflating a bladder. It is supposed to be due to the opening-up of enlarged vesicles. 4. *Compression or collapse râle*. When the lung is compressed or collapsed from any cause, a râle consisting of a number of small dry crepitations may be slowly evolved at the close of, or immediately after a deep inspiration.

(C.) **Crackling or Clicking Râles.**—During the process of softening which the consolidation of phthisis undergoes, certain râles of a crackling character are developed, such as would be expected from the passage of air through such a material. These are named :—1. *Dry crackling or dry crepitation*, which consists of some three or four crackles or clicks,

sharp, abrupt, and dry, only heard usually during inspiration. It indicates the commencement of softening. 2. *Moist* or *humid crackling* or *crepitation*. Crackling in character, this is more abundant than the dry râle, though the crackles are not large or numerous, but they are more moist, as if air were passing through a less consistent substance, and occur both during inspiration and expiration, being most marked usually in the former. It accompanies more advanced softening, and is also met with in connection with small cavities. These râles are chiefly observed at the apices of the lungs.

(D). **Mucous, Submucous, and Subcrepitant Râles.**—These constitute a very common class of adventitious sounds, the characters of which are readily appreciated. They result from the transmission of air through fluid contained in the air-tubes or vesicles, and the varieties observed depend upon the nature and quantity of the fluid, and its exact situation. Generally they consist of a number of distinct sounds, usually *bubbling*, but they may have a *crackling*, *rattling*, or somewhat *gurgling* character. The individual sounds vary in size considerably, as well as in number and pitch. If they are of large or medium size, the râle is called *mucous*; if small, *submucous*; and if very minute, *subcrepitant*, because it then resembles a crepitant sound. These râles accompany both inspiration and expiration, being generally more marked during the former act, and they may be so abundant as completely to obscure the breath-sounds. A cough often greatly influences this class of râles, both as to their amount and site, sometimes removing them completely. They are most common and most marked towards the bases of the lungs, but may be heard universally over the chest. In children, and when they originate in the larger tubes, they are apt to have a somewhat metallic or hollow character. *Causes.* (i.) Bronchitis. (ii.) Œdema of the lungs. (iii.) Hæmorrhage into the bronchial tubes. (iv.) Rarely fluid from outside the lung emptying itself through the bronchi, such as pleuritic effusion.

(E). **Hollow Râles.**—The essential character of these abnormal sounds is that they are all more or less hollow in quality, and convey the impression of being originated in a cavity. For their production a hollow space must exist, containing fluid, through which air passes; the size and other conditions of the space will influence their degree of hollowness and their pitch, while the amount and consistence of the fluid will affect the abundance and actual quality of the râle. Hence it may be *bubbling*, *crackling*, or *gurgling*; very variable in size and amount; and subject to change considerably from time to time. According to their degree of hollowness and pitch these râles have been subdivided into such varieties as *cavernulous*, *cavernous*, *amphoric*, *ringing*, *metallic*, &c. They are heard generally during inspiration and expiration, but may be limited to either portion of the respiratory act. A cough often causes them to disappear, or renders them more distinct. Ordinary mucous râles occasionally assume a hollow character, if produced near a large cavity. The heart's action sometimes causes cavernous râles, by agitating the fluid in a contiguous cavity. Phthisis is the pulmonary disease in which these râles are chiefly observed, but they may also be noticed in connection with an abscess, or with dilated bronchi. When the pleura contains air and fluid, provided a communication with the lung exists below the level of the fluid, metallic or amphoric râles may be elicited during the act of breathing or coughing.



## C. FRICTION OR ATTRITION-SOUNDS.

By the rubbing together of the adjacent surfaces of either pleura, when this is the seat of certain morbid changes, adventitious sounds may be elicited during the act of breathing, usually termed *friction-sounds*. In examining for this class of physical signs, it may be requisite to investigate every part of the chest, but especially its lower portion laterally and behind, as they may be present over but a very small area; the patient must also be made to breathe deeply, otherwise the requisite rubbing together of the surfaces may not be brought about.

The following are the chief points relating to friction-sounds which need to be noticed:—1. **Characters.** As a rule friction-sounds are more or less *rubbing* in quality, varying from a slight *graze* to a loud *grating* sound. They may, however, be *creaking*, *crepitant*, *crackling*, *clicking*, or *rumbling*. In not a few cases a friction-sound closely resembles crepitant, crackling, or even small mucous râles, but is distinguished from these by being unaffected by a cough. *Superficialness* is a prominent character of all these sounds. 2. **Site and extent.** Friction is usually observed on one side, and towards the lower part of the chest, especially below the angle of the scapula, or in the infra-axillary region. It is generally limited in extent, sometimes not covering an area of more than an inch, but may be heard all over one side, or even over a great part of both sides. 3. **Intensity.** This varies from a scarcely perceptible rub, to a sound audible even at a distance from the chest. Generally it is moderately loud. 4. **Rhythm.** Friction is usually most perceptible during inspiration, but often accompanies expiration also; it may only be heard at the end of a deep inspiration. It is frequently irregular and jerky. 5. **Causes.** The pathological conditions in connection with the pleura which may give rise to friction-sounds are:—*a.* Dryness of the surface and increased vascularity, with prominence of the vessels, which may cause a slight grazing. *b.* Deposit of exudation and proliferation of cellular tissue associated with pleurisy, the characters of the sound depending upon the thickness and density of the material formed, and the amount of fluid mixed with it. *c.* Tubercular and cancerous deposits very rarely. It may be mentioned that a cirrhotic liver may originate a sound simulating friction.

## D. VOCAL AND CRY RESONANCE.

The chief points to be noticed with regard to the *vocal resonance* are:—1. Its intensity and degree of clearness. 2. Its quality and pitch. 3. The area over which it can be heard. The following are the deviations from the normal which may be met with:—

1. **The resonance may be more or less weakened to complete extinction, over a variable extent of surface.**—*Causes.* (i.) Air or fluid in the pleura, separating the lung from the chest-wall. (ii.) Very extensive or dense consolidation of the lung, such as cancer or some cases of phthisis. (iii.) Emphysema in many cases. (iv.) Intra-thoracic tumours; or enlarged abdominal organs encroaching upon the

chest. (v.) Obstruction of the main bronchi, and consequent pulmonary collapse.

2. It may be increased in intensity or clearness, this being often associated with alterations in quality and pitch. Four varieties of abnormal vocal resonance are recognized, which may be considered under this head, namely:—

(i.) *Bronchophony*.—This term merely implies an increase in the clearness of the vocal resonance, which, however, is generally intensified as well. It is commonly noticed in health in the interscapular regions, especially at their upper part; as well as frequently just below the inner end of the clavicles. *Causes.* *a.* Any lung-consolidation, provided it is not excessive, as in phthisis or pneumonia; in the latter the bronchophony has often peculiar characters, being *metallic* and *sniffing*. *b.* Small cavities in the lung, with thickening and consolidation around. *c.* Condensation of the lung in most cases. *d.* Occasionally a solid mass intervening between a main bronchus and the chest-wall, provided it is not too large.

(ii.) *Pectoriloquy*.—Here the voice seems to be conducted directly to the ear along the stethoscope, and the words uttered may often be distinctly recognized. The resonance is not uncommonly very intense, and gives a most unpleasant sensation to the listener. *Causes.* *a.* Certain cavities in the lungs in the great majority of cases. The conditions required are that the cavity is of good size, but not too large, and tolerably smooth; does not contain much fluid; has firm but not too thick walls; lies near or is adherent to the chest-walls; and has one or more bronchi communicating with it, so that air may enter. *b.* Very rarely a solid mass between the main air-tube or a large bronchus and the chest-wall. *c.* Occasionally pneumothorax under certain conditions.

A variety of pectoriloquy is described under the term *whispering pectoriloquy*, in which a whisper is clearly heard, and often the separate words can be distinguished. This, however, is not an alteration in the voice, but merely a modified expiratory sound. It is only observed in connection with large and superficial cavities, and is best heard in cases of pneumothorax, but is not infrequently associated with phthisical cavities.

(iii.) *Ægophony*.—The vocal resonance has a peculiar *bleating* or *nasal* quality in some cases of pleuritic effusion, to which the term *ægophony* has been applied. As a rule it is best heard about the angle of the scapula, but may vary with a change in the position of the patient. Some authorities are of opinion that ægophony is due to the presence of a thin layer of fluid between the lung and the chest-wall; others that it is caused by superficial compression of the lung.

(iv.) *Amphoric resonance*.—In certain large cavities with firm walls, the voice may have the characteristic hollow and metallic quality known as *amphoric*. This is only very rarely noticed in connection with large phthisical cavities, or in cases of pneumothorax.

3. The *area* over which vocal resonance can be heard will be influenced by the same conditions as those which affect the extent of the vocal fremitus.

## E. TUSSIVE RESONANCE.

The cough may be intensified in connection with consolidations and cavities, and may assume peculiar characters, hence named *bronchial*, *cavernous*, *metallic*, *amphoric*, &c., but these alterations do not add much to the knowledge gained by studying the vocal resonance, though the tussive resonance may be advantageously made use of where the voice is weak, as in females. The uses of cough in the investigation of disease, which can be best turned to practical account, are these:—

1. During the act adventitious sounds may be elicited, which are not heard during the mere act of breathing, especially in connection with cavities.
2. Various fluid substances which have accumulated in bronchial tubes or cavities may be dispersed and expelled, râles being thus done away with, and the breath-sound rendered louder, so that its characters may be better appreciated. Thus the act of coughing may be the means of distinguishing between friction-sounds and sounds produced within the lung; and in connection with cavities, cavernous or some other breath-sound may sometimes be heard after a cough, where respiration has been previously quite inaudible.

## F. PECULIAR SOUNDS HEARD IN CONNECTION WITH LARGE CAVITIES.

1. **Metallic tinkling.**—Resembling the sound produced by striking a glass vessel with a pin, this is a single, clear, high-pitched, ringing sound, heard in connection with large air-containing spaces, in which there is a little fluid. It is supposed to be occasioned by the bursting of a bubble, or the dropping of fluid from the top of the cavity; and may be originated during the act of breathing, speaking, or coughing, or, rarely, by the action of the heart. Phthisis and hydro- or pyo-pneumothorax are the diseases in which metallic tinkling occurs, though it is very rare.

2. **Amphoric echo.**—An echo of amphoric character may attend the respiratory sounds, voice, cough, rhonchi, the heart-sounds, or even the act of swallowing. For its production there must be a large cavity, having a smooth interior, and containing air, such as is met with in pneumothorax, and occasionally in phthisis.

3. **Bell-sound.**—In some cases of pneumothorax, when a coin is placed on the affected side and struck with another coin, a clear and ringing sound, resembling that elicited by striking a bell, may be heard on listening over another part of the same side.

## VI. SUCCUSSION-SIGNS.

Succussion is a mode of examination very seldom required, and merely shows the presence of air and fluid in a large space. The signs produced by shaking a patient are:—

1. **A splashing-sensation** felt by the hand.
2. **A splashing-sound.** They are occasionally present in hydro- or pyo-pneumothorax; and extremely rarely in connection with large phthisical cavities.



## CHAPTER V.

## ON RESPIRATORY SYMPTOMS.

IN this chapter the more important symptoms associated with the respiratory organs will be briefly considered.

## I. DYSPNŒA.—APNŒA.

DYSPNŒA or *difficulty of breathing* is a symptom requiring careful investigation, as it may be made up of several elements which it is important to distinguish; while it is also requisite to localize the cause of the disturbance, which is not necessarily situated in the respiratory organs.

ÆTIOLOGY.—The conditions which give rise to dyspnœa may be thus arranged:—1. *Some impediment to the entrance of air* through the air-passages, owing to internal obstruction; spasmodic constriction; organic stricture; or external pressure. This may exist in any part of the respiratory passages, from the mouth to the bronchi. 2. *Mechanical interference with the expansion of the chest-walls*, from pressure or rigidity; or with the movements of the *diaphragm*. 3. *Loss of the muscular inspiratory force*, owing to paralysis or spasm. 4. *Deficient expiratory force*, as the result of loss of elasticity of the lungs; or of rigidity of the chest-walls. 5. *Diminution in the working portion of the lungs*, from actual destruction; consolidation; liquid accumulation in the air-cells and minute bronchi; collapse or compression; or obliteration of the minute pulmonary vessels. 6. *Affections of the chest or abdomen which render the movements of respiration painful*. 7. *Improper conditions of the air inspired*, such as its being too rarefied, or containing irrespirable gases. 8. *Excess or deficiency of blood in the lungs*, due to some organic or functional derangement of the heart; obstruction in the pulmonary vessels; actual loss of blood; or violent exercise. 9. *Altered quality of the blood*, associated with anæmia; imperfect aëration; or the presence of poisonous elements in fevers, renal diseases, pyæmia, diabetes, and other affections. 10. *Nervous disturbance*, in connection with hysteria; strong emotion; cerebral disease; brain-poisoning; pressure on the vagus nerves or on some of their branches; or reflex disturbance.

CHARACTERS OF DYSPNŒA.—In studying any case in which there is apparent dyspnœa, it is requisite to observe the following points—1. Whether any subjective sensation of want of air is experienced, as well as its degree, which may amount to a feeling of impending suffocation. 2. The rapidity of the breathing. 3. Whether respiration is increased in depth and force, or the reverse. 4. If the relative lengths of inspiration, expiration, and the interval are disturbed. 5. If there are objective signs of great want of breath, indicated by the posture of the patient; by the extraordinary muscles of respiration being brought into play; by working of the *alæ nasi*; or by inability to hold the

breath or to speak. 6. Whether any noise accompanies the act of breathing, such as stridor or rattling. 7. If air enters freely into the lungs or not. Deficient entrance of air is indicated by more or less sinking-in of the lower part of the chest, the epigastrium, and the supra-sternal fossa during inspiration. 8. If there are any signs of imperfect blood-aëration, with their degree. 9. Whether the dyspnœa is constant, paroxysmal, or subject to exacerbations; and if paroxysms are traceable to any evident cause, such as effort, emotion, taking food, or inhalation of cold air.

**SIGNS OF APNŒA OR ASPHYXIA.**—The phenomena which accompany interference with the respiratory functions are due partly to overloading of the venous portion of the circulatory system, and deficient supply of blood to the arterial portion; but chiefly to the blood being imperfectly aërated, and therefore overcharged with carbonic acid, which acts as a poison, especially affecting the nerve-centres. At first unusual efforts are made to breathe, more or less violent according to the cause of the disturbance of respiration, but these diminish and finally cease, as the central nervous system becomes more and more disturbed. The face is turgid, at first being flushed, but soon becoming purple or livid; or under certain conditions it is deadly pale or mottled, accompanied with lividity about the lips, nose, and eyes. Other regions are also blue or livid, especially the nails and other parts distant from the centre of the circulation. Distension of the veins is observed; and the eyes tend to be prominent, suffused, and watery. The temperature becomes reduced, while cold clammy sweats break out. Nervous symptoms soon set in, namely, at first vertigo, disturbance of the special senses, mental confusion and wandering, twitchings and tremors; followed by drowsiness and stupor ending in complete coma, with convulsions and subsequently relaxation of the muscles, including the sphincters. The pulse is feeble, frequent, and small, but it continues to beat after respiration has stopped, and the heart may be still acting when the pulse has ceased to be perceptible; finally this organ also fails, should the termination be fatal.

*Post-mortem* examination reveals distension of the right side of the heart and of the veins with dark blood; with intense venous congestion and its consequences, affecting all the organs and tissues of the body.

**TREATMENT.**—The main indications applicable to the treatment of different cases of dyspnœa are :—1. To remove its cause, if possible. 2. To attend to the posture of the patient, adopting that in which breathing is most easily performed. 3. To avoid all kinds of effort; as well as other causes which are liable to bring on attacks of dyspnœa. 4. To assist the patient by mechanical means, when these are available to make up for impaired respiratory forces. 5. To see that the supply of air is sufficient; as well as that it is pure and otherwise suitable to the conditions of the patient. 6. To remove blood in certain cases, either generally or locally. 7. To administer, either internally or by means of inhalation or subcutaneous injection, remedies which tend to relieve dyspnœa, especially *depressants*, *anti-spasmodics*, or *stimulants*. 8. To employ *local measures*, such as the application of sinapisms, fomentations of different kinds, turpentine stupes, or free dry-cupping, over the chest. 9. To treat the asphyxial condition. For this purpose the most reliable means are the application of sinapisms over the chest and other parts; a warm bath, with free cold affusion over the head and shoulders while the patient is in the bath; slapping the chest with

a wet towel; artificial respiration, by Marshall Hall's, Sylvester's, or Howard's method; galvanism along the vagus nerves; and, when necessary, the performance of laryngotomy or tracheotomy.

## II. COUGH.

**ÆTIOLOGY.**—The causes of cough may be summed up under the following heads:—1. An *irritable condition of the mucous membrane* in some part of the air-passages, but especially in the throat and larynx. This is particularly observed in connection with inflammatory affections, the sensibility being then much exalted. 2. Some source of *direct irritation or discomfort* in the throat, larynx, trachea, or bronchi. This may be due to morbid conditions of normal structures, such as the uvula, tonsils, epiglottis, or vocal cords; to the presence of irritating particles or of larger foreign bodies introduced during inspiration; to certain conditions of the air inhaled, such as a very low temperature, or impregnation with irritating gases: or to the collection or secretion of morbid products, for example, serum, mucus, pus, blood, croupous or diphtheritic deposit. At the same time there is frequently increased irritability of the mucous surface. Cough from this cause may be voluntarily excited, but it is often involuntary and irrepressible, especially when the irritation is in the neighbourhood of the glottis. 3. *Reflex cough*. In many cases cough probably depends upon some more or less distant reflex irritation, but before deciding that such is its origin, it is always well to look carefully for some more immediate cause. It is believed that the source of irritation may be associated with the lungs or pleuræ; the heart or pericardium; the alimentary canal (dyspepsia, dentition, worms); the liver; the peritoneum; the ear; the female generative organs; or the external surface. 4. An *unhealthy condition of the blood*. This is supposed to give rise to cough by its effect on the nervous system, for instance, the state of the blood associated with gout or rheumatism. Most commonly, however, some local cause can be detected in these cases. 5. *Nervous disturbance*, in connection with hysteria, brain-disease, or direct irritation of the respiratory nerves.

**CHARACTERS OF COUGH.**—This symptom requires investigation with respect to the following particulars:—1. The frequency of its occurrence; and whether it is constant or paroxysmal. 2. The severity and duration of the fits. 3. The mode of onset, whether the cough is a voluntary act, or involuntary and irrepressible, preceded by a sense of irritation in any part; or if it is brought on by some obvious cause, such as exertion, change of posture, or inhalation of cold air. 4. Its particular quality, and the sounds which are produced during the act, both in inspiration and expiration. The chief varieties of cough are hacking, hoarse, wheezing, barking, ringing, metallic, croupy, whooping, crepitous, and aphonic. 5. Whether it is dry or attended with expectoration. In the latter case it is necessary to ascertain whether expectoration is effected easily or with difficulty; and also to make a personal examination of the *sputa*, observing:—*a*. Their quantity. *b*. General characters, as to colour; odour; whether in one mass or in separate lumps, with the size and shape of the latter; transparency or opacity; amount of frothiness; consistence, and degree of adhesiveness. *c*. If any special substances are evident to the naked eye, such as blood, fibri-



nous casts, or calcareous particles. *d.* Microscopic characters. *e.* Chemical composition in some cases. 6. If the cough terminates in vomiting; or is followed by relief of any previous unpleasant symptoms.

TREATMENT.—It is not always desirable to check cough, provided it is not excessive, and that it serves some useful purpose, in the way of getting rid of materials accumulating in the lungs or air-passages; indeed in some instances the act should rather be encouraged. In order to put a stop to, or to relieve cough, the chief indications are:—1. To instruct the patient voluntarily to suppress the act as much as possible. 2. To remove the cause of cough, if practicable; and to avoid everything which is likely to excite it. 3. To administer *sedatives* and other suitable remedies, which will be pointed out under the several diseases. 4. To employ *sedative inhalations*, which are very valuable in some cases. 5. To apply *local remedies* to the throat or larynx, when these parts are in a morbid condition. 6. To limit or check the formation of secretion or morbid products in the lungs or air-tubes, and thus prevent the necessity for the act of coughing; or to alter its characters, so that it may be more easily discharged. When a patient is obliged to cough, the act may often be assisted by mechanical means, such as by applying a bandage around the lower part of the chest and upper part of the abdomen; or making the patient hold on to some fixed object, such as a jack towel fixed on the bed-rail, if he should be confined to bed.

### III. HÆMOPTYSIS.

ÆTIOLOGY.—Hæmoptysis or *spitting of blood* signifies the discharge of blood through the mouth, from any part of the respiratory apparatus below the upper opening of the larynx. The sources of the blood, and the immediate causes of this symptom, may be thus classified:—1. *Hæmoptysis independent of obvious local disease.* In this group may be included hæmoptysis from going up a height, from severe straining or coughing, or from blowing wind-instruments, which is especially apt to occur in those who are delicate, and whose tissues are weak; vicarious hæmoptysis; that due to the inhalation of irritating substances, or to local injury; and that dependent upon an unhealthy state of the blood, such as scurvy or purpura. 2. *Diseases of the larynx, trachea, or bronchi,* for example, congestion; inflammation; ulceration; or morbid growths, especially cancer. 3. *Diseases of the lungs,* namely, phthisis, cancer, congestion, acute or chronic pneumonia, abscess, gangrene, hydatids. 4. *Mediastinal tumours* opening into the air-passages, including also glandular enlargements. 5. *Cardiac diseases,* namely, mitral disease; hypertrophy of the right ventricle; or a weak and dilated left ventricle. 6. *Disease of the pulmonary vessels.* 7. *Aneurism* opening into the air-passages. It must be remembered that blood sometimes enters the windpipe from the throat or nose, and is then expectorated.

The blood in hæmoptysis generally comes from the capillaries; but not infrequently a considerable branch of the pulmonary artery either gives way, or is perforated by erosion. In phthisis minute aneurisms have been found upon the branches of this vessel, which have ruptured, and thus originated large hæmorrhages.

There may be no immediate *exciting cause* of hæmoptysis; or it is brought on by exertion, coughing, or some other disturbance which affects the pulmonary circulation.

**SYMPTOMS.**—Hæmoptysis may come on without any warning; or is preceded by *premonitory* symptoms, such as weight or fulness about the chest, dyspnoea, a sense of heat, tickling in the throat, or a saltish taste. Usually the blood is brought up by coughing, but it may rise in gulps without any effort, or sometimes comes in a sudden gush, when it may even escape through the nose as well as through the mouth. Not infrequently vomiting is excited. The quantity of blood varies from a few streaks or a slight admixture in the sputa to an amount sufficient to cause instant death. The blood is generally bright and florid, and more or less frothy; but occasionally it is dark and non-aërated, especially when abundant and suddenly discharged. Clots may be observed, but the greater portion is usually liquid. No change in the blood is evident as a rule, either as regards its general or microscopic characters. The duration of an attack of hæmoptysis varies much, but after the more urgent symptoms have subsided, the sputa are generally tinged for some time, or fragments of dark clot may be discharged. Recurrence is a frequent event, and in some cases spitting of blood occurs periodically.

If the blood comes from either lung in any quantity, *râles* of a moist character are generally heard over the corresponding part of the chest.

The effects on the general system will depend upon the amount of blood lost; and the rapidity and duration of its discharge. Death does not often result immediately from hæmoptysis, but may happen either from the direct loss of blood, or from its accumulation in the air-passages causing suffocation. Frequently some degree of febrile excitement is associated with hæmoptysis, the pulse being full and bounding, but soft. Should any blood remain in the lungs, it is liable to set up inflammation, and there can be no doubt that phthisis is originated in this way.

**DIAGNOSIS.**—Hæmoptysis may be simulated by bleeding from the mouth or throat; or by epistaxis. The quantity and characters of the blood discharged; the mode of ejection; and, above all, thorough examination of the nose, mouth, and fauces, as well as of the chest, will generally indicate the source of the bleeding. The part of the respiratory apparatus from which the blood escapes can also usually be ascertained by physical examination; and by observing the local symptoms present. Erosion of a large branch of the pulmonary artery is characterized by the discharge of a quantity of dark blood. The diagnosis between hæmoptysis and hæmatemesis will be pointed out under the latter. The cause of hæmoptysis can only be determined by a thorough investigation and consideration of each individual case.

**TREATMENT.**—Hæmoptysis must be treated on the principles applicable to hæmorrhages in general, but its management will necessarily vary according to its cause. Ordinarily, when the blood escapes from the lungs, the chief indications which require attention are to keep the patient entirely at rest, in a cool room, in the recumbent posture, with the head rather high; to subdue cough as much as possible; to give ice to suck freely; and to administer *astringents*, with *vascular sedatives*. Gallic acid in full doses with opium every two or three hours; pyrogallac acid; acetate of lead and opium; dilute sulphuric acid with alum; turpentine, internally or by inhalation; ergot; and hamamelis, are amongst the most useful remedies. The subcutaneous injection of ergotine, ergotinine, or sclerotic acid has of late years deservedly come into much repute in the treatment of hæmoptysis. Digitalis is of great value if the heart is acting excitedly. *Saline aperients* are

useful in plethoric patients. Some practitioners have recourse to venesection or local abstraction of blood, but this is rarely a desirable plan of treatment. The application of ice to the chest is often exceedingly beneficial, but it must be done carefully, the ice being removed by degrees. Dry-cupping over the chest is serviceable in some cases. It is sometimes useful, when a case does not yield to ordinary treatment, to draw the blood towards the limbs by means of hot foot-baths or a Junod's boot; or to apply ligatures around the extremities, should the patient be sinking from loss of blood, so as to confine the blood to the head and trunk. In extreme cases it might be necessary to have recourse to transfusion of blood. In hæmorrhage vicarious of menstruation, or due to stoppage of bleeding from piles, the application of leeches to the lower extremities, or around the anus, is said to do good. In all cases of hæmoptysis to any considerable amount, it is important to keep the patient under observation until any irritation due to the presence of blood in the lungs has entirely subsided. If there is any tendency to spitting of blood, everything likely to bring on an attack must be avoided; while at the same time the condition of the blood is improved by proper dieting, and by the administration of tincture of steel.

---

## CHAPTER VI.

### CATARRH—CORYZA—A COLD.

BEFORE considering the diseases affecting the several parts of the respiratory apparatus, a brief description will be given of the condition which frequently results from taking cold in some way or other, and which has been specially designated *catarrh* or *a cold*. The entire system is affected, but the more characteristic phenomena are due to a catarrhal inflammation, involving mainly the conjunctivæ, and the mucous lining of the upper air-passages, namely, the nose and its communicating sinuses, the throat, and the larynx and trachea. Not uncommonly it extends into the bronchi to a variable extent; and sometimes the mouth, œsophagus, stomach, or intestines are implicated. The complaint most commonly results from exposure to wet and cold, and many persons are liable to repeated attacks, which come on whenever the weather is unfavourable, and especially when it suddenly becomes damp and cold. The phenomena very much resemble those which are observed in influenza, and in the early stage of measles; as well as those which arise in some persons from inhaling the emanations from hay, ipecacuanha powder, and certain other vegetable products, or from the action of iodine upon the system.

**SYMPTOMS.**—These are both *general* and *local*, and the two classes are observed more or less simultaneously. The patient feels chilly and out-of-sorts, indisposed for any occupation or mental effort, languid and tired, and has a sensation of general aching or soreness of the limbs and body. More or less pyrexia supervenes, and the temperature may become raised to 100°, 101°, 102°, or even higher. This is accompanied with a somewhat frequent pulse, usually a dry skin, furred tongue, loss



of appetite, constipation, and concentrated high-coloured urine, which deposits urates on standing. Some persons feel exceedingly weak and depressed when suffering from catarrh, and especially if they have been previously debilitated. The *local* symptoms are in accordance with the parts involved in the catarrhal condition. At the outset various subjective sensations are experienced, namely, headache, especially over the forehead and temples, with a feeling of weight and heaviness; pains about the face, chiefly of a neuralgic character, which may be accompanied with tenderness; smarting of the eyelids, and aching in the eye-balls; dryness and heat of the nares; soreness of the throat; and not uncommonly pain and stiffness in the neck. Very soon the eyes become red and begin to water freely; while a running from the nose sets in, which is specially termed *coryza*, the discharge being at first thin and watery, as well as very irritating, so that it makes the parts over which it flows red and sore. Sneezing is often a troublesome symptom. On examination the throat is seen to be more or less red and swollen, and swallowing is painful; while the catarrhal condition of the air-tube gives rise to hoarseness or loss of voice, cough, and pain in speaking or coughing felt in the course of the larynx or trachea. If it extends into the bronchi, the patient experiences a sense of oppression or tightness across the chest, with more marked cough, wheezing, and the other signs of bronchial catarrh. In many cases partial deafness is noticed, owing to implication of the Eustachian tubes; and the senses of taste and smell are generally impaired or lost. Should the alimentary canal be involved, this may be indicated by soreness along the œsophagus during deglutition; pain and tenderness over the epigastrium; complete anorexia; nausea or vomiting; and diarrhœa. Occasionally a slight degree of jaundice is observed.

Catarrh assumes different degrees of intensity, but the symptoms generally increase in severity for two or three days, and then gradually diminish. The nasal discharge becomes more abundant, being often very profuse, and it alters in its characters, assuming a mucous or muco-purulent appearance. Not uncommonly the mouth becomes sore, and herpes appears about the lips; the nostrils are also often a little ulcerated. The patient generally feels worse during the night, when the painful sensations increase, and consequently sleep is liable to be much disturbed. Usually complete recovery is established in a few days, once convalescence sets in; but in some instances certain symptoms remain for a variable period, especially general debility, loss of appetite, cough, or impairment of the sense of smell or taste. Moreover, some more serious affection may be set up, particularly bronchitis or other disease of the respiratory organs. A simple cold is especially in danger of leading to these results in very young or old persons, in those who are constitutionally feeble and delicate, or in those who are depressed by excessive mental or bodily work.

DIAGNOSIS.—The symptoms of catarrh are readily recognized, but care must be exercised in discriminating between those cases which are simply due to a cold, and those in which the phenomena are associated with some other condition, especially with measles or epidemic influenza.

PROGNOSIS is generally favourable, and the circumstances which render catarrh more than usually serious have already been indicated. It must be remembered that attacks of this complaint are very liable to be repeated, and even to become habitual at certain seasons.

**TREATMENT.**—It is the safest plan for any one who is suffering from a cold to stay in bed for a day or two at the outset, if this is practicable, or at any rate to stop indoors, in a warm and comfortable room. Undoubtedly its effects may often be alleviated or prevented by immediately exciting free sweating. For this purpose a warm bath or a foot-bath is useful, the patient going immediately afterwards to bed, and having some hot drink, such as gruel, wine-negus, spirit and water, or one of the other drinks ordinarily employed for this purpose. Certain medicines are also useful, of which the most efficient are a saline draught with spirits of nitre; or a full dose of Dover's powder. A vapour, hot-air, or Turkish bath often proves of signal service in checking a cold at its commencement. Local applications have been much employed for this purpose, especially in the form of dry inhalations of iodine and other agents; and of combinations made into snuffs, which are sniffed up into the nostrils. Ferrier recommends a snuff composed of hydrochlorate of morphia gr. ij, nitrate of bismuth 3 vj, and powdered acacia, 3 ij, of which from a quarter to a half may be used in the twenty-four hours. It has been affirmed that the prolonged mastication and swallowing of a dried leaf or two of the eucalyptus globulus has proved very efficacious in curing a cold.

If the complaint does not yield at the commencement, the patient should certainly remain in bed, and be kept on a light diet. Some practitioners lay special stress on limiting the amount of liquids given, or even withholding them altogether. With regard to medicines, these must be varied according to circumstances. Generally it is sufficient to open the bowels, and to give a *saline* mixture, to which a few drops of ipecacuanha wine may be added if the air-passages are much affected, as well as some *sedative* to relieve cough if this is excessive. Should there be much sickness, effervescent are useful; and it may become necessary to administer remedies for the purpose of checking diarrhoea. Some *narcotic* may be required at night, in order to procure sleep and relieve pain. If there is much debility and depression, quinine is a valuable medicine. As the patient tends towards convalescence, the diet should be improved by degrees, and a little wine may be given with advantage at this time. Complications must be watched for, and treated according to their nature. During convalescence *tonics* are very useful. If recovery is retarded, as well as in cases where attacks of catarrh are of frequent occurrence, a change of air to some genial district or climate is of the utmost benefit.

---

## CHAPTER VII.

### DISEASES OF THE LARYNX AND TRACHEA.

#### I. ACUTE CONGESTION AND INFLAMMATION.—ACUTE LARYNGITIS AND TRACHEITIS.

THE cases of acute congestion or inflammation affecting the main air-tube may be arranged into three main groups, namely:—1. **Catarrhal**, which includes congestion and various degrees of catarrhal inflammation of the mucous membrane. 2. **Cedematous**, where there is con-



siderable sub-mucous œdema. 3. **Croupous, diphtheritic, or membranous**, in which a fibrinous deposit, or so-called *false membrane* forms upon the surface. Before discussing this class of diseases, it will be well to offer a word of explanation regarding the terms *croup* and *croupous*. Without entering into any discussion as to its etymology, it may be stated that the word *croup* was originally employed to indicate merely a particular symptom, namely, *stridulous breathing*. Subsequently it came to be recognized as the name of a supposed disease in children which caused this symptom, and as it was at one time believed that such disease was invariably *membranous laryngitis*, the term *croupous* afterwards acquired a pathological signification, becoming associated with exudations or deposits of a fibrinous character. Then, as knowledge advanced, and it was ascertained that croupy symptoms might arise from other morbid conditions, the form of laryngitis which is characterized by the deposit of fibrinous matter was specially named *croupous*, and the term was afterwards extended to croupous pneumonia and other diseases in which this kind of exudation forms. Moreover, when it was found that the phenomena of croup might be due to laryngeal spasm, this was named by French writers *false croup*, as distinguished from *true croup*, in which the symptoms were due to inflammation, and later writers have made further divisions, according to their views as to the nature of the morbid conditions upon which croupy symptoms might depend. At present the word *croup* is often employed very indefinitely, as the designation of a group of cases occurring in children, which are characterized by laryngeal obstruction with consequent stridulous breathing and other phenomena, but it must be remembered that these may be due to either of the varieties of laryngitis already mentioned, as well as to laryngeal spasm or so-called *laryngismus stridulus*. It would be well, in accordance with the suggestion of the Committee of the Medico-Chirurgical Society, to use the term merely as a clinical definition, implying laryngeal obstruction with febrile symptoms, occurring in children.

**ÆTIOLOGY.—Predisposing causes.** The different forms of acute inflammation of the larynx and trachea are all predisposed to by certain conditions, namely, a lax, weakly, and ill-nourished state of the system; effeminate habits; immoderate wrapping-up of the neck; previous attacks, especially if repeated; and a climate or season characterized by a cold moist atmosphere, cold winds, or rapid changes of temperature, the complaint being therefore more prevalent during winter and spring. Males suffer more than females. As regards age, simple *laryngeal catarrh* is most common among adults; but the more intense forms of the disease are chiefly confined to the period of childhood, especially from the first to the seventh year. Unfavourable sanitary conditions predispose to croup, and children of the poorer classes chiefly suffer from this complaint.

**Exciting causes.**—These may be arranged as follows:—1. *Direct irritation*, from breathing very cold or hot air, steam, acrid vapours, or air containing irritating particles; or from swallowing boiling or corrosive liquids. 2. *Excessive coughing*; or *violent exercise of the voice*, in speaking, shouting, and singing. 3. *Morbid conditions* affecting the larynx or trachea, such as ulcers or growths. 4. *Local injuries or operations*. 5. *Direct exposure* of the front of the neck to a cold draught. 6. *General exposure to cold*, the larynx being either involved alone, or



along with other mucous surfaces. 7. *Extension* of inflammation from the nose or pharynx; or occasionally from the bronchi. 8. Certain *febrile conditions*, in which laryngitis occurs as a complication, especially influenza, erysipelas, measles, and typhus fever. 9. *Secondary syphilis*.

**Special ætiology.**—The ætiology of certain special forms of laryngitis demands separate notice. *Edematous* laryngitis generally follows some previous laryngeal disease, but may be due to mere cold; it is also particularly liable to arise from swallowing boiling liquids. This usually happens among the children of the poor, who are in the habit of drinking out of the spout of the tea-kettle. Laryngeal inflammation complicating erysipelas is likewise prone to be attended with œdema.

The pathology of *croupous* laryngitis has of late years been much discussed, and is still a disputed question. French writers have always regarded it as being identical with *laryngeal diphtheria*, and therefore as always resulting from contagion or from unfavourable hygienic conditions; this is the view also now maintained by the principal authorities in this country. The common belief, however, is that there is a distinct form of croupous laryngitis, which is entirely independent of diphtheria, and results from causes which affect the larynx locally, and especially exposure to cold, particularly to northerly or north-easterly winds, or a general chill of the body. The subject has been investigated by the Medico-Chirurgical Society, and without giving at any length the conclusions arrived at, it must suffice to state that the large majority of cases of membranous laryngitis and tracheitis were clearly proved to be of a diphtheritic nature, but in exceptional instances this condition was traceable to other causes acting locally, namely, exposure to cold, the inhalation of hot water or steam, the contact of acids, the presence of a foreign body in the larynx, and a cut-throat. The complaint also occurred occasionally as an accompaniment of measles, scarlatina, or typhoid fever, independent of any ascertained exposure to the special diphtheritic infection. As a complication this form of laryngitis has likewise been described in connection with small-pox, typhus fever, and erysipelas. It was, however, found that the majority of cases exhibiting croupal symptoms definitely traceable to cold, were of the nature of laryngeal catarrh.

**ANATOMICAL CHARACTERS.**—The appearances in laryngitis and tracheitis will differ according to the nature and products of the morbid changes. The mucous membrane may be only more or less congested. In *catarrhal* laryngitis bright redness is usually observed, with swelling, opacity, and slight softening of the membrane. Spots of epithelial erosion are common, but actual ulceration is very rare. After a time secretion forms, which is sometimes very viscid, containing an abundance of young cells. *Edematous* laryngitis is characterized by the accumulation of serum in the sub-mucous tissue, especially where this tissue is very lax, which gives rise to much translucent swelling, with a sodden, flabby condition of the structures, but the redness is less marked. Very rarely sero-purulent or purulent fluid infiltrates the tissues. In low fevers gangrene may occur.

The special anatomical character of *croupous* or *plastic* laryngitis is that the surface of the mucous membrane becomes covered to a variable extent with croupous exudation or so-called *false membrane*, the epithelium being destroyed. In appearance and structure this cannot be distinguished from diphtheritic deposit. It may be limited to a small portion of the surface, or to separate patches; or may cover the whole

of the larynx and trachea, occasionally extending even into the smaller bronchi. Its main seat is said to be the trachea. In thickness the deposit may range from a mere film to two or three lines or more. The consistence varies, but the material is generally tolerably tough, compact, and adherent for a time, finally becoming detached. Its under-surface frequently presents little points of extravasated blood. A fresh deposit not uncommonly forms after the membrane is separated, and this may be repeated several times. Under the microscope it is seen to consist of an amorphous or finely-fibrillated substance, in which abundant young cells are involved. There is very little swelling of the mucous membrane as a rule; and its structure is not at all affected.

When death results from acute laryngitis, especially the membranous variety, some of the following morbid conditions are usually present, namely, bronchitis; congestion and cedema of the lungs; lobular or lobar pneumonia; localized insufflation and collapse; distension of the heart and venous system with dark blood; congestion of the organs generally; and slight serous effusions. The lymphatic glands which are situated beneath the thyroid body on each side of the trachea may be enlarged.

**SYMPTOMS.**—The clinical history of acute inflammation of the main air-tube differs in the several varieties of this disease, and therefore each form calls for separate consideration.

**1. Acute Catarrhal Laryngitis and Tracheitis. Laryngeal and Tracheal Catarrh. Cynanche Laryngea.**—This variety assumes very different degrees of severity, but the following are the usual symptoms observed in adults:—Unpleasant sensations are experienced over the windpipe, such as dryness, roughness, constriction, soreness, burning, or tickling, which are increased by coughing or speaking. These are chiefly felt when the upper part of the larynx is involved. Swallowing is often rather painful. The voice is husky, and frequently hoarse or cracked, or it may become quite lost. Cough is in many cases a prominent symptom, there being a constant tendency to paroxysmal attacks. It is harsh and hoarse in quality; or may become completely aphonic. The patient is also often disposed to hawk repeatedly. At first there is no expectoration, but after a while a little clear viscid mucus containing young cells may be expelled with difficulty, this finally becoming more abundant and muco-purulent. Respiration is not much interfered with in ordinary cases. In some cases of catarrhal laryngitis there are no general symptoms, but usually more or less pyrexia is observed.

Catarrhal laryngitis occurring in young children is liable to be attended with far more serious symptoms, owing to the small size of the air-tube in such subjects; to the edges of the glottis becoming glued together by viscid secretion; and to the tendency to laryngeal spasm. There can be no doubt but that this is the condition present in a large proportion of the cases designated *croup*, constituting what has been termed *stridulous laryngitis* or *inflammatory croup*. Therefore it will be expedient to describe here the phenomena characteristic of this class of diseases as they occur in children.

An attack of *idiopathic* or *primary* croup is frequently preceded by some *premonitory* symptoms for a day or two, such as slight harsh cough, hoarseness, and sore-throat; with a little pyrexia and constitutional disturbance. In some cases, however, the complaint comes on suddenly, and without any previous warning. An attack of croup usually sets in



during the night, while the child is asleep; and when established, the symptoms are very characteristic.

*Local.*—The voice is at first harsh and hoarse, or at times cracked and shrill, finally becoming whispering or being completely lost. Paroxysms of spasmodic “croupy” cough come on, short, sharp, and abrupt in character; dry; of high pitch; and of somewhat metallic, clanging, or brassy quality. The cough is interrupted by a shrill, ringing, whistling, or “crowing” inspiration. Soon it becomes husky and muffled, and at last loses all sound. Respiration is greatly impeded, and presents some peculiar characters. The act is exceedingly laboured, and attended with violent effort; it is also prolonged, and therefore not frequent; while inspiration is accompanied with a high-pitched, metallic, sibilant, or wheezing stridor, which may be heard at some distance off.

These symptoms are not persistent, there being intervals of complete or comparative ease, especially during the day. In severe cases, however, or in the advanced stage of those tending towards a fatal issue, there may be scarcely any remissions. The paroxysms of dyspnoea are commonly believed to be partly caused by spasm of the muscles, but Niemeyer maintained that these are paralyzed.

The child often grasps its throat, or puts its hand to its mouth, as if attempting to remove some obstruction, and if old enough may complain of local pain. A little thick viscid mucus is sometimes brought up by coughing. Deglutition is difficult in some cases.

*General.*—At first the general symptoms are of a febrile character, and the temperature may rise to 102°, 103°, or more; the pulse being frequent, full, and hard. Subsequently, however, the pyrexia abates; and in severe cases the prominent signs are those associated with imperfect aëration of blood, which are intensified during the paroxysms of dyspnoea. Ultimately in fatal cases gradual or rapid suffocation ensues, attended with the phenomena of apnoea. Pulmonary complications frequently arise, and the danger is thus increased. A good many cases recover, however, if there has only been catarrhal inflammation.

**2. Œdematous Laryngitis.**—Œdema may supervene very rapidly, and in the course of an apparently slight attack of laryngeal catarrh. It is a highly dangerous condition, and if not relieved, may cause speedy suffocation. The characteristic features of Œdematous laryngitis are a sensation of the presence of a foreign body in the larynx; more marked dysphagia than in the catarrhal form; and urgent dyspnoea of laryngeal characters, inspiration being whistling or hissing, but expiration being comparatively or quite easy. Voice is completely lost; and cough becomes also aphonic. It is usually believed that spasm of the glottis aids in the causation of dyspnoea in cases of Œdematous laryngitis; but some authorities are of opinion that the muscles are paralyzed.

The general symptoms are indicative of more or less grave interference with the respiratory functions, and consequent deficient blood-aëration.

**3. Croupous, Plastic, or Membranous Laryngitis. Membranous Croup. True Croup. Cynanche Trachealis.**—It is scarcely practicable or necessary to give a distinct clinical history of this form of laryngitis. In the large majority of cases it is that already described as belonging to *laryngeal diphtheria*, and in most instances, but not always, diphtheritic deposits may be observed on the throat, the laryngeal condition being either primary or secondary. This disease may occur in adults. When it affects children, or when they are the subjects of membranous laryngitis from any other cause, the symptoms



are similar to those described as characteristic of inflammatory croup, but they tend to be more urgent, and the termination is very frequently fatal. Flakes, or larger fragments, or even casts of membranous deposit may be expectorated, and this may be followed by temporary or permanent relief. *Secondary* croup, setting in in the course of febrile exanthemata, will be revealed by more or less severe symptoms indicative of laryngeal obstruction.

PHYSICAL EXAMINATION.—1. *Examination of the throat*, by inspection and the use of the finger, may reveal the condition of the epiglottis, should it be red and swollen; and also the presence of œdema. 2. *Auscultation* over the windpipe may detect changes in the laryngeal breath-sound, or local mucous râles; or sometimes in membranous croup a peculiar rhonchus, named *tremblement*, is heard during inspiration and expiration, supposed to be due to the flapping of a piece of false membrane; probably this sound can be produced by thick mucus. 3. The *laryngoscope* is often inapplicable, especially if the epiglottis is much affected, and also in cases of true croup; when it can be employed, it might reveal bright redness, turgidity, swelling, œdema, or alteration in shape of various parts; or thick secretion or membranous deposit might be visible. 4. *Examination of the chest* may indicate more or less interference with the entrance of air into the lungs, particularly in cases of œdematous or croupous laryngitis, and especially when children are affected. The pulmonary sound may be obscured by a loud laryngeal sound; and mucous râles are sometimes heard over the chest. Pulmonary complications give rise to their own special physical signs.

COURSE, DURATION, AND TERMINATIONS.—The course and duration of simple laryngeal catarrh vary considerably. Recovery is the usual termination, but the affection is very liable to recur, or it may become chronic; in young children it also may prove fatal. Œdematous laryngitis is often fatal, and death may take place very suddenly or with great rapidity. Croupous laryngitis in children usually runs a remittent course, exacerbations occurring during the night; occasionally it progresses continuously. Some cases terminate within twenty-four hours, and most end within five days; the duration may, however, be prolonged for ten days or a fortnight. The clinical terminations are in death or recovery. Death generally results from apnoea, but may be due to asthenia. Recovery is indicated by the subsidence of the local and general symptoms; at the same time the cough becoming looser, with more abundant muco-purulent expectoration, or sometimes a quantity of exudation being expelled, if there has been a membranous deposit.

DIAGNOSIS.—The diagnosis between affections of the throat and those of the larynx has already been pointed out when describing the former. In children the different forms of acute laryngitis have to be distinguished from whooping-cough; from bronchial catarrh with a tendency to spasm; as well as from other conditions affecting the larynx itself, namely, laryngismus stridulus; acute exacerbations in connection with chronic diseases, such as morbid growths; and conditions resulting from the presence of foreign bodies, or from external injury. Only *laryngismus* can be specially noticed here, but it is necessary to insist upon the importance of carrying out a thorough physical examination in all cases. *Laryngismus stridulus* is more sudden as regards the onset and cessation of the paroxysms of dyspnoea;

and these are often brought on by some obvious cause. A history of previous similar attacks can generally be obtained. There may be signs of general convulsions, with turning-in of the thumbs. The characteristic cough of croup is not present, but the child often cries. There is complete restoration between the paroxysms. Pyrexia is absent.

It is necessary to distinguish between the different forms of laryngitis. *Laryngeal catarrh* is most common in adults; it has less severe symptoms, and no croupous cough, but more abundant expectoration; there is but little pyrexia; and the complaint is often accompanied with much nasal catarrh. *Edematous* laryngitis is rare in children, except as the result of drinking boiling water. It generally supervenes upon some previous disease of the larynx. Expiration is comparatively easy. Cough soon becomes completely aphonic; and the voice is also lost. The edematous parts may be seen or felt. It is by no means easy in many cases to recognize the nature of an *inflammatory croupy attack* in children. *Catarrhal* laryngitis may, however, be often distinguished by the fact that the patient is subject to similar attacks in cold seasons; while the symptoms are less severe and more remittent; and the termination is not often fatal. *Diphtheria* involving the larynx may be generally diagnosed from other forms of laryngitis by the following characters:—

1. The complaint may be epidemic, or a history of contagion may be traced.
2. Premonitory general illness may have been felt for some days, with a tendency to asthenia.
3. Marked throat-symptoms are usually present, preceding the laryngeal symptoms.
4. The glands about the jaws are enlarged.
5. Epistaxis and albuminuria frequently occur.
6. Examination of the throat reveals the diphtheritic deposit.

When laryngeal diphtheria is primary, and the throat is free from disease, the diagnosis becomes very difficult if no history of diphtheria can be obtained. Croupous laryngitis arising from other causes it would be almost impossible to recognize positively, unless false membrane were expelled. Laryngoscopic examination might afford some aid in the diagnosis of the several forms of laryngeal inflammation.

**PROGNOSIS.**—All forms of laryngitis are to be regarded as serious. The *edematous* variety is very grave, especially that form which is due to the swallowing of boiling liquids. *Membranous croup* is a most fatal disease. The prognosis is worse in young infants; and there is more danger in proportion to the severity and persistence of the local symptoms, the degree of interference with respiration, and the signs of deficient blood-aëration. Early treatment gives a better chance of recovery.

**TREATMENT.**—There are certain *general* matters attention to which is essential in the treatment of all the varieties of acute laryngeal inflammation. The patient should remain in a warm room, the temperature being kept uniformly at 65° or higher, and the air rendered moist by means of steam. In the case of children it is advisable to make a tent over the bed, as described under diphtheria, and it may be necessary to raise the temperature of the air considerably. All exposure must be avoided, the throat and chest being warmly covered. Rest to the larynx is imperative, so far as it can be obtained: and in many cases it is requisite to urge patients to restrain cough as much as possible.

In the case of adults the assiduous employment of inhalations of steam is one of the chief measures to be adopted, and Dr. Morell-Mackenzie recommends the addition of some tincture of benzoin, hop,



or conium juice to the boiling water, or a few drops of chloroform occasionally, should there be much tendency to spasm. If the attack results from a cold, it is desirable to excite diaphoresis by means of warm drinks, a hot or vapour-bath, and external warmth. The diligent employment of hot applications over the front of the neck is useful, such as a sponge dipped in boiling water and then squeezed dry. Some practitioners prefer cold applications. If the inflammation is advancing, and especially if it has spread from the throat, it is recommended to apply directly and efficiently a solution of some *astringent*, either with a brush or sponge, or by the aid of an atomizer. Nitrate of silver, alum, tincture of iron, chloride of zinc, and tannin are the substances chiefly employed in this manner. In some severe cases an *emetic* of sulphate of zinc or tartar emetic at the outset is decidedly useful, but the practice formerly adopted of giving repeated depressing emetics, combined with bleeding, blistering, and the administration of calomel, is most objectionable. Occasionally it might be advisable in severe cases to apply a few leeches over the upper part of the sternum.

The bowels should be kept freely opened; and a *diaphoretic saline* mixture may be given. If there is much distressing cough, some *sedative* may be administered with due precautions, such as a few drops of compound tincture of camphor or liquor morphiae.

The treatment of children must be similar, whatever the nature of the inflammation may be, and it is necessary to be exceedingly prompt in attending to the first indications of anything wrong in connection with the windpipe in such subjects. A warm bath should be employed immediately, the patient being then dried and wrapped up in blankets, while hot sponges are constantly applied over the larynx. The persistent use of cold compresses has been advocated, and is certainly deserving of more extended trial. If the symptoms are at all severe, unquestionably great relief often follows the action of an *emetic*, though Niemeyer affirmed that "they are only indicated where obstructing croup-membranes play a part in producing the dyspnoea, and when the child's efforts at coughing are insufficient to expel them." Tartar emetic and ipecacuanha are preferred by many practitioners, and may be given to robust children; but sulphate of zinc answers best in weakly subjects.

*Bleeding* has been extensively practised in the treatment of croup, but it should not be had recourse to as a mere routine measure. Leeches are often of great service in the case of healthy, plethoric children, when applied at an early period, especially if there are signs of local blood-stasis. They are best applied over the upper piece of the sternum, the number being regulated according to circumstances. Notwithstanding the high authorities by which the regular administration of calomel is supported, I have never seen any good result from it in this disease, but have more than once known it to do a great deal of harm, and therefore cannot but express my decided objection to the repeated employment of this drug. It may be advantageous to give one dose as a purgative at the commencement. The bowels should be kept freely open, and for this purpose enemata answer best. It is difficult to determine what internal remedies are most serviceable in the treatment of laryngeal inflammation in children, but it has appeared to me that most benefit is derived at first from *salines* combined with small doses of tartar emetic or ipecacuanha wine. The cough must be rather encouraged than checked, unless it is unduly severe, and there-



fore sedatives are generally contra-indicated. Later on *stimulant expectorants* are required, such as ammonia with chloric ether and syrup of squills. Some practitioners recommend the administration of alkaline carbonates, or chlorate of potash. Dr. Ringer strongly advocates the use of drop doses of tincture of aconite at short intervals.

*Counter-irritation* is not attended with much benefit in croup, but Dr. William Squire considers the application of tincture of iodine to the sides of the neck of some service, especially if covered with water-dressing. Attention to diet is often of much importance. At first the food should consist chiefly of milk, with cooling drinks, but nourishing soups or meat-juices are required when the system shows any signs of failure. Alcoholic stimulants are not usually needed unless some pulmonary complication supervenes. If nourishment cannot be taken by the mouth, it may be necessary to have recourse to nutrient enemata.

The question of the performance of *tracheotomy* is in many cases one of the greatest moment. It seems to me that if the symptoms increase in gravity in spite of treatment, and if signs of apnoea set in, the operation ought to be performed without delay, as affording the only chance of recovery, due care being taken both in its performance, and in the subsequent treatment.

For *œdematous* laryngitis the most effectual remedies are *emetics* which act with rapidity; the constant sucking of fragments of ice; and efficient scarification. In case of need tracheotomy must be resorted to.

*Secondary croup* calls for the administration of *stimulants*, along with abundant nutriment. Tincture of steel and mineral acids are the most efficient medicines in this affection.

*Complications* must be treated as they arise. Signs of apnoea must be combated by the usual measures. It is necessary in cases of recovery to exercise great care during convalescence. As prophylactic measures in those subject to croupy attacks, cold douching of the throat and chest, with dry friction afterwards; the wearing of suitable warm clothing; and the avoidance of cold damp winds and night air, are the chief things to be attended to.

## II. CHRONIC LARYNGEAL CONGESTION.—CHRONIC LARYNGITIS.— CHRONIC LARYNGEAL CATARRH.

**ÆTIOLOGY.**—This class of affections may remain as a sequel of acute laryngitis, but their chief causes are:—1. *Excessive use of the voice*, as in speaking, shouting, or singing. A form of chronic laryngeal catarrh constitutes the chief morbid condition in *clergyman's sore-throat*. 2. *Phthisis* and *syphilis*. 3. *Irritation extending from the throat*. 4. Some *local irritation* in the larynx, especially from ulceration or morbid growths; and also *external pressure* upon this tube. 5. *Irritation of the recurrent nerve*. 6. *Habitual inhalation of irritant particles*. 7. *Chronic alcoholism*. 8. *Excessive smoking*. 9. Occasionally *general plethora*; or a peculiar *constitutional condition*, attended with a disposition to chronic catarrh of mucous surfaces.

**ANATOMICAL CHARACTERS.**—These differ according to the duration, seat, and extent of the affection; and the variety which it assumes. More or less hyperæmia is generally observed, and the vessels may be evidently enlarged, especially in the form known as *phlebectasis laryngea*. The mucous membrane tends to become thickened and firm,

as well as the submucous tissue, particularly in phthisis and syphilis. Sometimes a state of chronic œdema is present. The surface is either dry and shining, or presents small collections of mucus, or more abundant secretion. In *glandular* or *follicular* laryngitis, which is the condition observed in *clergyman's sore-throat*, the morbid changes are chiefly confined to the racemose glands of the larynx, these being enlarged and red. The canal of the air-tube may be much dilated or contracted, particularly in chronic laryngitis associated with phthisis or syphilis, its various parts being in some cases much altered in shape and appearance, and its surface uneven. Erosions and ulcerations are common. Sometimes hæmorrhage occurs. In phthisis a chronic œdema of one or both ary-epiglottic folds is often observed, which is said to be pathognomonic. They look like pale, solid, pyriform enlargements, the larger ends lying against each other in the middle line, and the smaller ones being directed upwards and outwards. Among the early manifestations of syphilis in the larynx, Dr. Whistler has described, in addition to ordinary catarrhal congestion, a condition in which there is diffuse redness and swelling. He states that the redness is not so bright as in other laryngeal catarrhs, and is often more limited in its distribution; while the swelling is more a general puffiness than any great swelling. Subsequently, in the intermediate stage, he describes a more chronic inflammation, of which the signs are diffuse redness, thickening, and ragged ulceration, especially of the vocal cords.

**SYMPTOMS.**—In many cases of chronic laryngitis unpleasant and irritating sensations are experienced in the larynx, which are worse after speaking, but they are not marked as a rule. Alterations in the voice constitute the most important, and not uncommonly the only symptom of this disease. The voice is more or less weakened to complete aphonia, often hoarse and harsh, deep-toned, or cracked. It is subject to variations, and in the slighter cases frequently improves after the patient has talked for a time. The changes in the voice may only be noticed during loud talking. Paroxysms of spasmodic cough cause much distress in some cases, but this symptom may be completely absent. Many patients have a short, tickling cough; others hawk frequently, in order to clear away viscid secretion. The cough may be hoarse, cracked, barking, or aphonic; and is sometimes attended with much expectoration. Breathing is only disturbed when there is much thickening of tissues or œdema, with consequent narrowing of the larynx, and under these circumstances there may be considerable dyspnoea, with stridulous inspiration. Slight dysphagia is sometimes experienced.

The laryngoscope reveals the precise appearances presented in different cases of chronic laryngitis; and may further show that the muscles of the glottis do not act properly. Sometimes mucous râles can be heard over the larynx with the stethoscope.

There are no general symptoms directly due to chronic laryngitis, but the system is often affected, owing to some other local or constitutional affection being associated with this complaint.

This disease frequently aggravates the symptoms due to other forms of organic mischief in the larynx, such as ulceration or morbid growths.

### III. ULCERS OF THE LARYNX.

The forms of ulceration which are met with in the larynx include:—

1. *Catarrhal*, which are superficial.
2. *Follicular*, chiefly associated with chronic laryngitis.
3. *Variculous*, resulting from small-pox pustules.
4. Ulcers in connection with *typhus* and *typhoid fever*, which generally spread extensively and deeply.
5. *Phthisical* or so-called *tubercular* ulcers. Laryngeal ulceration in cases of phthisis does not by any means always originate in tubercle, but no doubt miliary tubercle does occur here, and it may be primary. On the other hand, it is probable that ulceration in the larynx may lead to pulmonary phthisis. Some observers have asserted that they have seen tubercles with the laryngoscope, before and during ulceration. Usually the ulcers are minute and circular at first, being often observed at the back of the ventricular bands, and on the under-surface of the epiglottis; by their union they give rise to large and irregular ulcerations, which may spread extensively. Sometimes phthisical ulceration begins on the vocal cords. Generally it does not cause deep destruction of tissues, but it may do so. The epiglottis is often eroded at its margins, and its cartilage may be exposed or perforated. Calcification and necrosis of the cartilages not infrequently follow phthisical ulceration. On the other hand, it is affirmed that such ulceration may heal partially or completely.
6. *Syphilitic*. Occasionally secondary syphilitic ulcers are met with in the larynx, which are said to be limited, superficial, and to occur in any part. As already stated, Dr. Whistler has described a ragged ulceration of the larynx in the intermediate period of syphilis, particularly affecting the vocal cords, accompanied with diffuse redness and thickening, and liable to relapse again and again after partial cicatrization. Tertiary ulcers exhibit a special tendency to begin on the epiglottis; they spread rapidly, both in extent and depth, causing great destruction of tissues, and presenting irregular ragged edges. In some cases the ulceration invades the larynx from the throat; or it may originate in the breaking-down of gummata. Sometimes it extends at one part, while cicatrizing at another. The scars have a great tendency to contract, and thus to narrow the calibre of the larynx; or to cause adhesion and distortion of structures.
7. *Cancerous*. This form of ulcer is very rare in the larynx.

**SYMPTOMS.**—In many of the slighter cases of laryngeal ulceration no special symptoms are noticed, and the same remark applies to those forms which are associated with the acute fevers.

Painful sensations may be felt in the larynx, of a burning, smarting or pricking character, increased by coughing or speaking; with tenderness on pressure. Deglutition is difficult or painful if the epiglottis is involved, especially as regards liquids. The voice is often altered in quality, being harsh, hoarse, or cracked, as well as weak, and may become aphonic. Suffocating fits of cough are common; and pus, blood, or laryngeal tissues may be expectorated. Breathing is frequently noisy and of laryngeal characters, and there may be urgent dyspnoea. In many cases the throat is simultaneously affected. Cicatrization of an ulcer may give rise to signs of permanent narrowing or stricture of the larynx.



#### IV. MORBID GROWTHS IN THE LARYNX.

The abnormal growths and tumours which may be found in the larynx are either **malignant** or **non-malignant**. The **malignant** growths include:—1. *Epithelial*. 2. *Encephaloid*. 3. *Scirrhus*. All the varieties are extremely rare, epithelial being the most common, and they usually extend to the larynx from other parts. The **non-malignant** comprise:—1. *Syphilitic condylomata* and *mucous tubercles*. There has been much discussion as to whether these growths are found in the larynx. Dr. Whistler, however, observed mucous patches here in 24 out of 88 cases of secondary syphilis. 2. *Papillomata*. 3. *Mucous polypi* or *fibro-cellular tumours*, either pedunculated or sessile. 4. *Fibrous tumours* or *polypi*. 5. *Cystic growths*. 6. *Lipomata*. 7. *Erectile vascular tumours*. 8. *Enchondromata*. 9. *Hydatids*. Those last mentioned are of extremely rare occurrence.

**SYMPTOMS.**—The size, situation, number, and nature of the morbid growths, as well as the size of the larynx, will necessarily influence the local symptoms. Rarely is there any pain, but occasionally a feeling of the presence of a foreign body is experienced; or a sense of obstruction or uneasiness. Dysphagia is sometimes felt. The voice is often partially or completely lost, or altered in quality, and it is liable to sudden changes. More or less dyspnoea is usually felt, while the breathing may be stridulous; this symptom is also subject to rapid variations, and frequent suffocative attacks may come on, which are due to spasm. When the growth is situated above the glottis, expiration is often quite free. The removal of part of a growth may increase the dyspnoea, in consequence of its position being disturbed, or of inflammation being set up. Cough is present in many cases, varying in its characters, and it is not infrequently voluntarily excited with the view of trying to get rid of the obstruction. In the expectoration, which is usually increased and abnormal, fragments of the growth are sometimes expelled, but Dr. Morell-Mackenzie states that the microscopic examination of these fragments cannot be relied upon for differential diagnosis. Laryngoscopic examination reveals the nature and seat of any growth. Sometimes it extends through the upper opening of the larynx, so that it is visible on inspection of the throat, or can be felt with the finger. A characteristic valvular murmur has been described as being heard over the larynx during breathing, but this is not reliable. Examination of the chest often reveals obstruction to the entrance of air into the lungs.

The mere interference with the function of respiration is liable seriously to affect the general system; and if the growth is malignant, the cancerous cachexia is often observed.

#### V. ŒDEMA GLOTTIDIS.

**ÆTIOLOGY.**—The loose submucous tissue which is present in some parts of the larynx is very liable to become the seat of œdema, which may arise under the following circumstances:—1. In connection with *acute laryngitis*, especially that due to local irritation. 2. From the irritation induced by *chronic laryngeal diseases*, for example, ulcers, growths, or

necrosis of cartilages. 3. As a *complication* of some of the acute specific fevers, namely, scarlatina, erysipelas, small-pox, typhus, or typhoid. 4. By *extension* of inflammation from the throat. 5. Occasionally as a part of *general dropsy* from renal disease; and possibly from cardiac or venous obstruction.

The *symptoms* are similar to those described as indicative of *œdematous laryngitis*. (See page 380.)

## VI. LARYNGEAL PERICHONDritis.—Abscess.—Necrosis of the Cartilages.

These rare morbid conditions may be briefly considered together. In *perichondritis* an exudation is said to collect between the perichondrium and the laryngeal cartilages, especially the cricoid, where afterwards pus forms, while the cartilages necrose, and are finally discharged in fragments. The irritation thus induced may lead to the formation of abscesses in the parts around.

Usually these changes are associated with ulceration. Necrosis of the cartilages is by far most frequent in cases of phthisis. Syphilis, abuse of mercury, low fevers, and cold have been set down as very rare causes of this disease.

**SYMPTOMS.**—These are considerable localized pain; extremely irritable cough; marked alterations in voice; and usually severe dyspnoea; followed by expectoration of fragments of cartilage, and signs of abscesses.

## VII. FUNCTIONAL OR NERVOUS AFFECTIONS OF THE LARYNX.

**1. Disorders of Sensation.**—The larynx is occasionally the seat of hyperæsthesia with irritable cough; of neuralgia; or of more or less diminution in sensibility, which may amount to complete anæsthesia.

**2. Laryngismus Stridulus—Spasm of the Glottis—Spasmodic croup—False or spurious croup—Child-crowing.**—This condition results from a spasmodic action involving the muscles which close the glottis, the proximate cause being some irritation conveyed by the laryngeal nerves. The irritation may be:—1. *Centric*, originating in the brain, either from some organic mischief, such as hydrocephalus; or from disturbance of its circulation, or of its nutrition. 2. *Direct*, from irritation of either vagus or recurrent nerve by enlarged glands, tumours, or other morbid conditions. Formerly the complaint in children was called *thymic asthma*, on the assumption that it was due to pressure by an enlarged thymus gland. 3. *Reflex*. The reflex irritation may arise in the larynx itself; or may be associated with dentition, improper feeding, especially in the case of infants brought up by hand or nursed by unhealthy mothers, worms, a cold draught blowing on the skin, and various other reflex disturbances.

Laryngismus stridulus is a very common complaint in children, especially during the first and second years of life. In adults it is only rarely observed, either in connection with hysteria, or as the result of pressure on the laryngeal nerves by aneurisms and other tumours, or from direct irritation by foreign bodies or gases. The complaint is most frequent among male children, and among those living in the crowded parts of large towns and cities, especially if brought up by

hand, and exposed to unfavourable hygienic conditions. Scrofulous children are said to be more subject than others, and rickets decidedly predisposes to the affection.

There may or may not be some obvious *exciting cause* of an attack of laryngismus. Thus it sometimes comes on during the act of swallowing; from tossing up the child in the air; or from severe mental emotion, especially fright or anger.

**SYMPTOMS.**—In children an attack of laryngismus in most cases comes on at night during sleep, and is very sudden in its onset. The prominent symptom is dyspnoea, more or less intense, attended usually with stridulous crowing inspiration, but the glottis may be for a moment completely closed, so that no air can enter, and respiration ceases entirely. The child struggles for breath, and presents to a greater or less degree the appearances associated with apnoea. Often there are general convulsions, with “carpopedal” contractions, strabismus, and sometimes involuntary discharge of faeces and urine. The attack subsides suddenly or very rapidly, and in many cases the child cries. Restoration is complete, and there is neither alteration in the voice nor cough. Pyrexia is usually absent.

An essential character of this complaint is the great tendency to the recurrence of the attacks. They vary in frequency, duration, and severity, but tend to become more frequent, longer, and more intense as the case progresses. Ultimately death from suffocation may occur during one of the attacks.

The spasmodic affections met with in adults do not call for any particular remark. Hysterical cough is believed to be sometimes due to a spasmodic tendency in the adductors of the vocal cords during expiration; and the sharp ringing cough which occasionally affects children is supposed to have the same cause, the spasm being reflex (Morell-Mackenzie).

**DIAGNOSIS.**—The only affection likely to be mistaken for laryngismus is inflammatory croup, and the diagnosis between these affections has already been pointed out.

**PROGNOSIS.**—Most cases of reflex origin recover, but those due to other causes are very serious. Much will depend upon the state of health of the child, and the severity and frequency of the fits.

**TREATMENT.**—During a paroxysm of laryngismus in children, measures must be immediately adopted for the relief of the spasm. Slapping or rubbing the back; shaking the child; dashing cold water in the face; fanning; tickling the throat so as to excite vomiting; the use of a warm bath alone, or with cold douching while the patient is in it; and holding ammonia to the nostrils, are the most effectual measures. A rapid *emetic* is useful if it can be taken; and if the attack persists, enemata containing assafoetida or valerian may be employed, sinapisms being also applied to the chest. Artificial respiration is sometimes serviceable. At the same time it is most essential to seek for any source of reflex irritation, and to remove this; for instance, lancing the gums often gives speedy relief. In prolonged cases inhalation of chloroform may be carefully tried; and sometimes it is necessary to have recourse to tracheotomy, which may be performed even after apparent death.

During the intervals between the attacks of laryngismus it is important to look to the diet; to regulate the state of the alimentary canal; and to improve the general health, or to treat any special constitutional condition, especially rickets. Tonics, change of air, and



salt-water bathing often do much good in cases where there is a tendency to laryngismus.

In adults the main indications are to remove the cause of any spasm; and to use sedative inhalations.

**3. Laryngeal Paralysis—Paralysis of the muscles of the vocal cords.**—Paralysis of the muscles of the larynx may arise:—1. As the result of some *local organic mischief*, past or present. 2. From *pressure* upon, or *traction* of the pneumogastric or recurrent nerves, one or both, by tumours, enlarged glands, &c. 3. After *diphtheria*; and, rarely, after *typhus* and other fevers. 4. In connection with *hysteria*, especially if there is much debility. 5. From chronic poisoning by *lead* or *arsenic*. 6. Very rarely from *centric disease* in the brain or upper portion of the spinal cord. 7. As a consequence of *atrophy and degeneration of the muscles*.

**VARIETIES AND THEIR SYMPTOMS.**—Four chief varieties of laryngeal paralysis are described, namely:—

(i.) *Bilateral paralysis of adductors—Hysterical or Functional aphonia.*—Voice is lost, but cough is usually attended with sound. The patient sometimes speaks in a scarcely audible whisper. The laryngoscope shows that the vocal cords remain apart, either partially or entirely, during attempted phonation; and they may be perfectly motionless.

(ii.) *Unilateral paralysis of adductors.*—Voice is altered, and there may be a permanent falsetto. The sound produced during coughing, sneezing, or laughing is usually much changed and weakened. The laryngoscope reveals that one cord does not act when the patient attempts to speak or cough, and that it is usually congested. This form of paralysis is generally due to some direct cause acting upon the supplying nerve.

(iii.) *Bilateral paralysis of abductors.*—Here the prominent symptom is dyspnoea, with noisy, stridulous inspiration, always present more or less, but subject to severe exacerbations, especially after exertion, or on taking a deep inspiration. Voice is not much affected, but may be harsh. Laryngoscopy discloses that the cords lie close together near the median line, and do not separate when an inspiration is made. Some observers maintain that paralysis is the pathological condition present in laryngismus, and not spasm.

(iv.) *Unilateral paralysis of the abductors* is attended with some degree of dyspnoea and noisy breathing; and the affected cord does not move during breathing, but remains near the median line.

In some cases both sets of muscles are involved, there being a combination of the symptoms and signs just described, which is usually the case when pressure is exerted on the recurrent nerves. In rare instances only a single muscle is paralyzed, and then the voice may merely be somewhat altered, being monotonous, or some notes not being capable of production. It is said that when there is unilateral paralysis of the larynx, the vocal fremitus communicated to the finger is less marked on that side than on the healthy one.

### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT OF CHRONIC LARYNGEAL AFFECTIONS.

**DIAGNOSIS.**—When symptoms indicate the existence of some chronic disorder in connection with the larynx, the diagnosis has to be made between:—1. Mere functional disturbance. 2. Pressure or irritation, either affecting the air-tube directly; or indirectly through its nerves. 3. Organic disease, of which, if present, it is requisite to determine as accurately as possible the nature, seat, and extent. The chief points to be taken into consideration are:—*a.* The personal and family history of the patient, as revealing some constitutional diathesis. *b.* The existing evidence of certain special diseases, especially phthisis, syphilis, or cancer; or of a hysterical condition. *c.* The exact local symptoms present, particularly as regards respiration and voice. *d.* The conditions revealed on careful examination of the chest, those to be specially looked for being phthisical disease of the lungs; and morbid conditions which might directly affect the windpipe or its nerves. *e.* The results of laryngoscopic examination. It is only by the efficient employment of the laryngoscope that positive and accurate information can be obtained in many cases.

**PROGNOSIS.**—All *organic* laryngeal affections are troublesome, and some of them highly dangerous, especially extensive ulceration; great thickening of tissues; destruction of the cartilages; and the existence of certain morbid growths, or of such growths as cannot be removed. The danger to life is in proportion to the degree of interference with breathing; and the liability to spasm. In many cases there is no fear of a fatal result, but the prognosis as regards the restoration of the functions of the larynx is unfavourable. Much will depend upon the constitutional condition. Syphilitic disease may often be rapidly cured under proper treatment. Laryngeal phthisis is very serious and intractable as a rule. Cancer is necessarily fatal. With respect to the *functional* disorders, paralysis of the adductors is favourable usually; that of the abductors very unfavourable, the patient being in great danger. The cause of the paralysis will necessarily influence the prognosis.

**TREATMENT.**—1. **General management.**—Rest to the larynx as far as possible; residence in a dry atmosphere, of warm and uniform temperature; the removal of all causes of local irritation; the stoppage of any injurious habit, such as excessive smoking; and the wearing of sufficient warm clothing over the neck and chest, are the principal *general* matters requiring attention in the treatment of any organic laryngeal disease. In some cases a change of climate is imperative, if it can be obtained; but if not, a respirator should be worn, at the same time damp, cold, and especially night air being avoided.

2. **Constitutional treatment** is often of great importance, especially for syphilitic and phthisical affections. In many cases of chronic laryngitis *tonics* are useful; or treatment directed to the alimentary canal may be called for. Iodide of potassium is a valuable remedy in some cases of chronic laryngitis which are quite independent of syphilis. Various German waters and those of the Pyrenees are recommended in

obstinate cases of this disease. Sometimes deglutition is much affected, owing to the condition of the epiglottis, especially in laryngeal phthisis, and then particular attention is required as regards the feeding of the patient, care being taken that a sufficient quantity of nutriment is consumed. It is useful in these cases to thicken liquids with corn-flour or arrowroot. Sometimes the food must be administered through an œsophageal tube, or by enemata.

3. **Local treatment** is, however, in most cases that requiring the chief attention in laryngeal diseases. Remedies are best applied by means of a camel's-hair brush attached to a bent handle; by inhalation or the spray-inhaler; or by blowing in powders. Lozenges are very useful if the throat is affected at the same time. The applications should be made effectually, regularly, and as frequently as each individual case may require, the laryngoscope mirror being made use of, if needed, in order to give sufficient light. The chief substances thus employed are:—

1. *Mineral astringents* and *caustics*, namely, nitrate of silver; chloride, sulphate, or acetate of zinc; alum and chloride of aluminium; perchloride of iron; or sulphate of copper. 2. *Vegetable astringents*, such as tannin or kino. 3. *Volatile stimulating liquids* in inhalations, as creosote, carbolic acid, oil of pine or juniper. 4. *Sedatives* in inhalations, especially conium, tincture of benzoin, ether or chloroform. Glycerine is the best solvent when any remedy is applied with the brush. Different applications are required in different cases, but Dr. Morell-Mackenzie finds chloride of zinc one of the most useful mineral astringents in chronic laryngitis. Tannin is said to be very valuable in some cases of laryngeal phthisis; and nitrate of silver in syphilitic ulceration. Prof. Krishaber, however, maintains that no topical remedies are of any service in curing laryngeal phthisis, and merely recommends narcotic applications to relieve suffering.

As regards morbid growths, it is generally considered that these require removal by operation. *Evulsion* is the chief method of operation usually employed, and various instruments have been invented for carrying it out, but it is useless to enter into any description, as only those who have had considerable practical experience would be likely to undertake such operative interference. Even cancer may sometimes be removed with temporary benefit. Mr. Lennox Browne has advocated the treatment of benign growths by the use of topical applications, instead of by their removal. Caustic remedies are of no value except for the destruction of condylomata. The *galvanic cautery* has been employed in the treatment of laryngeal growths.

Other operations performed in connection with the larynx are catheterism, "tubage," or mechanical dilatation; and partial or complete extirpation. For details on these points reference must, however, be made to special works.

4. Not uncommonly **tracheotomy** is called for, in order to prevent suffocation, in cases of extensive ulceration, morbid growths, or great thickening and contraction of the larynx, and the results are sometimes very satisfactory. Subsequently it may be desirable to remove a growth by dividing the thyroid cartilage. The entire larynx has been removed with success.

5. **Prevention.**—In all cases where there is a liability to chronic laryngeal disease, every precaution must be taken to guard against its development, by avoiding cold, excessive use of the voice, and other causes which are known to affect the air-tube. The front of



the neck should also be properly protected. In cases of phthisis the least indication of any laryngeal irritation demands prompt attention.

6. For laryngeal functional disorders general treatment is often indicated. All obvious causes of irritation must be removed, if possible. Local faradization is the great remedy in paralysis of the abductors, one pole being placed over the thyroid or cricoid cartilage, and the other in contact with the vocal cords. Paralysis of the adductors usually demands the performance of tracheotomy, in order to avert suffocation. The treatment of laryngismus stridulus has already been considered.

## CHAPTER VIII.

### DISEASES OF THE BRONCHI.

#### I. ACUTE BRONCHIAL CATARRH—ACUTE CATARRHAL BRONCHITIS.

**ÆTIOLOGY.—Predisposing causes.**—These are:—Early or advanced age; indulgence in relaxing and enervating habits; immoderate clothing of children; debility from any cause; the presence of certain constitutional diseases, for example, rickets, gout; chronic pulmonary affections, or previous attacks of bronchitis; cardiac diseases or other conditions which induce overloading of the bronchial vessels; a cold and damp climate or season, especially if liable to sudden changes of temperature; occupations involving exposure, rapid changes of temperature, or the breathing of irritating particles; and residence in the poorer and unhealthy districts of large towns.

**Exciting causes.**—1. In the great majority of cases bronchitis results from *taking cold* in some way or other, such as by exposure to cold and wet, sitting in a draught when perspiring, sudden change in temperature, wearing damp or insufficient clothing, or sleeping in a damp bed. Undoubtedly many children suffer in consequence of the legs and lower part of the body being left unprotected. 2. *Direct irritation* of the bronchial mucous membrane is another frequent cause, set up by very hot or cold air; irritant gases; mechanical particles in the inspired air, such as cotton, wool, dust, steel; blood; irritating secretions; and morbid growths, for example, tubercle or cancer. 3. *Blood-poisoning* may induce bronchial catarrh, as in various fevers, especially typhoid and measles; in gout, rheumatism, or syphilis; after the sudden disappearance of acute or chronic skin-affections, or the suppression of habitual discharges; or during the administration of certain medicines, especially iodine. 4. Bronchitis occurs as an *epidemic*, associated with influenza.

**ANATOMICAL CHARACTERS.**—The morbid appearances directly indicating bronchial catarrh include redness, varying in its hue and arrangement; swelling, opacity, relaxation, and diminution in consistency of the mucous membrane; at first dryness of the surface, soon followed, however, by excessive secretion, which changes in its

characters as the case progresses, consisting at first of clear frothy mucus, but afterwards becoming more opaque and viscid, mucopurulent or purulent, owing to the abundance of cells; and often epithelial abrasions, or even slight ulcerations. Occasionally blood is present in the tubes; or fibrinous particles or casts may be visible.

The appearances will necessarily vary considerably according to the extent, severity, and stage of the disease. The redness is most marked towards the upper part of the lungs, and at the bifurcations of the bronchi, but is rarely perceptible beyond their fourth or fifth divisions, and it may disappear after death, owing to the contraction of the muscular and elastic fibres. The inflammatory products are most abundant towards the bases, and in the dependent parts of the lungs; by their accumulation in the air-cells and minute bronchi they sometimes give rise to yellow spots near the surface, especially in children. Both lungs are usually affected in bronchitis, but to an unequal extent.

As complications associated with bronchitis the chief conditions observed are pulmonary congestion and œdema; lobular or more extensive collapse; acute emphysema or insufflation; lobular or rarely lobar pneumonia; and pleurisy. The venous portion of the circulation is liable to be overloaded with dark blood. The bronchial glands are often red, soft, and enlarged.

**SYMPTOMS.**—It will be necessary to allude to certain varieties which acute bronchitis presents in its clinical history, but in a general way the *local* symptoms may be summed up as:—Unpleasant or painful sensations in the chest; interference with breathing; and cough, with expectoration of the materials formed in the tubes. More or less pyrexia is almost always present; while in some cases there is a tendency to suffocation, from blocking-up of the bronchial tubes: in others to adynamic symptoms.

**1. Primary or Idiopathic Bronchitis.**—*a. Involving the larger and medium-sized tubes.* When due to a cold, acute bronchitis is usually ushered in by coryza, sore-throat, and some degree of hoarseness; chilliness or slight shivering, alternating with a sense of heat; general pains and languor; drowsiness with restlessness; furred tongue, anorexia, and constipation. Occasionally slight delirium is observed; or in very young and weakly children convulsions may occur. The symptoms of the established disease are *local* and *general*.

*Local.* Subjective sensations of heat, burning, rawness, soreness, tickling, or actual pain are experienced to a greater or less degree over the front of the chest, but especially behind the upper part of the sternum, and in the supra-sternal notch. These are increased by a full inspiration, and the act of coughing often gives rise to much tearing pain. There may be tenderness over the sternum. Muscular pains are common as the result of cough, especially towards the sides and base of the thorax. A sense of oppression, weight, or tightness is felt across the chest; and respiration may be somewhat hurried and laborious, but there is no evident dyspnoea. Cough is a prominent symptom, being due at first to the irritable condition of the lining membrane of the air-passages, and subsequently to the secretions formed in them. It is paroxysmal in character, often irrepressible and violent, especially on lying down and on waking up in the morning. Expectoration soon occurs, the sputa consisting at first of a little clear, thin, frothy mucus;

and afterwards increasing in quantity, and becoming muco-purulent, more or less opaque, viscid, and scarcely at all aerated. Sometimes they are very tenacious, and adhesive or ropy, and may form distinct "nummulated" masses. As they alter in their characters, they are more easily expelled. Occasionally the expectoration is streaked with blood. Under the microscope epithelial cells, numerous young cells, exudation- and pus-corpuscles are the chief elements observed, along with abundant granular and molecular matter; sometimes a few blood-discs, fibrinous coagula, or crystals are visible.

*General.* If the bronchitis is at all extensive, a certain degree of pyrexia is present, but it is never very marked. The patient frequently feels very languid and weak. Other mucous membranes are often the seat of catarrh along with that lining the bronchi.

*b. Bronchitis extending into the minute tubes. Capillary Bronchitis.* In most cases this form of bronchitis is but an extension of that already described, being preceded by its symptoms, but sometimes the smaller tubes seem to be affected at the same time as the larger, or quite independently, and then well-marked rigors may occur at the outset, with headache and vomiting. The peculiar features of capillary bronchitis are as follows:—1. Pain is often slight or absent, except the muscular pains resulting from cough, which are very severe. 2. Breathing is always greatly disturbed, being accelerated sometimes to 50 or more per minute; it is wheezing or crepitous in character: and attended with effort, as well as with a considerable sense of want of air. The pulse-respiration ratio is altered, being in some instances about 2.5 to 1. Urgent dyspnoea is observed in severe cases, either constant or paroxysmal, which may amount to orthopnoea. 3. Cough is exceedingly frequent and violent, and during the act patients often sit up or bend forward, and hold their sides. 4. Expectoration is very difficult, the sputa being abundant, as well as usually viscid and tenacious, containing also minute fibrinous casts of the tubes. 5. The general symptoms are of an aggravated character, there being at first considerable fever, the temperature occasionally rising to 103° or more, with much exhaustion and weakness. The urine sometimes contains a little albumen, or a trace of sugar. As the case advances, the tendency is to the development of the usual symptoms indicating suffocation and venous congestion, usually gradual in their onset, occasionally rapid or sudden, owing to the speedy filling-up of the tubes, the cough diminishing, the breathing becoming shallow, and the expired air cool. In some instances, however, typhoid symptoms set in; or there may be a combination of both classes of phenomena.

It is necessary to allude to certain individual peculiarities. Children are very liable to show signs of deficient blood-aëration, even in the slighter forms of bronchitis, especially if they are feeble and badly nourished or rickety, because they cannot expel the sputa. They usually swallow any materials coughed up, and, therefore, in order to examine the expectoration it is necessary to wipe the base of the tongue with a handkerchief after a cough. Healthy adults do not suffer nearly so severely as a rule. In aged persons, or in those who are constitutionally weak from any cause, the fever is very apt to assume an adynamic type, even though the bronchitis is not extensive. The term *peripneumonia notha*, formerly much employed, properly includes cases of capillary bronchitis occurring in old or weak subjects after some chronic malady, attended with febrile symptoms at first, signs of adynamia and deficient aëration of blood setting in, however, at an early period.



**2. Secondary Bronchitis.**—This term is applied to bronchitis occurring in connection with the exanthemata; in the course of blood-diseases, such as gout, rheumatism, Bright's disease; or in cases of chronic pulmonary or cardiac affections. In nearly all these conditions the complaint is apt to come on very insidiously, without any of the usual symptoms being at all prominent, and it is often a dangerous complication. The expectoration is said sometimes to contain peculiar materials which accumulate in the blood, for instance, uric acid in cases of gout. Pulmonary deposits usually give rise to localized bronchitis. When acute bronchitis complicates emphysema and chronic bronchial catarrh, especially if associated with cardiac disease, urgent dyspnoea and signs of apnoea are likely to set in speedily, accompanied with general venous congestion and dropsy; the expectoration is also very abundant and frothy at first in these cases, and subsequently its discharge may be exceedingly difficult.

**3. Mechanical Bronchitis.**—When due to the inhalation of irritant particles, the attacks of bronchitis are of frequent occurrence, but comparatively slight in degree, there being no pain or fever, the chief symptom being an irritable cough with but little expectoration, which may contain some of the particles inhaled.

**4. Epidemic Bronchitis** has been already sufficiently described in connection with influenza.

It must be remembered that the complications previously mentioned may be present in cases of bronchitis, modifying the symptoms, as well as the physical signs, which now remain to be considered.

**PHYSICAL SIGNS.** 1.—The chest may be somewhat *enlarged* in bronchitis, from insufflation of the lungs. 2. *Respiratory movements* tend to be more or less frequent and deep; expiration is sometimes prolonged; and if the tubes are extensively filled, the upper part of the chest moves unduly. In children signs of *inspiratory dyspnoea* are very common. 3. *Rhonchal fremitus* is frequently present, varying in its characters. 4. *Percussion* may reveal increase in extent and degree of pulmonary resonance, on account of distension of the lungs; or occasionally some deficiency of resonance at the bases is observed, due to accumulation of secretion, congestion and cedema, or collapse. In infants a sound resembling the crack-pot sound may be frequently elicited. 5. *Respiratory sounds* are loud and harsh, with prolonged expiration, where the tubes are free; where these are obstructed, they are weak or absent, or may be completely obscured by rhonchi. 6. The various *rhonchi* due to the narrowing of the tubes, or to the fluids contained within them, constitute the most important physical signs of bronchitis. They may be of sonorous, sibilant, mucous, submucous, or subcrepitant character, according to the exact physical conditions present, and these adventitious sounds often co-exist in different parts of the chest. At first the dry rhonchi are only or chiefly heard, while the moist râles are principally observed towards the bases of the lungs. When fluids collect in the larger tubes, rhonchal sounds may be audible at a distance from the patient. Cough affects them considerably. Occasionally the heart's action may originate râles in the neighbouring tubes.

**DURATION AND TERMINATIONS.**—According to its severity, a case of bronchitis may end in three or four days, or be prolonged for two or three weeks or more. Capillary bronchitis generally proves fatal from the sixth to the twelfth day, death occurring earlier in children than in adults as a rule. There is always the danger of a relapse, or of extension

of the inflammation. The *terminations* are :—*a.* In recovery, but in severe cases convalescence may be very prolonged, and cough is liable to remain for some time. *b.* In death, either from gradual or sudden apnoea; or from adynamia. *c.* Occasionally by transition into the chronic state. As *sequelæ*, emphysema, pulmonary collapse, deformed thorax in children, or acute or chronic phthisis may remain.

DIAGNOSIS.—The diagnosis of the diseases of the lungs will be hereafter considered in a separate chapter. At present it need only be remarked that the chief diseases from which acute bronchitis requires to be distinguished are whooping cough; croupous and other forms of laryngitis; pneumonia, especially lobular; and acute phthisis. It is of importance to recognize any complication occurring during the course of an attack; and also not to mistake bronchitis associated with one of the exanthemata for the sole complaint.

PROGNOSIS.—Bronchitis is often a very dangerous disease, and stands high as a cause of death in this country. The circumstances which increase its gravity are :—Very early or advanced age; a bad state of health, or the existence of some chronic or acute disease of a general character; previous organic mischief in the lungs, especially extensive emphysema; the presence of disease of the heart; extensive implication of the smaller bronchial tubes, with great difficulty of expectoration; signs of accumulation of materials in the tubes, with shallow breathing and cessation of cough, or of their extensive obstruction, the latter to be especially looked for in children; urgent dyspnoea, with signs of apnoea; the presence of adynamic symptoms: the occurrence of dangerous complications; neglect of treatment; and a low epidemic type.

TREATMENT.—Early attention is required in all cases of bronchitis, but especially when children are affected. Confinement to the house or even to one room is advisable; and if the case is at all severe, the patient should remain in bed, warmly clad in flannel, avoiding exposure of the chest, the room being kept at a temperature of from 65° to 68°, and it may be necessary to moisten the air by means of steam. When the attack results from a cold, it is useful at the outset to induce free perspiration by means of a copious hot drink, aided by a warm foot-bath, to which may be added a little mustard; or a hot-air, vapour, or Turkish bath may be employed, the patient then going to bed, and lying between blankets, covered with abundant bedclothes. A full dose of Dover's powder may be given, or a *saline diaphoretic* draught. A sinapism over the chest is useful; and if the larynx is at all involved, steam inhalation should be resorted to. An *emetic* at the outset is in much favour with some practitioners in severe cases of bronchitis, and might occasionally be serviceable in the treatment of children.

Should the symptoms not subside, the indications are :—1. To subdue the inflammation as soon as possible. 2. To promote the discharge of the materials forming in the tubes, and to diminish their quantity if they are excessive. 3. To relieve unnecessary cough. 4. To allay spasm of the bronchial tubes, if present. 5. To pay attention to the constitutional condition, and to support the strength if it fails. 6. To treat apnoea, excessive fever, or adynamia, should either of these conditions set in. 7. To attend to complications.

1. For the purpose of fulfilling the first indication, general or local bleeding, and the administration of tartar emetic, tincture of digitalis, or tincture of aconite are the chief measures advocated. Venesection is very rarely required or admissible, but moderate local bleeding, by means



of leeches or cupping, may be occasionally beneficial, though much discrimination is necessary in adopting this measure. The front of the chest, and the posterior base are the sites from which blood may be taken with most advantage. The application of two or three leeches sometimes proves highly efficient in relieving severe dyspnoea in plethoric children.

Tartar emetic is decidedly valuable in the early stage of severe cases of bronchitis, provided the patient is strong and not too old. It may be given with liquor ammoniæ acetatis and a few drops of compound tincture of camphor, in doses of  $\frac{1}{4}$  to  $\frac{1}{2}$  gr. for an adult. Tincture of digitalis and tincture of aconite have been well spoken of, and are deserving of more extended trial.

2. The next three indications are carried out mainly by the administration, in various combinations as they are required, of:—*a. Expectorants*, namely, at first ipecacuanha wine, tincture or syrup of squills, compound tincture of camphor; and later on carbonate of ammonia, chloride of ammonium, infusion of senega or serpentary, ammoniacum, galbanum, or tincture of benzoin. *b. Sedatives and narcotics*, especially opium or morphia, henbane, conium, hydrocyanic acid, or chlorodyne. *c. Antispasmodics*, such as the various ethers, tincture of lobelia, or spirits of chloroform. Each case of acute bronchitis must be studied carefully, and the remedies varied according to its requirements. They may be combined with *demulcents* or *diaphoretics*. Should the tubes be much loaded, and expectoration be difficult, narcotics, particularly opium, must be avoided, and the patient should lie with the head high, and should be encouraged to cough frequently, not being allowed to sleep for too long a time. It is especially necessary to attend to these matters in the treatment of children. Should there be indications of dangerous accumulation, an emetic of sulphate of zinc is very useful. On the other hand, if there is an irritable cough, it may often be voluntarily repressed by the patient, and sedatives are then most valuable. Strychnine is of great value for stimulating the respiratory function; and digitalis to improve the cardiac action should it be failing. *Inhalations* are frequently beneficial, those of conium, ether, chloroform, hop, or benzoin, for the purpose of relieving cough and checking spasm; later on those of tar, creosote, or carbolic acid, to diminish or improve the sputa.

3. *Local treatment* is generally called for in bronchitis. Repeated sinapisms, hot or turpentine fomentations, and linseed-meal poultices over the chest are beneficial at first. When the acute symptoms have subsided, a blister may be useful; or, if there is a tendency to chronic catarrh, some more powerful application may be required, turpentine or croton oil liniment being the most efficacious. Free dry-cupping frequently relieves troublesome dyspnoea and oppression about the chest, especially when acute bronchitis complicates emphysema with chronic catarrh. In these cases flying blisters and turpentine stupes are also very serviceable.

4. The *constitutional conditions* chiefly requiring attention in cases of bronchitis are general debility, rickets, tuberculosis, and gout. Lowering treatment is not borne when either of these is present. It is quite needless to keep patients on too low a diet, and considerable support is often called for in bad cases. Stimulants are not requisite as a rule, and may do harm, but they must be given if necessary, particularly if signs of adynamia or apnoea set in, their effects being carefully watched. Excessive pyrexia calls for full doses of quinine. Any tendency to asphyxia must be combated by the usual measures.



5. In the treatment of children, one of the best remedies in most cases of bronchitis is ipecacuanha wine in moderate doses, repeated every three or four hours. When it attacks old people or those who are enfeebled from any cause, or when it occurs as a secondary complication, wine or brandy and abundant nourishment are usually required, and a mixture should be given containing carbonate and muriate of ammonia, ether or spirits of chloroform, and tincture of squills, with camphor mixture, decoction of bark, infusion of senega, or ammoniacum mixture. Capillary bronchitis in the majority of cases demands a stimulant and supporting treatment throughout.

6. Proper precautions must be taken until *convalescence* is thoroughly established, especially against cold, damp, and night air; while flannel should be worn next the skin. *Tonics* are often useful during recovery, such as quinine, mineral acids, or iron. Due care must also be exercised in the case of those who are subject to bronchitis, and for such persons a change to some genial climate during the winter season is very desirable.

## II. CHRONIC BRONCHITIS—CHRONIC BRONCHIAL CATARRH.

**ÆTIOLOGY.**—As a rule chronic bronchitis follows repeated attacks of the acute disease, but occasionally it remains after one attack, or begins as a chronic affection. It is frequently associated with gout and other constitutional complaints, chronic lung-diseases, heart-affections causing pulmonary congestion, or chronic alcoholism; or it results from breathing irritant particles. Persons advanced in years are by far most subject to chronic bronchial catarrh, but even children are liable to suffer.

**ANATOMICAL CHARACTERS.**—When this complaint has been long established, it leads to considerable changes in the bronchial tubes. Their lining membrane becomes dark-coloured, often of a venous hue, or here and there greyish or brownish; and the capillaries are visibly enlarged and varicose. Thickening of tissues, increased firmness, amounting in some instances to marked induration, and contraction of the tubes are observed, with loss of elasticity and muscular hypertrophy. The cartilages may ultimately calcify. The small tubes are narrowed or closed up; while the larger ones are often dilated, and gape on section. The surface of the mucous lining is uneven, frequently presenting extensive epithelial abrasions, or occasionally follicular ulcers. In some cases there is only a little tenacious mucus in the tubes, but usually they contain abundant muco-purulent or purulent matter, or frothy mucus.

**SYMPTOMS.**—Cases of chronic bronchitis present much variety as regards the severity and exact characters of their symptoms, depending upon the extent of the affection; and upon its frequent association with other morbid conditions in the lungs, especially emphysema, dilated bronchi, or phthisis; with cardiac affections; or with some constitutional diathesis. They may, however, be conveniently classed under three main groups:—

1. **Ordinary Chronic Bronchitis.**—In many instances patients only suffer from this complaint during the cold season, having a winter-cough; but after a while the symptoms often become permanent to a greater or less degree, being liable to exacerbations in cold and damp weather. A little uneasiness or soreness may be felt behind the ster-

num, increased by coughing; and a sense of oppression across the chest, with shortness of breath on exertion, is usually experienced in severe cases. Cough is the main symptom, occurring chiefly in paroxysms, varying greatly in severity and frequency; it is often very annoying on first going to bed, and early in the morning. The cough is attended with expectoration, the sputa being frequently abundant and difficult to expel; and consisting of greyish mucus, yellowish or greenish mucopurulent or purulent matter, or a mixture of these materials, usually running into one mass, but occasionally remaining in separate lumps, which may be nummulated. Being but slightly aerated, the masses not infrequently sink in water. Occasionally blood-streaks are observed. Sometimes a most offensive or even gangrenous odour is given off, supposed to be due to decomposition of the sputa, or to the presence of microscopic sloughs. The microscope reveals much granular matter, with imperfect epithelial and pus-cells, and often blood-corpuscles.

Severe cases of chronic bronchitis may be attended with considerable wasting and debility, slight evening pyrexia, and night-sweats, but when these symptoms are present, phthisis should always be carefully looked for.

**2. Dry catarrh—Dry bronchial irritation.**—This variety is particularly observed in connection with gout or emphysema; as a result of irritant inhalations; and in sea-side places. More or less dyspnoea is experienced, with a sense of tightness and oppression across the chest, and wheezing; very distressing paroxysms of irritable cough come on, either quite dry, or only followed by the expectoration of a small pellet of greyish, pearl-like, tough mucus, compared to boiled starch, or of a little watery fluid.

**3. Bronchorrhœa.**—Most frequent in old people, especially in connection with cardiac diseases, this form is characterized by the expectoration being very abundant, sometimes amounting to as much as four or five pints in the twenty-four hours; in character being either watery and transparent, or glutinous and ropy, resembling a mixture of white of egg and water, and scarcely at all frothy. The cough is paroxysmal and often violent, but may be insignificant compared with the quantity of fluid discharged. Patients frequently obtain relief from dyspnoea and other unpleasant sensations after a spell of coughing. In severe cases there may be a loss of flesh, and proportionate weakness.

**PHYSICAL SIGNS.**—The only signs directly due to chronic bronchial catarrh are:—1. *Rhonchal fremitus*. 2. *Harsh respiratory sounds*, with prolonged expiration. 3. *Sonorous and sibilant rhonchi*, with large *mucous râles* towards the bases, the latter being rarely abundant, and varying in characters according to the consistence of the contents of the tubes. Other signs are generally present in cases of long duration, but they are dependent upon emphysema and other morbid changes accompanying the bronchial catarrh.

**PROGNOSIS.**—When chronic bronchitis is confirmed, only rarely can the complaint be thoroughly cured. In less advanced cases, however, complete restoration may be effected if due precautions are exercised. Patients suffering from chronic bronchitis often live to a good old age, but lead an uncomfortable existence. The chief dangers to which they are liable are that the disease should become more and more extensive, or should induce emphysema, dilated bronchi, collapse, or phthisis; or that an acute attack might supervene, which is frequently highly dangerous.

TREATMENT.—1. From what has just been stated, it is obvious that all cases of chronic bronchitis ought to be thoroughly attended to at as early a period as possible. The patient must be removed from every source of irritation, and must observe due precautions against exposure, wearing warm clothing, with flannel next the skin. If a suitable climate cannot be obtained, it will be well for the patient to keep indoors during bad weather, or if obliged to go out, a respirator should be worn in appropriate cases.

2. It is very important to look to the state of the *heart*, of the *digestive organs*, and of the *general system*. If cardiac disease is present, infusion or tincture of digitalis is often very useful. By relieving dyspeptic symptoms, and keeping the bowels freely open, much good may also frequently be effected in cases of chronic bronchitis. Any constitutional diathesis present must be attended to, especially gout, rheumatism, rickets, or tuberculosis; and a plethoric or anæmic state of the blood should be corrected. A great many cases of chronic bronchitis do well under a course of treatment by *tonics* and good diet, with some stimulant, especially if there is abundant expectoration, causing debility and wasting. Quinine, strychnine, preparations of iron, hypophosphites, or mineral acids with bitter infusions, are often very valuable, as well as cod-liver oil. In some cases mineral *nervine tonics*, such as sulphate or oxide of zinc, are beneficial.

3. Much discrimination is requisite in chronic bronchitis in the employment of remedies which have a *local* action. The main indications are to limit excessive secretion; to assist expectoration, should the act be difficult; to allay irritable cough; and to subdue spasm of the bronchial muscular fibres. The first indication is carried out by the internal administration of chloride of ammonium; of balsams and resins, especially balsam copaibæ, ammoniacum, or galbanum; or of astringent preparations of iron, acetate of lead, mineral acids, tannic or gallic acid: and by employing inhalations of steam impregnated with tar, creosote, carbolic acid, or naphtha; or very dilute dry inhalations of iodine, chlorine, balsamic and resinous vapours, or the vapour of chloride of ammonium. The other indications are fulfilled by means similar to those mentioned under acute bronchitis, and like precautions must be observed in the use of narcotics, should there be a tendency to accumulation of secretion. If the sputa are very viscid, alkaline carbonates or liquor potassæ may prove beneficial. Sedative inhalations are most valuable should there be much irritable cough. Tincture of Indian hemp is sometimes useful when there is marked tendency to spasm.

4. The chest should be covered in front with some warm plaster, or with cotton-wool. Free dry-cupping, sinapisms, blisters, turpentine liniment, croton oil liniment, strong or diluted, chloroform liniment, and other local applications are frequently serviceable in chronic bronchitis.

5. Change of climate or a sea-voyage proves most beneficial in many cases. All forms of bronchitis require a tolerably warm region, which is not subject to rapid changes of temperature, or exposed to cold winds, and which is situated at a moderate elevation. Dry catarrh needs a soft and relaxing atmosphere, of moderately high temperature. If there is much expectoration a dry, warm, and more or less stimulating atmosphere answers best. Torquay, Penzance, Bournemouth, Grange, Clifton, and Tunbridge Wells in this country; and Cannes, Mentone, San Remo, Bordighera, Pisa, Rome, Algiers, and Corfu, in foreign countries, are the chief places available for patients suffering from chronic bronchitis.



## III. PLASTIC OR CROUPOUS BRONCHITIS.

ÆTIOLOGY.—Young adults suffer most frequently from this rare complaint, and it is stated to be rather more common among females. It is supposed to be due to some diathetic condition, and to be generally associated with a weak constitution, or sometimes with tuberculosis. The affection may, however, certainly be met with in strong and apparently healthy persons.

ANATOMICAL CHARACTERS.—A plastic exudation collects in the bronchial tubes, forming whitish casts, varying in size according to the tubes affected, as well as in extent; being either hollow or solid; and sometimes presenting concentric layers. It consists of an amorphous or fibrillated substance, enclosing granular matter, oil-globules, and cells, some of which are nucleated. Some pathologists have supposed that the material is merely altered blood, the result of bronchial hæmorrhage, but this is not a correct view, the exudation being probably an inflammatory product usually.

SYMPTOMS.—Plastic bronchitis is almost always chronic in its course, but presents acute exacerbations. The affection is characterized by fits of cough and dyspnœa, more or less frequent, severe, and prolonged, being sometimes extremely aggravated; followed and usually relieved by the expectoration of fibrinous masses, which on being unravelled under water exhibit tree-like casts of the tubes. There may be more or less hæmoptysis, which may be on a large scale. Sometimes extensive bronchial catarrh or pneumonia is set up; and considerable pyrexia may be observed. In the intervals patients suffering from this complaint often feel perfectly well.

The *physical signs* indicate obstruction of the bronchial tubes, more or less complete and extensive, leading to emphysema or pulmonary collapse. *Dry rhonchi* are often audible on auscultation, especially those of a *sibilant* character, with a few *mucous râles*.

A case came under my notice, in which a tolerably healthy-looking young man was affected with plastic bronchitis, bringing up a quantity of casts almost daily, but who scarcely suffered any inconvenience.

TREATMENT.—During the attacks of dyspnœa and cough associated with plastic bronchitis the use of inhalations; the external application of sinapisms, turpentine fomentations, or a blister to the chest; and the internal administration of *sedatives*, with tartar emetic or ipecacuanha wine, might be tried. For the cure of the complaint there is no known remedy. *Tonics*, cod-liver oil, change to a warm climate, or a long sea-voyage seem to be most beneficial. Tartar emetic, iodide of potassium, alkalis and their carbonates, mercury, inhalations of iodine, and various other remedies have been tried, but usually without success.

## IV. DILATATION OF THE BRONCHI—BRONCHIECTASIS.

ÆTIOLOGY.—Bronchiectasis generally arises in the course of some chronic lung-disease, especially bronchitis, phthisis, and chronic interstitial pneumonia. Its immediate causes are supposed to be:—1. *Morbid changes* in the walls of the bronchi, diminishing their resisting power.

2. *Increased pressure of air* from within, either during cough in parts unsupported; or during inspiration, in consequence of obliteration of a number of air-vesicles, the dilatation of the bronchi being then compensatory. 3. *Persistent pressure of stagnant secretion.* 4. *Contraction of lung tissue*, in connection with chronic interstitial pneumonia, the enclosed bronchi becoming dilated in the process.

ANATOMICAL CHARACTERS.—The bronchi may be extensively dilated, and of a fusiform shape; or they present one or more limited globular enlargements. Their size varies considerably. After a time their inner surface becomes irregular, and occasionally ulcerated; they contain a muco-purulent or purulent substance, which is often foetid; and they are sometimes the seat of gangrene or of hæmorrhage. Their contents may ultimately dry up, becoming caseous and even calcareous; and finally the dilated tubes may become obliterated.

SYMPTOMS.—The only significant symptom of dilated bronchi is the occurrence of severe paroxysms of cough, ending with abundant expectoration, which is discharged with much difficulty, depositing a thick sediment on standing, being often exceedingly foetid, and also containing caseous particles. The expired air has generally an extremely foul odour when the patient coughs.

PHYSICAL SIGNS.—The signs which may give evidence of bronchiectasis are:—1. *Tubular percussion-sound* occasionally. 2. Loud bronchial, blowing, tubular, or cavernous *breath-sound*, which may be heard after a cough when previously absent. 3. Various *moist râles*, which may be of hollow character. 4. Loud *bronchophony* or *pectoriloquy*.

TREATMENT.—The chief matter requiring attention is to see that the secretions are not allowed to stagnate in the dilated bronchi, the patient being encouraged to cough, and expectoration being assisted. The sputa may be improved and limited in their amount by means of inhalations of carbolic acid, creosote, or other *antiseptics*.

---

## CHAPTER IX.

### PULMONARY CONGESTION.—CEDEMA.—HÆMORRHAGE.

THESE morbid conditions may be considered together, as they are in many instances but stages of the same process.

ÆTIOLOGY.—Hyperæmia of the lung may be *active*, *mechanical*, or *passive*. *Active* congestion results from:—1. *Increased cardiac action* from any cause. 2. Probably *hypertrophy of the right ventricle* sometimes. 3. *Irritation* set up by certain conditions of the air inspired, or by morbid formations in the lungs. 4. Various pulmonary affections which *interfere with the capillary circulation* in some parts of the lungs, in consequence of which the vessels of other parts become overloaded. 5. *Inflammatory* pulmonary diseases, of which congestion is the first stage, and which it often accompanies. 6. *Obstruction to the entrance of air* into the lungs during inspiration, and hence rarefaction of the residual air, with diminished pressure on the vessels. The causes of

*mechanical* hyperæmia are:—1. Some *cardiac disease* in the great majority of cases, interfering with the passage of blood through the left cavities of the heart, especially mitral disease, but probably also a feeble and dilated condition of the left ventricle. 2. Very rarely a *tumour pressing on the pulmonary veins*. *Passive* congestion is most frequently observed in connection with low fevers and other conditions which greatly depress the action of the heart, and disturb the capillary circulation, especially in aged and feeble individuals; it is seen chiefly in dependent parts, usually the bases and posterior portions of the lungs, on account of the influence of gravitation, and the congestion is then termed *hypostatic*. Probably it may also arise in connection with a very weak dilated right ventricle.

*Pulmonary œdema* is as a rule the result of long-continued or intense congestion from any cause, but especially when this is associated with cardiac disease. It may be but a part of general dropsy.

*Hæmorrhage* into the lungs may occur under the following circumstances:—1. As a result of *congestion*. 2. From the lodgment of an *embolus* in one of the branches of the pulmonary artery. This embolus is usually associated with cardiac disease, and is detached from a clot in the right ventricle, but it may be conveyed from more distant parts. 3. From a diseased condition of the branches of the *pulmonary artery*. This often materially aids in the causation of pulmonary hæmorrhage. 4. As a consequence of *injury* affecting the lungs. 5. In connection with *pulmonary diseases*, such as morbid growths, cavities, or ulceration in the lungs, particularly in cases of phthisis or cancer. 6. Owing to some *morbid condition of the blood*, such as that associated with scurvy, purpura, or malignant fevers.

**ANATOMICAL CHARACTERS.**—Hyperæmia of the lung gives rise to a more or less deep-red colour, which may become bluish, purple, livid, or blackish-red. The part affected is enlarged, relaxed, and moist; crepitates imperfectly; and a quantity of aerated bloody fluid escapes from its tissue on section. Pieces of congested lung float in water. In extreme cases the vesicular structure is scarcely apparent, and the tissue breaks down very readily, this condition being termed *splenification*. Hypostatic congestion may end in hypostatic pneumonia.

Œdema is necessarily chiefly observed in dependent parts, and is always present, more or less, when the lungs are congested. The lungs are enlarged, tense, and do not collapse when the chest is opened; while they have a peculiar feel, and after pressure retain the impression of the finger for a time. The tissues are very moist, and on section a large quantity of serous fluid escapes, either red or colourless according as the œdema is associated with congestion or not, and it may or may not be frothy. The lungs are either congested, or pale and anæmic.

Hæmorrhage is described as occurring under four forms, namely:—1. *Circumscribed* or *nodular*—*Hæmorrhagic infarction* or *pulmonary apoplexy*. 2. *Diffuse* or *true pulmonary hæmorrhage*. 3. *Interlobular*. 4. *Petechial*, in connection with blood-diseases. The last two are very rare, and do not call for further notice.

Hæmorrhagic infarction is due to embolism, and the blood comes from the capillaries of the pulmonary artery, collecting within, as well as outside the alveoli and minute bronchi, but there is no laceration of tissue. The size of an accumulation varies considerably, depending upon that of the branch of artery obstructed, and it may measure from half an inch to four inches or more in diameter. When situated in the interior



of the lung, the infarction is large; when near the surface it is small and wedge-shaped or pyramidal, with the base projecting outwards a little beyond the surface. The most frequent seats of infarction are the interior of the lower lobe, and the vicinity of the root of the lung, but at the same time there may be others more superficial, and they are often numerous. Each hæmorrhage is circumscribed and defined, and it may only correspond to a single lobule, but the surrounding tissue is congested and œdematous. The infarctions feel very firm and hard; a section presents a solid, airless, slightly-granulated, dark-red or blackish appearance; while coagulated blood can often be scraped away, and then the lung-structure may become perceptible.

Apoplectic clots in the lungs are liable to the usual changes, and the blood may ultimately be completely removed, the tissues being restored to their normal condition. In many cases a permanent, blackish, pigmented knot is left. Pneumonia is sometimes excited, or an abscess may form, the clot softening in the centre: or it may undergo caseous or calcareous degeneration, and become subsequently encapsuled.

In the *diffuse* form of hæmorrhage a vessel of some size gives way, the lung-tissue being lacerated, and an irregular potential cavity being formed, varying in size, and containing a mixture of fluid and clotted blood. The pleura may rupture, the blood consequently escaping into its cavity.

In cases of hæmorrhage into the lung in connection with hæmoptysis from phthisis, Dr. Reginald Thompson\* has described two classes of anatomical appearances, which deserve notice. The first consist of well-defined circumscribed nodules, of an oval or round form, in colour varying, according to their age, from blood-red to yellowish-red and white, in size ranging from  $\frac{1}{8}$  of an inch to an inch, but being generally about  $\frac{1}{2}$  an inch in diameter, marked in their centre with the openings of two or more bronchioles which are usually surrounded or spotted with pigment; they have a slightly granular surface when red, but when white are firm, tough, unyielding, and hard. They show a marked tendency to separate around their circumference from the lung-tissue which surrounds them. These nodules are usually regarded as pneumonic, but Dr. Thompson believes that they are altered blood. Moreover, he considers that they are not the results of a hæmorrhage *in situ*, for they may be found in the lung opposite to that which is the source of the bleeding. Nor are they due to the simple gravitation of the blood dribbling downwards, as they are found in the apex of the lung. The localities which they occupy are sometimes the upper lobe, where they run into each other and occasionally form rather large masses; the base, being often found close to the periphery of the diaphragmatic surface, and sometimes only in a narrow region corresponding to the arched part of the diaphragm; and the anterior axillary border close to the periphery, in the region of the nipple, or between the third and fifth ribs. Dr. Thompson regards these nodules as due to the forcible impaction of clotted blood, driven from a distance into the bronchi and alveoli by forced inspiratory efforts. The second group of appearances arise from hæmorrhage *in situ*, of some violence, and producing laceration and contusion of the lung-tissue. They consist of irregular blackened patches, sometimes of considerable size, as much as two inches and a half across, which may be found in the upper part

\* "Medico-Chirurgical Transactions," Vol. LXI. p. 253.

of the lower lobes, or irregularly placed in the upper lobe. They are formed of calcareous matter, loosely coherent, mingled with the black pigment of old blood, and surrounded with a defined but irregular envelope of some thickness, which is deeply pigmented with the same black granules. Occasionally the yellow colouring matter, which is often found in old blood-clots, is observed scattered about.

A few remarks may be made here with reference to a condition known as *brown induration of the lung*. This follows long-continued pulmonary congestion, especially that due to mitral disease, and it is characterized by the accumulation of granular yellowish pigment, probably of the nature of hæmatoidine, in enlarged epithelial and granular cells, which collect in the alveoli; accompanied with varicose dilatation of the capillaries; and probably thickening of the alveolar walls. The pigment may become black, and may finally be found free. The lungs are increased in bulk, and do not collapse; they feel heavy, compact, and inelastic; and present a yellowish tint, passing into brown or reddish-brown. On section, in addition to the general change in colour, red spots are seen, shading into black, and a brownish fluid may be expressed. Various degrees of the change are observed, and the extent of tissue affected differs much in different cases, while infarctions are often present at the same time.

**SYMPTOMS.**—The symptoms resulting from pulmonary congestion and its consequences are not easy to define, as they are usually only exacerbations of previously-existing phenomena. One of the most obvious is dyspnoea, either coming on for the first time, or being more intense than before, and sometimes amounting to orthopnoea. A feeling of tightness or oppression is often experienced across the chest, but pain is generally absent. There is more or less cough, which in pulmonary cedema is attended with very profuse watery expectoration; and when hæmorrhage takes place, a variable quantity of blood is usually discharged, which may have a dull-brownish or bister-colour, or be almost black. If a clot excites inflammation, pyrexia and other symptoms indicating this complication will supervene.

**PHYSICAL SIGNS.**—1. *Respiratory movements* are often diminished. 2. *Percussion-sound* may be at first abnormally clear in pulmonary congestion; but afterwards becomes more or less deficient at the bases. There may be localized dulness in connection with hæmorrhage. 3. *Respiratory sounds* are usually weak and harsh; over the seat of hæmorrhage they may be bronchial. 4. *Cedema* is characterized by abundant, small, liquid bubbling *râles*; localized moist *râles* may also be perceptible over the part of the chest corresponding to pulmonary hæmorrhage, and here signs of pneumonia or abscess may ultimately be detected. 5. *Vocal fremitus* and *resonance* may be increased or diminished, but are quite unreliable.

**PROGNOSIS.**—As a rule the affections now under discussion are serious, and they often increase the gravity of the prognosis considerably, because they complicate other dangerous conditions.

**TREATMENT.**—Much will depend on the exact nature and extent of the morbid changes; and on the conditions which cause them, or with which they are associated. Free dry-cupping of the chest is often very useful in pulmonary congestion, and sometimes local removal of blood is indicated. It is important to attend to posture; and to keep the patient at rest. Good diet, *tonics*, and *stimulants* are frequently necessary. Care must be taken in cases of cedema that the fluid



not allowed to accumulate in the lungs, and cough must be encouraged. In diffuse hæmorrhage *astringents* are required. Remedies which act upon the heart and vessels may be of much service, especially *digitalis*.

---

## CHAPTER X.

### PNEUMONIA—INFLAMMATION OF THE LUNGS.

INFLAMMATION of the tissues of the lungs occurs under different forms, each of which requires separate consideration.

#### I. ACUTE CROUPOUS PNEUMONIA—LOBAR PNEUMONIA.

**ÆTIOLOGY.—Predisposing causes.**—1. *Age*. Most cases of acute lobar pneumonia occur between 20 and 30 years of age, but no age is exempt, and the young and old are very liable to suffer. 2. *Sex*. More males are attacked, probably from their greater exposure to the exciting causes. 3. *Social position, habits, and occupation*. Poverty, residence in large towns, intemperance, and occupations involving exposure or over-exertion, predispose to pneumonia. 4. *State of health*. Acute pneumonia is very liable to occur in those who are constitutionally feeble, or who suffer from any lowering chronic or acute disease; as well as during convalescence from the latter. It may, however, attack persons in the most robust health. 5. *Previous attacks* increase the liability to the complaint. 6. *Climate and season*. Those characterized by coldness, rapid changes in temperature, much moisture, or the prevalence of northerly and easterly winds, greatly predispose to attacks of pneumonia.

**Exciting causes.**—1. Most cases of *primary* pneumonia arise from a *cold*, induced by a sudden chill when the body is heated; by exposure to cold or wet; or by a cold draught. 2. *Direct irritation* not infrequently sets up pneumonia, which irritation may be due to the inhalation of very hot or cold air, or of irritating gases; foreign bodies, such as food; blood, especially apoplectic clots; or morbid formations, for example, tubercle, cancer, diphtheritic or croupous exudation. 3. *Injury* to the chest often excites local inflammation, such as contusion, fracture of the ribs, or a perforating wound. It has been stated that violent exertion is occasionally a cause of pneumonia, but this is very doubtful. 4. Pneumonia is frequently *secondary* to various acute affections, especially low fevers and blood-diseases, such as measles, small-pox, typhus, typhoid, pyæmia, and puerperal fever. It is also very apt to arise in the course of chronic blood-diseases, but in these cases there is some other exciting cause, which may be undiscoverable, acting on a depraved system. 5. *Epidemic* pneumonia has been described, due to unfavourable hygienic conditions. The complaint may assume this character in connection with influenza or other epidemic diseases, especially if there is much over-crowding, with deficient ventilation. It is also said to prevail in malarial districts. 6. Intense or long-continued pulmonary congestion is very liable to cause pneumonia, especially that which



results from heart-disease, or the hypostatic congestion which affects dependent parts in old and weak individuals who are confined to bed from any cause—*hypostatic pneumonia*.

Acute primary pneumonia has been regarded as a *specific fever*, of which the pulmonary inflammation is but a local manifestation; and it has even been attributed to the action of a specific organism. Klebs, Koch, Friedlander, and others have described specific micrococci, which have been found in the exudation, in the lymphatics of the lungs, and in blister-fluid. It has also been affirmed that pneumonia has been produced in rabbits by injection of the cultivated cocci, and in mice by inhalation of spray charged with them. Hueter and Ogston would attribute the disease to organisms in the atmosphere, which, according to their view, ordinarily set up inflammation.

**ANATOMICAL CHARACTERS.**—Pneumonia is characterized pathologically by hyperæmia and oedema of the lung-tissue; followed by the formation of a fibrinous exudation in the interior of the air-vesicles and minute bronchi, which undergoes various changes. It is necessary to describe the appearances presented at different stages.

Dr. Stokes has described a *preliminary stage*, characterized by brilliant arterial redness of the pulmonary tissue, with abnormal dryness, but no other alterations. Those usually seen, however, are as follows:—

**First or Engorgement-stage.**—Colour is dark-red, reddish-brown, violet, or livid; not uniform, but mottled. The lung feels heavy; and the affected part is firmer, more resisting, and less elastic than in health, retaining impressions of the finger, and crepitating imperfectly. On section a quantity of reddish or brown bloody serum escapes, which is more or less aerated, and somewhat viscid. The lung-tissue is still perceptible, and pieces of the organ float in water. Consistence is diminished, the tissue being more easily torn.

**Second or Exudation-stage.—Red hepatization.**—Colour is more uniform and dull-reddish. Weight is remarkably increased, and the lung is sometimes evidently distended, being marked by the ribs. The affected tissue feels solid and firm, absolutely inelastic, and non-crepitant. A section presents a dull reddish-brown colour, with some greyish variegation, and is opaque, but the hue becomes brighter after exposure. Very little fluid escapes, often none except on pressure, what is then obtained being thick, dirty, sanguineous, and non-aerated. A characteristic granular appearance is usually visible, especially on tearing the affected part, but it is less marked in children, or when the exudation is of soft consistence, as is the case in low fevers, and when the disease attacks old people. All trace of lung-texture has disappeared, and the tissues are very brittle, breaking down easily under pressure. Fragments of the affected part sink in water instantly. The microscope reveals amorphous fibrin, with abundant newly-formed cells, and some granules.

**Third stage.—Grey hepatization.**—In this stage the colour gradually changes, becoming ultimately grey, combined with a greenish or yellowish tint. The granular appearance on section is less distinct or altogether lost; and the lung-tissue becomes more or less soft or pulpy. A quantity of dirty, greyish, almost puriform fluid escapes, either spontaneously, or on pressure or scraping. This stage presents various grades, from slight softening to what is termed *purulent infiltration*. The changes consist in excessive cell-formation, with fatty degeneration and liquefaction of the inflammatory products. In favourable cases

these materials are ultimately either absorbed or expectorated, and the lung-tissue remains unaltered in its structure.

Such being the ordinary course of a case of pneumonia, other pathological *terminations* are observed in some instances, namely:—1. Formation of one or more abscesses, which ultimately may either open into the bronchi, the pus being discharged, and a cavity being left; or communicate with the pleura; or become encapsuled, the contents undergoing cheesy or calcareous changes, and the spaces being finally completely closed up. 2. Gangrene. 3. Caseous degeneration and destruction of lung-tissue. 4. Chronic induration or cirrhosis.

The *right lower lobe* is the most frequent seat of acute pneumonia, but the inflammation may spread through an entire lung, or may involve more or less of both organs. Sometimes it begins in the middle of the upper lobe, or at the apex; and in old or cachectic subjects it often extends from above downwards.

The parts of the lungs which are not pneumonic frequently present a congested and œdematous appearance; while more or less bronchitis is present. Pleuritic exudation is commonly observed, but not often abundant effusion. The right cavities of the heart and the general venous system tend to be overloaded, the various organs being congested; and fibrinous coagula are liable to form in the heart and vessels, the blood being exceeding rich in fibrinogenous elements, exhibiting the “buffy” coat markedly.

**SYMPTOMS.**—In some cases an attack of pneumonia is preceded for a short time by *premonitory* symptoms, indicating general indisposition. *Primary* pneumonia usually sets in very suddenly, the invasion being attended with a single, severe, and more or less prolonged rigor. There may be great prostration, with pyrexia; severe vomiting; or nervous symptoms, namely, headache, delirium, restlessness, stupor, or in children convulsions. The symptoms of the established disease are *local* and *general*.

*Local.* Pain in the side is usually complained of, occasionally commencing simultaneously with the rigor, or even preceding it, but as a rule only setting in after a variable interval has elapsed. Its seat is generally about the mammary region; and though considerable in degree, it is not very intense in most cases, at all events for any lengthened period, being tolerably easily relieved. In character it is commonly stabbing or piercing, being increased by a deep breath and by cough. Tenderness is often observed, and sometimes hyperæsthesia of the skin. Dyspnoea is an early and prominent symptom, as evidenced by the sensations of the patient; by the rapidity of the breathing, which, however, is abrupt and shallow; by the working of the nostrils; and by difficulty of speech. The pulse-respiration ratio is greatly disturbed, the respirations usually ranging from 30 to 60, or occasionally even reaching 80 per minute. There may be orthopnoea. Cough also commences very soon. It does not come on in violent paroxysms, but is short and hacking, being often of a spasmodic character and difficult to repress, especially when the patient is made to breathe deeply or to sit up, while the act causes much distress. Expectoration speedily ensues, the sputa presenting peculiar characters. They are scarcely at all frothy, but exceedingly viscid and adhesive, so that they are discharged with much difficulty, often having to be wiped from the mouth, and not falling out when the vessel which receives them is overturned. They present a “rusty” colour, or various tints of red, owing to admixture of blood,



but as the disease progresses changes of colour are observed, passing through different shades of yellow, until finally the expectoration becomes merely bronchitic in character. The microscope reveals epithelium; blood-discs; so-called granular or exudation-cells; sometimes minute ramifying coagula, which may be evident to the naked eye as small structureless masses in the sputa; and later on pigment-cells or free pigment; abundant granules and oil-globules; free nuclei; or pus-cells. Chemical examination reveals the presence of mucin; albumen; often a little sugar; salts, especially chlorides; and, it is said, occasionally a special acid. The expired air may be cool, and is deficient in carbonic anhydride.

Such being the ordinary local symptoms of acute pneumonia, it must be borne in mind that considerable deviations may be noticed, dependent upon the age and condition of the patient; the portion and extent of lung-tissue affected; the type and course of the pneumonia; or upon the disease being secondary. Pain and other local symptoms are sometimes very slight or absent—*latent pneumonia*; while the sputa may be absent or merely bronchitic, or in low cases sometimes present the appearance of a dark, offensive, thin fluid, resembling liquorice or prune-juice. Occasionally they are tinged with bile.

*General.* These may be summed up as high pyrexia, with great depression and prostration.

The skin in pneumonia soon becomes exceedingly hot and dry, having a burning acrid feel. Sometimes perspiration takes place, but the patient experiences no relief. The temperature rises with great rapidity to 102°, 103°, 105°, or even higher. The maximum is generally reached on the second or third day, but the temperature may continue to ascend until near the termination of the case. It has been known to rise to 107° in cases which have recovered; and in fatal cases it has attained 109·4°. In a large number of instances the temperature does not go beyond 103° or 104°. The daily variations are usually as follows:—The temperature is lowest in early morning, and begins to rise in the forenoon or soon after, attaining its maximum early in the evening; it then falls, but in some cases a slight rise is again observed at midnight, after which a gradual fall takes place. The remission ranges from  $\frac{3}{4}$ ° to 2·5°, but is seldom more than 1·8°. Usually it ceases altogether a day or two before the crisis occurs. In rare instances, when pneumonia is associated with intermittent fever, the temperature becomes quite normal in the mornings—*intermittent pneumonia*. An extension of inflammation or a relapse will disturb its normal course. There is usually considerable flushing of the cheeks in pneumonia, which may be more marked on the affected side; sometimes a tendency to duskiness or lividity is noticed; or the face may present a yellowish earthy tint. The expression is either painful and anxious, or heavy and stupid. Herpes is frequently observed on the face about the second or third day.

The pulse is usually frequent, being as a rule proportionate to the extent of the pneumonia. It ranges generally from 90 to 120, but may be much above this. At first strong, full, and incompressible, it subsequently becomes weak, small, and yielding, or sometimes intermittent or irregular. The sphygmograph affords useful indications as to the characters of the pulse.

A prominent symptom of pneumonia in most instances is the great and evident prostration and feebleness of the patient. The position



assumed is generally dorsal, with the head rather high, and it is often only with difficulty that the patient can be made to sit up.

The digestive organs present to a marked degree the ordinary symptoms associated with pyrexia. The tongue is much furred, and tends to be dry; and the lips frequently become cracked. As occasional and usually unfavourable symptoms there may be dysphagia; severe vomiting; jaundice with enlarged liver; or diarrhoea. The ordinary cerebral symptoms are headache, sleeplessness, and restlessness, often combined with slight nocturnal delirium. The urine, in addition to being highly febrile, frequently contains a little albumen, and chlorides are strikingly deficient or entirely absent.

In some cases the symptoms assume an adynamic character, indicated by a dry brown tongue, with sordes on the lips and teeth; and low nervous phenomena, such as delirium, stupor, coma, convulsions, twitchings and tremors, and disorder of the special senses—*typhoid pneumonia*. This course of events is particularly apt to occur if the patient is old, very weak, or intemperate; if the disease is secondary to certain acute and chronic affections, or is attended with high pyrexia; or if it terminates in suppuration or gangrene, which causes extreme prostration. In drunkards the symptoms at first often resemble those of delirium tremens, followed by collapse. Occasionally they simulate symptoms indicative of mania or cerebral inflammation. The formation of pus is usually attended with severe rigors and increase of pyrexia. If it collects in an abscess, it may be suddenly discharged, along with fragments of lung-tissue.

Sometimes distinct signs of cyanosis supervene in pneumonia, with distension of the right side of the heart and of the venous system, and the formation of coagula in the pulmonary vessels.

**PHYSICAL SIGNS.**—I. **Stokes's stage.** At this time the only sign is a harshness and roughness of the breath-sounds over the involved portion of lung, these being usually exaggerated in intensity. I have had several opportunities of verifying that this stage can be recognized.

II. **Engorgement-stage.**—1. *Respiratory movements* are deficient, partly on account of pain. 2. *Vocal fremitus* is often increased. 3. *Percussion-sound* is usually not much altered, but may be abnormally clear, or slightly deficient in resonance. 4. *Respiratory sounds* are harsh and weak, or occasionally somewhat bronchial. 5. The principal physical sign is the *true crepitant râle*, which is heard over the affected portion of lung.

III. **Red hepatization-stage.**—1. There may be slight *enlargement* of the side. 2. *Movements* are greatly impaired, especially expansion. 3. *Vocal fremitus* is in excess. 4. *Percussion* as a rule reveals dullness with increased resistance; sometimes the percussion-note is rather hollow, and of tubular or even amphoric quality. In basic pneumonia a tubular or tympanitic note can sometimes be elicited over the front of the upper part of the chest. 5. The *respiratory sounds* afford one of the most important signs of this stage. Frequently they are typically tubular, dry, high-pitched, whiffing or metallic; sometimes merely blowing or bronchial. 6. *Crepitant rhonchus* is often heard at the confines of the inflamed part. 7. *Vocal* or *cry-resonance* is intensified, high-pitched, sniffing, and metallic. It may be almost ægophonic or pectoriloquous; and occasionally whispering pectoriloquy is observed. 8. There is no *displacement of organs*. The *heart-sounds* are frequently intensified over the affected part.

**IV. Resolution-stage.** The chief additional physical signs of this stage are *reduæ crepitant rhonchus*; or thin *bubbling râles*, either large or small, of ringing or metallic character. The other abnormal signs usually disappear, sometimes with great rapidity, in other cases only slowly and gradually; sometimes they remain permanently. The dullness may subside in patches. Occasionally slight retraction of the chest follows an attack of pneumonia.

The signs just described are commonly observed at one or both bases, but may be noticed at the apex or other parts of the lungs. Variations may be met with, due to the consolidation becoming extreme, the tubes being completely blocked up; to the inflamed part lying deep in the lung; or to other unusual conditions. *Diffuse suppuration* gives rise to abundant, liquid, bubbling râles. *Abscess* and *gangrene* are followed by the signs of a cavity. The signs of pulmonary congestion, bronchitis, or pleurisy are often present along with those of pneumonia. In the unaffected parts of the lungs respiration is exaggerated.

**TERMINATIONS AND DURATION.**—1. In the majority of cases acute pneumonia ends in complete recovery by *resolution*. Usually a marked *crisis* takes place, the temperature falling rapidly to or even below the normal, while the pulse and respirations also diminish in frequency, and the other symptoms speedily abate, convalescence being soon established. This happens usually from the third to the eleventh day, being most frequent about the end of the first week, but not necessarily on odd days, as some have supposed. The crisis is attended either with profuse perspiration; with an abundant discharge of urine, which deposits lithates, oxalates, and phosphates, or sometimes contains blood; or occasionally with diarrhœa, epistaxis and other hæmorrhages, or the development of a skin-eruption. It may be followed by considerable and even fatal collapse. In some cases defervescence takes place by *lysis*, convalescence being protracted. Recovery may gradually ensue even after the termination in gangrene or abscess. A *relapse* sometimes happens. 2. Death may occur, either from asphyxia; or more commonly from collapse and exhaustion. This event may take place even after the crisis. 3. Now and then pneumonia becomes chronic, the exudation remaining unabsorbed, and the symptoms continuing, with irregular fever and loss of flesh. Ultimately a form of phthisis is sometimes set up.

**DIAGNOSIS.**—This subject will be again considered under the general diagnosis of acute lung-affections. At present it is only needful to call attention to the fact that pneumonia often comes on insidiously, and whenever this is probable, the chest should be examined at frequent intervals. This disease may also simulate low fevers; cerebral inflammations; or acute alcoholism.

**PROGNOSIS.**—Different observers have given very conflicting statements as to the rate of mortality in pneumonia, but it must always be looked upon as a serious affection. The chief circumstances which increase the danger are:—very early or advanced age; the female sex; pregnancy; debility from any cause; previous intemperance; the presence of chronic pulmonary, cardiac, or renal disease; extensive adhesions of the pleura or pericardium; the disease being secondary; both lungs being involved, or the whole of one, or its central or upper part; the sputa being very abundant and watery, or like prune-juice, or absent along with signs of accumulation in the lungs; the termination in diffuse suppuration, abscess, or gangrene; the development of typhoid and low nervous symptoms, or of those indicating marked collapse; signs of apnœa; the



existence of serious complications, such as gastro-enteric catarrh or pericarditis; and a low epidemic type.

**TREATMENT.**—Of course it is highly important in treating pneumonia to observe the precautions demanded in the management of all acute pulmonary diseases, but at the same time the sick-room must be well-ventilated. Three main plans of treatment have been adopted in the management of this disease, namely:—1. **Expectant.** 2. **Antiphlogistic.** 3. **Stimulant.** No constant and routine method ought, however, to be followed, but it is essential that each case should be carefully considered in all its details, and the treatment modified accordingly.

1. The **expectant** plan, in which the patient is merely protected against injurious influences, and properly nourished, while symptoms are relieved, the cure of the disease being left to nature, is one which unquestionably may be carried out successfully in many instances; but to apply it to all cases of pneumonia indiscriminately is most injudicious.

2. In the **antiphlogistic** treatment, the chief remedies employed are venesection, or local removal of blood; tartar emetic; calomel and opium; digitalis, aconite, or veratria. It has been satisfactorily proved that venesection is rarely required in the treatment of pneumonia, cases in which this measure might be indicated doing just as well without it, and in a great many instances it would be most injurious. The removal of blood may relieve dyspnoea and diminish fever, but only temporarily. Moderate bleeding is occasionally demanded, in order to avert death from apnoea. Local bleeding cannot subdue the inflammation, but it is useful sometimes for the purpose of mitigating symptoms. Tartar emetic is decidedly a serviceable drug in pneumonia, when the patient is strong and plethoric. It should not be given in large doses, from gr.  $\frac{1}{4}$  to  $\frac{1}{2}$  every four hours being quite sufficient for an adult, and it may be combined with compound tincture of camphor and hydrocyanic acid. Of the use in this disease of the other drugs mentioned I have no experience.

3. Many practitioners have recourse to the **stimulant** treatment, giving large quantities of alcohol, in the form of wine or brandy, along with ammonia, chloric or sulphuric ether, camphor, and similar remedies. The routine employment of these agents, however, is to be equally deprecated with those of the opposite class, for they are often unnecessary, and may do more harm than good. In many instances stimulants are most valuable, the quantity to be given depending upon the nature of the case, and their usefulness or the reverse being judged of by the effects produced. It is a good rule to try them carefully in doubtful cases. The main indications for stimulants are the occurrence of delirium, if not associated with vascular excitement; a very rapid, weak, or dicrotic pulse; any signs of adynamia or collapse, with low nervous symptoms; the patient being old or feeble; and the pneumonia being secondary. In all low forms of the disease the only chance of recovery lies in free stimulation, eight to twenty ounces or more of brandy being often required in the twenty-four hours, and if this quantity is needed there ought to be no hesitation about giving it. At the same time full doses of carbonate of ammonia with decoction of bark, spirits of chloroform, ether, camphor, musk, and such remedies must be administered. In some cases quinine with iron is useful; and in very adynamic conditions oil of turpentine has been recommended, which may be introduced by enema. Phosphorus has also been given in low forms of pneumonia. In most instances it is advisable to give a



little wine or brandy after the crisis, as there is often much exhaustion at this time.

Attention to *diet* is always of considerable moment in acute pneumonia. In all cases a good quantity of beef-tea and milk should be administered at regular intervals, and abundant support is often needed. Cooling drinks are useful; or some saline drink may be freely allowed.

4. **Local treatment.**—The application of cold to the chest has been advocated in the treatment of pneumonia, either by means of cold compresses frequently changed, or of ice-bags covered with muslin. Great caution must be exercised in their employment. Hot fomentations or poultices, either alone or with anodynes; turpentine fomentations; or sinapisms are useful for the relief of pain. Blisters are only needed in the advanced stage of pneumonia as a rule, and not even then if absorption is going on satisfactorily.

5. **Symptomatic treatment.**—Opiates are frequently required to relieve pain, to procure sleep, or to alleviate distressing cough. They must be given with due caution. Hypodermic injection of morphia is often most valuable. Hydrate of chloral is a useful substitute in many cases, as well as other *sedatives* and *narcotics*. If there is high pyrexia, full doses of quinine should be tried, or it might be requisite to have recourse to cold baths. The *anti-pyretic* treatment of acute pneumonia has been advocated as a special method. Should expectoration be very difficult on account of excessive viscosity of the sputa, alkalies are recommended. Chloride of ammonium, tincture of squills, and decoction of senega are also useful under such circumstances, especially during the later stages.

6. Every care is necessary during **convalescence** after pneumonia, and the patient should be kept under observation until thoroughly restored to health. *Tonics* are serviceable at this time, with good diet; and cod-liver oil proves beneficial in some cases.

## II. CATARRHAL PNEUMONIA—DISSEMINATED OR LOBULAR PNEUMONIA—BRONCHO-PNEUMONIA.

**ÆTIOLOGY.**—This variety of lung-inflammation may assume an *acute* or *chronic* form, and in the great majority of cases it arises in the course of bronchitis, being either the result of direct extension of inflammation along the minute bronchi to the air-vesicles; or more commonly being set up in collapsed lobules. *Acute catarrhal pneumonia* is by far most prevalent amongst children, being particularly observed in connection with whooping-cough, measles, diphtheria, and influenza, but it may arise independently of these affections. The complaint is predisposed to by debility; by breathing impure air; and by a long-continued recumbent posture. There is reason to believe that the pneumonia occurring in the aged and feeble, and in those dying from various acute or chronic diseases, is not infrequently of this nature. As a more or less *chronic* affection, resulting from gradual extension of bronchial catarrh into the alveoli, catarrhal pneumonia is believed by many pathologists to originate a large proportion of cases of phthisis. The disease may also be set up in connection with dilated bronchi.

**PATHOLOGY AND ANATOMICAL CHARACTERS.**—There is no fibrinous exudation in catarrhal pneumonia, such as is characteristic of the croupous variety, but merely a proliferation of the epithelial elements lining the alveoli, which become so abundant as to fill and distend these

spaces. In favourable cases the new cells undergo liquefaction, and are absorbed or expectorated. Sometimes abscesses are formed; or caseous degeneration ensues, ultimately leading to destruction of the lung-tissue, or to tuberculosis. Chronic interstitial pneumonia may also be set up. When catarrhal pneumonia follows lobular collapse, the morbid appearances are usually confined to isolated lobules, but by their coalescence large tracts of the pulmonary tissue may become involved, especially at the bases and along the posterior borders of the lungs. Generally they are accompanied with bronchitis; congestion and œdema; or collapsed lobules. The inflamed lobules are disseminated irregularly through both lungs, being most abundant towards the bases, along the lower free border, and at the surface. They vary in size considerably, and when superficial have a pyramidal or wedge-like form, with the base directed outwards, projecting somewhat beyond the surface. They feel like firm solid knots, but are in reality friable, breaking down readily under pressure. A section presents a more or less greyish-yellow colour, gradually fading into surrounding congestion, and it has also generally a granular aspect. A whitish, opaque, non-frothy fluid can be scraped or pressed from the surface, containing abundant cells, many of which resemble under the microscope pus- and mucus-corpuscles. Within the affected lobules there are often small dilated bronchi, containing a purulent fluid. The inflamed parts sink instantly in water. The appearances just described are those met with when the inflammatory process is well-established, but gradual transitions are observed from merely collapsed lobules.

When the inflammation is independent of collapse, very numerous, small, ill-defined, whitish-yellow spots are seen scattered through congested and œdematous lung-tissue, only slightly granular, and yielding an opaque milky fluid on pressure. In some parts little cavities form, containing a pus-like matter, but many observers are of opinion that this has gravitated into the minute bronchi or air-vesicles, or has been drawn in during inspiration.

**SYMPTOMS.**—Usually occurring in the course of some other complaint, especially bronchitis, the symptoms of acute catarrhal pneumonia may set in very speedily, as in measles; or gradually, as in whooping-cough. Generally they are merely modifications of previously-existing clinical phenomena. Very rarely is the onset indicated by any rigors or other marked premonitory symptoms, such as are observed in croupous pneumonia. Pyrexia is a most important sign, the temperature rising often to 103°, 104°, or 105°; the remissions, however, are considerable, and irregular as to time, while renewed exacerbations are liable to occur after the temperature has become normal. The skin often perspires freely, and is not pungent or burning to the touch. The pulse increases in frequency, but soon tends to become feeble or irregular. The *local* symptoms, when the complaint follows bronchitis, are increased dyspnoea, the respirations being exceedingly frequent; a change in the characters of the cough, which often becomes short, harsh, hacking, and painful, the child endeavouring to repress the act, and presenting an expression of pain, or crying on account of the suffering; and diminished expectoration, the sputa being scarcely ever “rusty.” *Physical signs* are exceedingly uncertain and ill-defined. In the parts corresponding to the consolidated portions of lung there may be increased vocal fremitus; deficient resonance; bronchial breathing; small, scattered, crepitant or crackling, and sometimes ringing râles; and bronchophony.

The *course* of catarrhal pneumonia may be extremely acute and rapid ; or sub-acute. In the former class of cases there is generally great restlessness and anxiety ; or the patient may soon fall into a stupid and apathetic state. Signs of cyanosis are common. Loss of strength and emaciation are prominent characters, the latter being especially marked in the less rapid cases. The subsidence of the disease in cases of recovery is usually very gradual and protracted, there being no crisis, but an irregular defervescence by lysis. As already mentioned, catarrhal pneumonia may lead to permanent destructive changes in the lungs.

TREATMENT.—All lowering measures are decidedly injurious in cases of catarrhal pneumonia. Ipecacuanha wine is useful, with *salines*, when it is associated with bronchitis ; or ammonia and senega may be given if there is much debility. Abundant nourishment is required, with alcoholic stimulants in many cases. *Emetics* are sometimes serviceable, to aid in unloading the lungs. The continued application of cold compresses to the chest has been strongly recommended. Sinapisms are often of much benefit. Great care is required during convalescence ; and *tonics*, cod-liver oil, good diet, with wine, are indicated at this time.

### III. CHRONIC OR INTERSTITIAL PNEUMONIA.—CIRRHOSIS OF LUNG.—FIBROID PHTHISIS.—FIBROID DEGENERATION.—INDURATION WITH DILATED BRONCHI.

ÆTIOLOGY AND PATHOLOGY.—The forms of pneumonia already described may become more or less chronic, but the condition now under consideration is essentially of this character, and is one in which the affected portion of the lung becomes greatly contracted and indurated, as well as much pigmented, the air-vesicles being more or less obliterated, and the bronchial tubes usually dilated. These changes are generally regarded as being partly due to proliferation of the normal interlobular and sub-pleural connective-tissue, and partly to the formation of a nuclear growth, which develops into extensive tracts of fibroid tissue : but some pathologists look upon them as the result of a chronic inflammatory process, or of a fibroid change affecting the walls of the alveoli themselves.

There can be no doubt but that in the great majority of cases interstitial pneumonia is *secondary* to some previous pulmonary affection, being set up in consequence of long-continued irritation. The conditions of which it may thus be a sequel are :—1. Acute croupous pneumonia very rarely. 2. Catarrhal pneumonia frequently. 3. Dilatation of the bronchi, though Dr. Wilson Fox thinks that the fibroid change is then preceded by catarrhal pneumonia. 4. Collapse or compression of the lung. 5. Pleurisy, but it is doubtful whether the change can then extend to any depth, unless pneumonia precedes it. 6. Bronchial irritation from inhalation of mineral and other particles, such as steel, coal or stone-dust, or cotton. 7. Various local pulmonary lesions, such as the formation of tubercle or cancer ; phthisical cavities ; pulmonary hæmorrhage or abscess ; or injury to the lung. In these conditions the morbid process is localized, and may really be of a curative nature.

Some pathologists, however, consider that interstitial pneumonia is in some instances essentially *primary*, being, as some suppose, the result of a chronic inflammatory process in the interstitial tissue ; or, as others believe, a direct, idiopathic, fibroid change, degeneration, or substitution in the walls of the alveoli, quite independent of inflammation, which



process tends to spread through the lung. No cases bearing out this view have ever come under my own notice.

It is necessary to allude to the relation of dilated bronchi to chronic pneumonia. Doubtless in many cases this dilatation is secondary to the induration; but there is every reason to believe that the former is sometimes the original morbid condition, and gives rise to the fibroid change.

**ANATOMICAL CHARACTERS.**—In the early stage of interstitial pneumonia the pulmonary tissue is congested, but it afterwards becomes paler, and may exhibit extensive tracts of a homogeneous-looking, nucleated substance. When the process is advanced the appearances are very characteristic. The lung is contracted and shrunken; while its tissue is hard and dense, cannot be torn, and creaks on being cut. A section is smooth, dry, and pigmented, often presenting a marbled grey aspect; while fibrous bands or masses may be seen traversing the surface, some of the former being probably obliterated and thickened bronchi or blood-vessels. The vesicular tissue is destroyed, but many of the bronchi are usually dilated. The fibrous growths may ultimately become caseous.

The extent of lung-structure involved varies considerably. The change may be limited at first to the bronchi and the tissue immediately surrounding them; or it may only be visible around morbid deposits or cavities. A peculiar feature of interest is that the condition is usually limited to one lung, which it may affect throughout, or be confined to its base, apex, or middle part.

The pleura is generally thickened, sometimes extremely so, and its surfaces are adherent. The lobes of the lung are also often united by dense fibrous tissue. Emphysema is common in unaffected parts of the lungs; and other morbid conditions are frequently seen, of which the chronic pneumonia is a sequel.

**SYMPTOMS.**—Interstitial pneumonia runs a very chronic course, and its symptoms at first are indefinite, but when it is fully-established well-marked clinical characters may be present. The *local* symptoms include dragging pains about the sides; shortness of breath; and cough, which is often irritable, but at the same time difficult and ineffectual, or it comes on in fits, attended with the expectoration characteristic of dilated bronchi. The complaint is often attended with *general* symptoms, namely, very gradual loss of flesh and strength, anæmia, and sometimes night-sweats; but pyrexia is absent as a rule, or it is but slight. After a time signs of obstructed circulation in the right side of the heart and venous system may supervene.

**PHYSICAL SIGNS.**—These indicate dense consolidation and contraction of the lung-tissue; which may be combined with signs of cavities due to enlarged bronchi, or with other conditions. 1. The chest is more or less *retracted* on the affected side, often to an extreme degree. 2. *Movement* is deficient or absent. 3. *Vocal fremitus* may be increased or diminished. 4. *Percussion* gives a hard, wooden, high-pitched sound, with marked resistance. Occasionally the sound is tubular in some parts. 5. *Respiration sounds* differ in different parts, being weak or absent, bronchial, tubular, or occasionally cavernous, owing to the presence of dilated bronchi or cavities. After a cough the breath-sounds are frequently heard where previously absent. 6. Various *râles* may be audible in the dilated bronchi. 7. *Vocal resonance* is variable, being deficient, bronchophonic, or occasionally pectoriloquous. 8. The heart is often displaced towards the affected side; the opposite lung is

enlarged and encroaches in this direction ; and the diaphragm, liver, or stomach may be drawn up.

TREATMENT.—The management of chronic interstitial pneumonia is really that of a certain form of phthisis. Nourishing diet is necessary, with *tonics*, iron, and cod-liver oil. Counter-irritation is often useful, especially by means of tincture of iodine. Iodide of potassium has been recommended internally, for the purpose of promoting absorption, but it is of very questionable value for this end. Cough must be alleviated, and expectoration improved, by means of the usual remedies. The patient must be warned against unnecessary exertion if the disease is extensive, as this is sure to bring on shortness of breath.

## CHAPTER XI.

### GANGRENE OF THE LUNG.

ÆTIOLOGY.—The conditions under which gangrene of the pulmonary tissue may arise are the following:—1. As the result of *local disease*, namely, acute or chronic pneumonia, phthisis, cancer, hydatids, or bronchial dilatation. 2. From *obstruction* of one or more of the nutrient vessels by an embolus. 3. In connection with *blood-poisoning*, as after low fevers, pyæmia or septicæmia, glanders, or poisoning by venomous animals. 4. In consequence of *extreme exhaustion*, arising from want of food and bad hygienic conditions, or from disease. 5. In certain *nervous diseases*, pulmonary gangrene being observed occasionally in cases of chronic dementia, chronic softening of the brain, alcoholism, and epilepsy.

ANATOMICAL CHARACTERS.—Pulmonary gangrene is either *circumscribed* or *diffuse*. In the *circumscribed* variety, which is the usual form, the part involved is distinctly defined, but its extent varies much. The size generally ranges from that of a hazel-nut to a walnut, but a considerable portion of a lobe may become gangrenous. The lower lobes, and the superficial parts of the lungs, are most liable to be affected. The gangrenous portion soon becomes moist, softened, pulpy, bluish-green, and extremely fetid ; or it may have a greenish-black core, with broken-down lung-tissue around, a stinking, irritating liquid escaping on pressure. The products may be discharged through a bronchus, leaving a ragged sloughy cavity, often with inflamed tissue around. Vessels frequently traverse this space, but as the blood contained in them has coagulated, hæmorrhage does not take place as a rule. Rarely it communicates with the pleural cavity, or even opens into the subcutaneous cellular tissue, in consequence of adhesions having formed between the contiguous surfaces of the pleura. Subsequently in very exceptional cases a fibrous capsule is developed, the sphacelated portion is expelled, and a cavity secreting healthy pus remains, which may ultimately close up and cicatrize.

The *diffuse* form of pulmonary gangrene does not present any line of demarcation, but runs into, or is mixed up with congested, inflamed, or cedematous lung-tissue. A whole lobe or even the greater part of

a lung may be implicated, being more or less softened, sometimes in a state of pulpiness; of a greenish or brownish-black or black colour; more or less saturated with a dirty greyish-black liquid; and, in short, in the condition of a moist, stinking, putrid slough.

**SYMPTOMS.**—The only symptoms which are characteristic of gangrene of the lung are an extremely foetid and peculiar smell of the breath, especially after a cough; and the expectoration of gangrenous matters, emitting a similar foul odour, and in which fragments of lung-tissue may sometimes be discovered. The former may precede the latter symptom for some days, and it is sometimes only observed at intervals. It must be remembered, however, that the breath may be very foul in some cases of chronic bronchitis, and in connection with dilated bronchi or certain cavities. The sputa subsequently become foul and frothy; partly liquid, partly muco-purulent; often dirty, and brownish or blackish in colour; while they contain gangrenous particles and occasionally fat crystals, and elastic fibres may be detected in some cases. More or less blood is often present, and death may result from hæmorrhage. On standing the sputa separate into layers, and a thick sediment falls. The *general* symptoms in most cases are those of extreme depression, adynamia, and collapse, accompanied with low nervous phenomena, ending in speedy death. If the gangrenous materials are swallowed, severe diarrhœa, with tympanites, is liable to set in. Emboli may be carried from the lung, which originate septic abscesses elsewhere. Occasionally death takes place slowly, preceded by the signs of hectic fever; or very rarely recovery may ensue.

**PHYSICAL SIGNS.**—At first these are merely indistinct breathing, with moist *râles*; followed, if the gangrene is circumscribed, by more or less marked signs of a cavity, containing thin fluid. Extensive bronchitis or pleurisy is often set up, with corresponding signs.

**PROGNOSIS** is necessarily exceeding grave in cases of pulmonary gangrene, the termination being generally fatal.

**TREATMENT.**—The measures to be adopted are to administer abundant nourishment, as well as large quantities of alcoholic stimulants, with ammonia, bark, ether, camphor, mineral acids, or quinine; to use frequent inhalations of creosote, carbolic acid, tar-vapour, or turpentine; to encourage expectoration in every possible way; and to make the patient gargle freely with some *antiseptic*, especially Condyl's fluid, and drink a solution of chlorate of potash or yeast. Various *antiseptics* have been recommended internally, such as carbolic acid or sulpho-carbolates, sulphites, or hypochlorites. Should the disease become chronic, as well as during convalescence in those cases in which recovery ensues, *tonics* are needed, with cod-liver oil, change of air, nutritious diet, and other measures for improving the general health.

---



## CHAPTER XII.

## EMPHYSEMA OF THE LUNGS.

Two primary forms of pulmonary emphysema are met with, named respectively VESICULAR and INTERLOBULAR. *Vesicular* emphysema is characterized by enlargement of air-vesicles, resulting either from their excessive distension, from destruction of the septa, or from both causes combined. *Interlobular* emphysema signifies the presence of air in the sub-pleural and interlobular cellular tissue, which is usually the consequence of rupture of air-vesicles.

## I. VESICULAR EMPHYSEMA.

**ÆTIOLOGY AND PATHOLOGY.**—Within the definition of this morbid condition as above given cases are comprised which differ considerably in their clinical aspects and importance. They may, however, be arranged in four groups, though these are frequently more or less conjoined, namely:—1. *Acute emphysema*, either general or local. 2. *Chronic hypertrophous* or “*large-lunged*.” 3. *Chronic limited*. 4. *Atrophous*, or “*small-lunged*.” It will be convenient first to consider their ætiology as a whole; and then to indicate the special causation of each individual form.

**Immediate, direct, or determining causes.**—1. *Inspiratory theory*. According to this view emphysema is the result of excessive or long-continued distension of the air-vesicles during inspiration. Thus it is supposed that general emphysema may arise as a consequence of diminished expiratory force, such as that which accompanies the loss of elasticity in the lungs and chest-walls in old age, the inspiratory force remaining unimpaired. Hence the lungs are kept constantly distended, the more so in proportion to the vigour with which inspiration is performed. Again, when portions of the lungs are from any cause, such as pleuritic adhesions, collapse, or consolidation, rendered partially or entirely incapable of expansion, should the chest still enlarge to the usual extent during inspiration, the air which ought to enter these unused portions passes into other parts, and stretches their vesicles unduly. This is named *vicarious* emphysema. Dr. C. J. B. Williams believes that in this way emphysema is originated in bronchitis, the secretions formed or the thickened mucous membrane obstructing some of the bronchi, and preventing the air from entering the corresponding vesicles, whilst those which are adjacent, and have free tubes communicating with them, receive an excessive amount of air. On the other hand, Laennec held the view that the vesicles terminating the obstructed bronchi became themselves dilated, in consequence of air entering during inspiration which cannot be forced out during expiration; hence the emphysema was termed *substantive*. It has been argued against this theory that expiration is a more powerful act than inspiration; to which it is replied that a forced expiratory effort has very little influence in emptying the air-vesicles, especially if the smaller tubes are obstructed.

2. *Expiratory theory.* Sir William Jenner strongly advocates the theory that emphysema is commonly the result of violent expiratory efforts with partial closure of the glottis, such as are carried on during the act of coughing, lifting heavy weights, playing wind-instruments, and various other actions. There are certain parts of the lungs which are much less supported and compressed by the chest-walls and surrounding structures than others, and hence they yield under the pressure of air from within, and become distended. This applies especially to the apices, the anterior margins, and the edges of the bases, particularly the left. The tendency to the development of emphysema in this way is greater in proportion to the degree to which the lung is inflated; to the obstruction against the escape of air through the air-tubes; to the force which is exercised in its attempted expulsion; and to the want of compression and support of the lung-tissue.

Niemeyer laid much stress on the direction of the expiratory force in originating emphysema. He observes, "in all these acts (*i.e.*, coughing, straining, &c.,) contraction of the chest is effected by vigorous upheaval of the diaphragm. The result is the expulsion of a strong current of air from the lower bronchi, the direction of which is obliquely upward, and if the air be prevented from escaping through the larynx, a portion of it, in a compressed state, must be driven into the upper bronchi, whose direction is obliquely downward. By the centrifugal pressure exerted, by the air thus compressed, upon the vesicles of the upper lobes of the lung, and upon the adjacent thoracic wall, the latter become distended as far as it is possible for them to yield."

3. Some pathologists are of opinion that emphysema is due to a *primary nutritive derangement of the walls of the air-vesicles*. Villemin describes a "hypertrophy of the elements of the vesicular membrane, causing an extension of this, and an increase in the capacity of the vesicles." As a secondary result of emphysema, nutritive changes in the walls of the vesicles are constantly seen; and should these be impaired in their resisting power, they are far more liable to become distended by any force acting upon them from within. Hence in old persons one attack of bronchitis will often set up a considerable amount of emphysema; and when chronic bronchitis or pulmonary congestion has existed for a length of time, the resulting alterations in structure render the vesicles much more liable to become distended. Atrophous emphysema is the consequence of a *primary degeneration*, the partitions wasting and disappearing, several vesicles being thus thrown into one; but in other forms of the complaint, degenerative changes must probably be rather looked upon as *predisposing*, or "*permanence securing*" causes of emphysema, as Sir William Jenner terms them, than as actual determining causes. These morbid changes will be further considered under the ANATOMICAL CHARACTERS.

4. Another theory, that of Freund, is, that in some cases there is a *primary chronic enlargement of the chest*, in consequence of *hypertrophy and rigidity of the cartilages*, and that the lungs become distended and emphysematous in order to fill up the increased space. This must be a very exceptional cause.

No exclusive theory as to the mode of production of emphysema can apply to all cases; and in many undoubtedly more than one of the causes just considered has contributed to the development of the morbid changes.

**Exciting causes.**—1. Emphysema is liable to arise in connection with several pulmonary affections, namely, bronchitis, especially chronic dry catarrh; consolidation, collapse, or destruction of portions of the lung from any cause; or extensive pleuritic adhesions or effusion. 2. Hooping-cough is a common cause in children. 3. Croup and other affections which obstruct the main wind-pipe, and excite much cough at the same time, are often followed by emphysema. 4. Cardiac diseases which lead to permanent congestion of the pulmonary capillaries materially aid in its production, by inducing degenerative changes in the walls of the air-vesicles. 5. Emphysema may be directly originated by playing wind-instruments, excessive effort, lifting heavy weights, straining at stool, climbing hills, and other forms of exertion.

**Predisposing causes.**—Hereditary influence has been regarded as predisposing to emphysema, especially in those cases in which it comes on during early life, but this is by no means certain. The complaint is by far most common in persons advanced in years. Children, however, often suffer, in consequence of their liability to pulmonary affections, and the weakness of their chest-walls. Gouty and fat subjects are said to be predisposed to emphysema.

**Special ætiology.**—The causation of the several forms of emphysema demands brief consideration. What is termed *acute general emphysema*, which is common in connection with extensive bronchitis, is due to an inability to expel the air out of the lungs, in consequence of obstruction of the bronchi, and it therefore accumulates in the small air-tubes and air-vesicles, and inflates the lungs. Many authorities justly object to this condition being called emphysema, and name it *insufflation* or *inspiratory expansion*, because there is no actual disease, but merely an inflation of the lungs, which will subside if the obstruction is speedily removed, but if this does not happen permanent emphysema is liable to become established.

The main difficulty lies in determining the mode of production of *chronic hypertrophous emphysema* following chronic bronchial catarrh. It is regarded by many as being *inspiratory* in its origin, but Sir William Jenner considers it to be the result of forcible *expiration*, and explains its general distribution by the fact that as the lungs and chest enlarge, the relative position of the former to the ribs and intercostal spaces becomes constantly changed, so that successive portions of the lung are brought into correspondence with the spaces, and these being less supported than the parts opposite the ribs, they are unduly distended during cough, and thus the lungs ultimately become more or less emphysematous throughout, though the condition is at the same time most marked at the apices and margins, which are least supported of all.

*Localized emphysema*, whether acute or chronic, is probably as a rule developed during *expiration*. In some instances it is *inspiratory* in its origin, being either *substantive* or *vicarious*.

Ordinary *atrophous emphysema* is merely due to wasting of the septa, which become more or less obliterated, so that the vesicles coalesce to a variable degree; in short, it consists in an atrophy of the lung-tissue, usually observed in old age, along with other atrophic and degenerative changes.

**ANATOMICAL CHARACTERS.**—In *acute general emphysema* the lungs are distended throughout; and do not collapse, or may even bulge out



when the chest is opened. The degree of expansion varies much. The lungs appear pale, the capillaries being stretched, and their network enlarged. The bronchi will be found to be more or less obstructed.

*Chronic hypertrophous emphysema* is also attended with enlargement of the lungs, and on opening the thorax these organs are seen to extend beyond their ordinary limits, often covering the pericardium completely, and they may protrude, or collapse only very imperfectly, this necessarily depending upon the extent of the disease. Though the morbid condition is more or less general, the apices, the anterior borders, and other parts of the lungs which are least supported present the most marked evidences of the change, and the surface is usually more affected than the deeper parts. The emphysematous portions have a peculiar soft feel, compared to that of a "cushion of down," and they retain the impression of the finger, elasticity being impaired. The so-called crepitant sensation of healthy lung is deficient or absent, and on cutting a dull creaking sound is often heard. The affected tissue is pale, bloodless, and dry, but presents irregular spots of black pigment, derived from altered blood contained in obliterated capillaries. The vesicles are seen to be enlarged more or less, varying usually from a hemp-seed to a pea in size, but often many of them are thrown into one, thus giving rise to irregular spaces of considerable size, which are traversed by slender bands, the septa being either visible as slight ridges, or having disappeared entirely. Contiguous lobules may freely communicate, and ultimately nothing may be left but a coarse network. These appearances are best observed after inflating the lung, drying it, and then making a section.

The nature of the changes which the alveolar walls undergo has been much discussed, but there is no reason whatever to suppose that these should be identical in all cases. The violence of the pressure of the air which originates the emphysematous condition may rupture the septa and walls of the air-vesicles directly, but usually their destruction is gradual. They become stretched and atrophied; present perforations varying in size and number; and ultimately only traces of them are seen, or they may disappear altogether. The structural alterations which have been described are the formation of an imperfect fibrous tissue, inducing toughness and thickening, as the result of long-continued congestion (Jenner); or fatty degeneration (Rainey). Dr. Waters of Liverpool considers that there is a primary mal-nutrition of the pulmonary tissue leading to its degeneration, but the exact nature of this he has been unable to ascertain. The elastic and other elementary tissues disappear. The capillaries in the affected part become stretched, narrowed, or obliterated; or some of them may even rupture. Ultimately they are absorbed, and only pigment is left, the remains of the colouring matter of the blood.

In the *localized* variety of emphysema the appearances are confined to certain parts, especially the apices and the anterior and lower edges, being similar to those described as characteristic of the more extensive form.

In true *atrophous* emphysema the lungs are diminished in size; shrink into a very small bulk when the chest is opened; and are very light. The divisions between the lobes are unusually vertical. The pulmonary tissue is pale but much pigmented, dry, and deficient in elasticity. The air-vesicles are enlarged, owing to atrophy of their septa.

Other morbid conditions are often seen in emphysematous lungs, such as bronchitis, collapse in some parts, or, not uncommonly, dilated bronchi. Pleuritic adhesions generally exist. When the emphysema is extensive, the contiguous structures are displaced, and after a time all the organs of the body become the seat of congestion and the changes resulting therefrom. Different statements have been made as to the position of the heart. My own observations would lead me to agree with those who describe this organ as lying with the right border horizontally on the diaphragm, and the apex too low and displaced to the left. Its right cavities become dilated and hypertrophied in course of time.

**SYMPTOMS.**—It is only the *chronic hypertrophous* form of emphysema which leads to any prominent symptoms, and these are chiefly of an indirect character. This condition interferes with the due aëration of the blood, while the pulmonary circulation is obstructed from several causes, but especially on account of the destruction of the capillaries; consequently the right side of the heart is affected, in time becoming the seat of dilatation and hypertrophy, with tricuspid regurgitation; the general venous system also becomes overloaded, the various tissues and organs being then permanently congested, leading to dropsy and important organic changes. The lungs, moreover, are generally the seat of bronchial catarrh or other morbid conditions; and fits of spasmodic asthma, or acute attacks of bronchitis are liable to occur.

Dyspnœa, variable in degree, is the main symptom directly due to emphysema. At first there is merely "shortness of breath" on exertion, especially on going upstairs or up a hill, as well as after a full meal, but ultimately persistent *expiratory* dyspnœa is experienced, though not accompanied with much distress ordinarily, but rather with a sense of discomfort and uneasiness. It is often relieved by pressing the sides, or by lying on the abdomen. After a meal the breathing is worse, especially should the patient be suffering from dyspepsia, which is often the case; and dyspnœa becomes necessarily much aggravated if bronchitis or asthma should set in. The causes of the dyspnœa are the interference with the respiratory movements, owing to the depressed state of the diaphragm, and the rigid state of the chest-walls; the difficulty in expelling the residual air, and the small amount of pure air inhaled; and the actual loss of surface fit for aërating the blood. Cough is frequently present, but is chiefly the result of bronchial catarrh, when it is attended with expectoration; otherwise it is dry. There is no pain in the chest directly dependent upon emphysema; but a dragging sensation or actual pain may be experienced just below the ensiform cartilage.

The remaining symptoms which may be observed in cases of emphysema are indirect. Those due to interference with the circulation will be more appropriately described in connection with heart-diseases. As the result of the increased respiratory efforts the respiratory muscles often hypertrophy; hence the neck appears to be large. The fat may be absorbed, giving rise to emaciation, with strongly-marked features; but some emphysematous patients are more or less obese. The symptoms due to imperfect blood-aëration are similar to those previously described, only that they are gradually produced; and there is generally apathy and languor, with a flabby and relaxed state of the muscles from this cause.

**PHYSICAL SIGNS.**—These will necessarily differ much, according to the extent and variety of the emphysema; and the morbid conditions with



which it is associated. 1. *Shape and size of the chest.* In general *hyper-trophous* emphysema the chest is more or less enlarged bilaterally, either throughout, or only in its upper or lower part. It may assume a permanent inspiratory form, or even go beyond this, becoming "barrel-shaped" and almost circular. There is often a rounding of the chest in front, and of the back behind, but sometimes the change in shape is chiefly observed on one of these aspects. The ribs become more horizontal, and the intercostal spaces wider, in proportion to the enlargement; while the cartilages are frequently quite rigid. In *localized* emphysema there may be corresponding bulging. *Atrophous* emphysema is associated with a small chest, the ribs being very oblique, the lower ones almost vertical. 2. *Respiratory movements.* Expansion is more or less deficient or absent, and there may be merely a general elevation of the chest. Expiration tends to be prolonged. 3. *Percussion* reveals increased area of pulmonary sound, except in atrophous emphysema; and also in most cases hyper-resonance, with fall in pitch, the sound tending towards a tympanitic character, but being frequently more or less muffled. If the distension of the lungs is extreme, there is deficient resonance, with undue resistance. 4. *Respiratory sounds.* The most important change usually noticed is the marked prolongation of the expiratory sound, but this is not observed in the atrophous variety. In pure emphysema the breath-sounds are weak, sometimes remarkably so, but of harsh quality. The extent over which they are heard is increased. 5. A *crepitant rhonchus* is said to be sometimes heard in emphysematous vesicles after a deep inspiration. *Râles* due to bronchial catarrh are often present, especially sonorous and sibilant rhonchi. 6. *Vocal fremitus and resonance* are quite unreliable. As a rule they are deficient. They may be observed over a larger area than usual. 7. There are signs of *displacement of organs* in cases of extensive emphysema, especially of the heart. Epigastric impulse is common. 8. The *veins in the neck* often afford signs of obstruction to the circulation, after the right side of the heart has become affected.

**PROGNOSIS.**—Chronic hypertrophous emphysema is serious in proportion to its extent. It lays the foundation for a very miserable existence in many cases; increases the tendency to bronchial catarrh; and adds greatly to the danger from an acute attack of bronchitis. Once it is thoroughly established, emphysema cannot be cured.

**TREATMENT.**—This part of the subject may be very briefly summed up, inasmuch as the principles on which the treatment of emphysema must be conducted need only be mentioned here, the means for carrying these out being described in other parts of this work. 1. Every precaution must be taken against the occurrence of bronchial catarrh, not only on account of its danger, but because each attack tends to increase the emphysematous condition. Other known causes of emphysema must be avoided. 2. It is very important to attend to the alimentary canal, as a deranged condition of its functions frequently considerably increases the discomfort attending emphysema. 3. The conditions which may be associated with this complaint must be treated as they arise, especially asthma; cardiac diseases; venous congestion and its results, including dropsy; and the pulmonary complications which occur in its course, particularly bronchitis. *Narcotics* must be used with particular caution when the lungs are extensively emphysematous. 4. It is often requisite to improve the general health and the condition of the blood, by the aid of *tonics*, iron, and cod-liver oil; or to treat some constitutional diathesis,



especially gout. 5. Whether there is any curative remedy for emphysema is very questionable. Degenerative changes may to some extent be checked by proper dieting. The administration of strychnine, the use of galvanism, breathing compressed air, and other measures have been stated to produce some improvement in cases of emphysema. A change of climate is often exceedingly beneficial. Usually a mild climate, not too dry, suits best; but it is frequently a matter of personal experience as to the kind of climate which is most suitable. It has been recommended for emphysematous patients to spend the summer in pine-wood regions, where there is a heavy fall of dew.

## II. INTERLOBULAR OR INTERSTITIAL EMPHYSEMA.

**ÆTIOLOGY.**—This is a very rare condition, resulting usually from rupture of the air-vesicles, as a consequence of excessive pressure upon their interior during forcible expiration, the glottis being at the same time much contracted. Thus it may be induced by violent cough, laughing, or straining during defæcation or parturition. Interlobular emphysema is said to be not uncommon in croup; and to occur sometimes as the result of extensive pulmonary collapse. Gangrene or *post-mortem* decomposition may lead to the presence of air in the interlobular tissue of the lung.

**ANATOMICAL CHARACTERS.**—Accumulations of air are seen under the pleura, varying in size, but generally small, and they may form a border of minute vesicles around the lobules. The air can by pressure be displaced along the course of the boundaries of the alveoli. Superficial collections occasionally give way, opening into the pleura, and thus giving rise to pneumothorax; or into the posterior mediastinum, leading to general subcutaneous emphysema.

**SYMPTOMS.**—The only symptom which might lead to the suspicion of interstitial emphysema is the occurrence of severe dyspnœa following one of its causes. It is said that a faint *friction-sound* is sometimes heard. Should pneumothorax or general subcutaneous emphysema be produced, these conditions would be indicated by their usual signs.

**TREATMENT.**—This consists in taking every precaution to prevent the mischief from extending; and in attending to its consequences.

## CHAPTER XIII.

### ASTHMA.

THE use of this term is ambiguous, but it may be employed to include all cases characterized by the occurrence of severe paroxysmal attacks of dyspnœa. Four chief forms may be enumerated, viz. :—1. *Laryngeal*. 2. *Bronchial*, either *spasmodic* or *paralytic*, depending upon spasm or paralysis of the muscular fibres of the bronchial tubes. 3. *Hæmic*, due to an abnormal state of the blood or of the circulation. 4. *Diaphragmatic*, associated with spasm of the diaphragm and other respiratory

muscles. Only *bronchial* and *diaphragmatic* asthma need be discussed in the present chapter.

### I. BRONCHIAL ASTHMA.—SPASMODIC ASTHMA.

**ÆTIOLOGY.**—It is not improbable that in some instances asthmatic attacks depend upon a *paralytic* condition of the bronchial tubes. This may be the effect produced by certain poisonous gases; and by paralysis of the vagus nerve. Ordinarily, however, the paroxysms seem to be *spasmodic* in their origin, being due to spasm of the bronchial muscular fibres excited through the nerves, the irritation being either *centric*, *direct*, or *reflex*. The causes of spasmodic asthma may be arranged as follows:—1. The complaint may be *idiopathic* or *primary*, there being no obvious source of irritation, and the attacks are then sometimes distinctly periodic. 2. *Direct inhalation* of certain materials is a frequent cause, such as fog or smoke; irritating gases and vapours; dust; odiferous emanations from animals, or from vegetable matters, especially hay, ipecacuanha, and certain flowers. The conditions of the atmosphere breathed often materially influence the occurrence of asthmatic attacks, these being especially liable to be brought on by excessively damp or dry air, or by cold easterly winds. Different asthmatic patients present remarkable peculiarities as to the qualities of the air which suits them best, but as a rule a rather moist and relaxing atmosphere is least injurious, and that of elevated and country districts is worse than that of low districts or of large towns and cities. 3. Asthma is very commonly associated with *bronchitis*, *bronchial irritation*, or *emphysema*. 4. *Cardiac diseases* may induce true spasmodic asthma, by giving rise to pulmonary congestion. 5. Asthma may be *gastric* in its origin, following more or less speedily the introduction of food into the stomach. In some cases any kind of food will bring on a fit; in others only indigestible or special articles, such as stimulants or sweets. Usually this form of asthma is looked upon as being due to reflex irritation, but Dr. Hyde Salter was of opinion that it generally depends upon an “offending condition of the blood,” brought about by the introduction into the circulation of deleterious matters during digestion. 6. Various *reflex* sources of irritation may induce asthma, such as uterine derangements; hardened fæces in the rectum; the sudden application of cold to the skin; cold feet; or boils. 7. Occasionally an asthmatic attack is *centric* in its origin, as when it accompanies violent emotion or hysteria; or in those very rare instances where it results from organic disease affecting the roots of the vagus nerves. 8. Irritation of the *pneumogastric nerves* in their course may, in exceptional cases, be the cause of asthma.

Dr. Berkhart advocates the following view with regard to the nature of asthma. He considers it to be a symptom attending all diseases of the lungs in which the pulmonary tissue is deficient in elasticity. It most frequently accompanies emphysema, when this is fully developed, and during its latent stage of development. In consequence of the deficiency of the elasticity, the force of expiration is greatly reduced, and obstacles to the interchange of gases are overcome only by prolonged and unusual efforts. Such obstacles are:—1. Hyperæmia of the mucous membrane causing occlusion of the bronchial tubes, from atmospheric influences, and the inhalation of foreign bodies. 2. Inflammation of the mucous membrane, followed by the production of

thick fibrinous sputa. 3. Compression of the bronchial tubes. 4. Interstitial œdema. 5. Embolism of the pulmonary artery.

*Predisposing causes.*—In a large number of cases asthma commences within the first ten years of life, but the complaint increases in frequency from 20 to 50 (Salter). Men suffer much more than women. Hereditary predisposition appears to have some influence.

*SYMPTOMS.*—In some cases *premonitory* indications of the approach of a fit of asthma are observed, especially in connection with the nervous system. Occasionally there is abundant discharge of pale watery urine. There may be gradually increasing dyspnœa and other chest-symptoms for a variable period before the actual attack. In many cases, however, this is quite sudden in its onset, coming on without any warning. It sets in, in the great majority of cases, early in the morning, especially from two to three o'clock; but the taking of meals, the recumbent position, effort, sleep, and other causes may determine the time of the occurrence of a paroxysm. In many instances a distinctly periodic tendency is noticed, and the interval may be remarkably uniform, the attack being either associated with some evident cause, or being independent of any such cause.

*Characters of a paroxysm.*—The patient experiences an extreme sense of suffocation and want of breath, with tightness and oppression across the chest; loosens every article of clothing; and seizes upon every means for obtaining fresh air. The position assumed varies in different cases, the patient either sitting, standing, or kneeling, and fixing the hands or elbows on some support; or the attitude may be frequently changed. Violent respiratory efforts are made, every muscle being called into action, while the shoulders are raised, and the head is thrown back, the mouth being kept widely open. In consequence of these exertions the sweat often pours off the upper part of the body. The rate of breathing is frequently not increased, but inspiration is very short, abrupt, and jerky, while expiration is greatly prolonged, often terminating with a sudden effort at expulsion of the air, and being immediately followed by the inspiratory act. Respiration is noisy and wheezing. Soon signs of overloading of the venous system and of deficient aëration of the blood appear, and they may become very marked, the extremities being cold, and the pulse small and quick or sometimes irregular. The duration of the struggle varies greatly in different cases, and it may go on for a long time with remissions or intermissions. The length of the asthmatic fits is often remarkably uniform in any particular case. The attack ends either suddenly or gradually, this depending much upon its duration; and upon whether it is allowed to run its course, or is checked by some powerful therapeutic agent. Generally a cough sets in towards the close, followed by a small amount of expectoration, in the form of little pearl-like grey pellets of mucus. In some cases the expectoration is considerable, and continues for some time, especially if the paroxysm is prolonged, and then the asthma is termed *humid*. Occasionally hæmoptysis occurs, usually very slight, but sometimes abundant.

*Physical signs.*—During a paroxysm of asthma the physical signs are very characteristic, as evidencing constriction of the bronchial tubes, and interference with the passage of air. 1. The chest is frequently *enlarged*, the lungs being inflated. 2. *Expansile movements* are greatly deficient or absent; while the intercostal spaces, supra-sternal and supra-clavicular fossæ, and the epigastrium sink in markedly during



inspiration. The *rhythm* of the movements is altered, as above described, expiration being prolonged. 3. *Percussion-sound* is extra-resonant; and inspiration or expiration produces little or no effect upon it. 4. *Auscultation* discloses feeble or absent breath-sounds where the tubes are constricted, with loud puerile sounds where they are free; along with *dry rhonchi* in every conceivable variety. At the close some *moist râles* may often be heard. Important characters presented by these auscultatory signs are that they are frequently limited in their extent; and are constantly liable to change their place rapidly. Upon the sudden cessation of the bronchial spasm an exaggerated breath-sound may be heard where a moment before no sound was audible. Usually both lungs are affected, but occasionally only or chiefly one of them, and then breathing is excessive on the unaffected side.

*State in the intervals.*—This will depend upon whether the asthma is or is not due to organic disease. Immediately after an attack a feeling of exhaustion is usually experienced, with uncomfortable sensations about the chest; but when these pass off patients generally feel relieved, and enjoy an immunity from further paroxysms for a time. As a case of asthma progresses, the fits tend to become more frequent but less severe.

It will be convenient briefly to allude here to the affection named *hay-asthma* or *hay-fever*. This appears to be due to idiosyncrasy, being only observed in particular subjects, who suffer every hay-season, often without any evident exposure to the exciting cause. It has been attributed to the pollen of *anthoxanthum odoratum*. The same effects may be produced by breathing the powder of *ipécacuanha*. The symptoms are those of coryza and bronchial irritation, attended with severe cough; short asthmatic attacks, especially at night; as well as much languor and a sense of depression and want of energy, but no pyrexia. They set in acutely, and last for a variable time.

**DIAGNOSIS.**—Bronchial asthma is sufficiently characterized by the paroxysmal and usually sudden nature of the attacks; their peculiar characters, severity, duration, and often sudden termination; the physical signs of temporary constriction of the bronchial tubes, with absence of fluid in them; the effects of treatment; and the complete or comparative absence of dyspnœa in the intervals. It has chiefly to be distinguished from emphysema, bronchitis, and cardiac dyspnœa, but it must be remembered that asthma may complicate these conditions. It may also be mistaken for laryngeal or diaphragmatic asthma.

**PROGNOSIS.**—The immediate prognosis in cases of asthma is favourable, death during a paroxysm being a rare event. The prognosis as to recovery is more hopeful if the patient is young; if the attacks only come on at long intervals, and are not severe or prolonged; if during the intervals the patient feels well, and there is no organic disease; and if the paroxysms are traceable to some obvious cause, which can be avoided. The history of the progress of the case will afford some aid in determining the prognosis.

**TREATMENT.**—1. **Prevention of an impending attack.**—In those cases where there are premonitory signs of a fit of asthma it may be possible to avert this by drinking strong coffee; removing every source of irritation; heating the body, or in some cases applying cold to the back; or smoking stramonium or belladonna. Various other preventive measures are recommended.

2. **During a paroxysm.**—Any obvious exciting cause must be at once removed, for instance an emetic or enema being employed should the attack be due to a loaded stomach or rectum; as much fresh, dry, warm air as possible must be obtained; and everything that can obstruct the breathing should be loosened. The position of the patient needs to be studied: the sitting or kneeling posture is usually the best, with the elbows supported so as to raise the shoulders, but not uncommonly patients must be allowed to choose the posture which they find most comfortable.

The remedies recommended for asthma are exceedingly numerous, chiefly belonging to the class of *depressants*; *sedatives* and *anti-spasmodics*; or *stimulants*. Different cases are relieved by totally different lines of treatment, and in many instances it is at first quite an experiment as to what will suit best, but patients learn by experience what gives them most speedy relief. The chief remedies which may be of service when given internally are depressing *emetics* and *nauseants*, especially ipecacuanha or tartar emetic; tincture of belladonna, conium, hyoscyamus, stramonium or datura tatula; opium or morphia; ether; hydrate of chloral; tincture of lobelia in gradually increasing doses, frequently repeated; cannabis indica; nitrite of sodium and nitro-glycerine; jaborandi; grindelia; monobromated camphor; strong hot coffee without milk or sugar, taken on an empty stomach, to which it has been recommended to add 1 to 5 grains of caffeine; some spirit with boiling water in equal parts; or fragments of ice rapidly swallowed. *Inhalations* are of great value, when judiciously employed, some materials thus employed being directly inspired; others being smoked, either in a pipe or in the form of a cigarette. The most important remedies for direct inhalation are ether, chloroform, or a mixture of these agents; sulphurous acid; nitrite of amyl, which has been strongly recommended by Dr. Talfourd Jones of Brecon, but must be very cautiously employed; iodide of ethyl; and the white fumes which arise from ignited nitre-paper, or paper impregnated with both nitre and chlorate of potash. The principal substances smoked are tobacco, stramonium, datura tatula, and belladonna, either separately or mixed, and considerable relief is often thus obtained, but of course due care must be exercised in conducting this mode of treatment. Coca-leaves and eucalyptus have also been thus used. Special powders or pastilles are likewise made, which are burnt for the purpose of inhaling the fumes. The powders are of various compositions, but contain some of the following ingredients:—nitre, stramonium, datura tatula, lobelia, cannabis indica, and eucalyptus oil. The pastilles consist of chlorate and nitrate of potash. These preparations may be burnt in a room, as well as directly inhaled. A small quantity of the following powder is now used with much benefit at the Brompton Hospital:—R. Pulv. stramonii,  $\mathfrak{z}\frac{1}{2}$ ; Pulv. anisi, Potass. nitratis,  $\mathfrak{aa}$   $\mathfrak{z}\text{ij}$ . Cigarettes have likewise been employed, made of paper, each containing a minute proportion of morphia, arseniate of soda, or other powerful drugs. Cigarettes containing cubebs have been favourably spoken of. Subcutaneous injection of morphia, atropia, pilocarpine, chloral, or other agents may be demanded in severe cases.

Various other measures prove serviceable in some instances in the treatment of asthma, such as applying cold or heat to the surface of the chest; ice to the spine; the use of warm friction or turpentine fomentations over the chest; sinapisms to various parts; putting the hands and arms into warm water; a warm foot-bath with mustard in it, cold water



being drunk at the same time; or a weak galvanic current along the course of the vagus nerves. The compressed air-bath may give much relief if it should be available.

3. **During the intervals.**—At this time the main points to be attended to are to study the locality which suits the patient best, as regards the qualities of the air and other conditions; to pay strict attention to the state of the alimentary canal and to the diet, as well as to the functions of the organs generally; and to avoid everything which is known to bring on an attack of asthma, especially cold and damp. It is remarkable that asthmatic patients are usually better in the impure atmosphere of large towns and cities, but damp is almost always injurious. In many cases the habitual use of some of the remedies already mentioned may ward off the fits, such as smoking tobacco or stramonium, or inhaling the fumes of nitre-paper or the vapour of chloroform. If any organic disease is present, this must be treated accordingly, especially emphysema and bronchitis. Any constitutional disease, such as gout, must also be attended to. A course of quinine, strychnine, arsenic, or some metallic tonic is very serviceable in many cases. Galvanism or counter-irritation along the vagus nerves, the inhalation of compressed or rarefied air, and various other measures have been advocated for the cure of asthma.

With regard to **hay-asthma**, it is necessary to avoid the cause of this complaint, and the sea-side seems to offer the best protection, or if possible a voyage should be taken. During the attack small doses of hydrocyanic acid with tincture of lobelia or other *antispasmodics* may be given at frequent intervals. Iodide of potassium, ammonium chloride, and belladonna are amongst the numerous remedies advocated for the treatment of hay-asthma. A tincture of *anthoxanthum odoratum* has been used on homœopathic principles, both internally and locally. Weak inhalations of creosote, carbolic acid, or chlorine have been recommended; and it has been affirmed that the asthmatic attacks are relieved by inhaling the fumes of the anti-asthmatic powders already mentioned. Carbolyzed smelling-salts is also used. Injection of quinine into the nostrils has been found useful; and also plugging of the nostrils with glycerine. Moreover, snuffs have been used, containing salicylic acid, camphor, bismuth, morphia, and other ingredients. As preventive measures, the administration of quinine and iron, arsenic, nux vomica or strychnine, and other *tonics* might be tried, along with cold bathing. Dr. Russell Reynolds has found the systematic inhalation of a few drops of chloroform useful.

## II. DIAPHRAGMATIC ASTHMA.

A form of asthma is supposed to be due to spasm of the diaphragm and other muscles of respiration. The respirations are diminished in number, and the difficulty in breathing is limited to expiration, which is greatly prolonged, inspiration being short and abrupt, while but little air enters the lungs. The abdominal muscles become rigid and hard, and may cause expulsion of the urine and feces. There is a sense of much distress, and signs of imminent suffocation may appear. Should the spasm subside, this event is not followed by cough or expectoration. *Physical examination* reveals distension of the lungs, undiminished during expiration. There are no dry rhonchi. I have seen symptoms very similar to those just described brought on by an immoderate fit of laughter.



## CHAPTER XIV.

ATELECTASIS, APNEUMATOSIS, PULMONARY COLLAPSE.  
—PULMONARY COMPRESSION.—CARNIFICATION.

THESE terms all signify a condition in which the lungs are to a greater or less extent merely devoid of air, so that the affected portions are useless for respiratory purposes. *Atelectasis* strictly refers only to lungs which are more or less in their foetal condition, never having expanded properly. The other terms indicate a return to this condition, either from *collapse* in consequence of air being prevented from entering the vesicles through the air-passages; or as the result of external *compression* of the lung.

ÆTIOLOGY AND PATHOLOGY.—1. *Collapse*.—The results of experiment and observation show that the ultimate effect of complete and continued obstruction or narrowing of a bronchial tube from any cause is collapse of the vesicles which it supplies. The explanation of this fact is as follows:—The bronchi become smaller as they divide, and the air drawn in during inspiration drives on any obstructing material, so that at last it reaches a point where it causes complete closure, and thus no air can enter the vesicles. During expiration the obstructing plug is forced out to some extent, and a certain quantity of air escapes, but it again returns with inspiration, acting in fact like a “ball-valve.” Hence, as no new supply of air enters the vesicles, and that previously contained in them is gradually expelled, they finally collapse entirely. It is supposed also that some of the imprisoned air may be absorbed. In the great majority of cases the obstruction is associated with bronchitis, either simple or complicated with measles, whooping-cough, or croup, especially if its products are very tenacious and viscid, but they need not be of this character. Infants are extremely prone to suffer from pulmonary collapse, and this condition is especially frequent during the first year of life, and in children who are ill-nourished or rickety. Among the chief *predisposing* causes are a yielding condition of the chest-walls, and a weak state of the inspiratory muscles; inability to cough and expectorate; distension of, or pressure upon the abdomen, preventing the movements of the diaphragm; and the previous existence of atelectasis.

When pressure is exerted on a main bronchus, as by an aneurismal or other tumour, the whole lung may ultimately become collapsed.

2. The chief causes of direct *compression* of the lung are accumulations of fluid or air in the pleural cavity, or agglutination of its surfaces; great pericardial effusion or cardiac enlargement; an intra-thoracic tumour or aneurism; deformities of the thorax; and abdominal enlargements invading the chest, as from ascites, an ovarian tumour, an enlarged liver or spleen, or a hydatid tumour.

ANATOMICAL CHARACTERS.—The morbid appearances in *atelectasis* and *collapse* are very similar. Usually distinct lobules are involved—*lobular collapse*, these being scattered through different parts of the lungs. The

margin of the bases, the tongue-like prolongation of the left upper lobe, and the middle lobe of the right lung present collapsed lobules most frequently; and next in order come the back of the upper and lower lobes on both sides. Superficial lobules are much more commonly affected than those which lie deep in the lungs.

The precise characters presented will vary according to the duration of the collapse, and the amount and conditions of the blood in the affected lobules. At first there is congestion, but soon the blood coagulates in the vessels, and then undergoes changes, becoming decolorized, firm, and contracted, the vessels being finally obliterated. The walls of the alveoli after a time adhere together, and catarrhal pneumonia is frequently set up. As seen on the surface of the lung, the collapsed portions have a well-defined outline, and are usually sunk below the surrounding level, but not always. Their size depends upon that of the bronchus obstructed. The colour varies considerably, ranging from deep purple to light red, but being usually dark-red or of a somewhat violet hue. Whitish streaks are evident on close examination, indicating the division into smaller lobules. A section is quite smooth, but varies in colour, and it shows the collapsed part to be somewhat pyramidal in shape, with the base outwards. The tissue is quite airless and non-crepitant, usually of a tough and firm consistence, and when situated in a thin margin of lung the collapsed portion may be felt between the finger and thumb. Pieces sink in water. The affected lobules can usually be inflated to a greater or less degree by means of a blowpipe introduced into the communicating bronchus, and they then enlarge, assume a light red colour, and come to resemble normal lung-tissue, but they soon subside unless the bronchus is tied. In proportion to the degree of congestion will be the depth of colour, bulk, firmness, and difficulty of inflating the collapsed portions. In course of time they become paler, looser but tough in texture, and cannot be expanded, as the walls of the vesicles adhere. The tubes proceeding to collapsed lobules will usually be found to contain some obstructing secretion. Other parts of the lungs are frequently the seat of emphysema.

*Compression* drives the air and blood out of the lungs to a variable degree, and the appearances differ accordingly. When the air is alone expelled, while the blood remains, the lung-tissue is dark red, moist, but very firm and dense, and this is the condition known as *carnification*. Finally it becomes grey; anæmic, but pigmented; dry; of a tough leathery consistence; and incapable of insufflation.

**SYMPTOMS.**—Dyspnœa, with quick and shallow breathing; feeble and ineffectual cough; signs of deficient blood-aëration; and marked wasting and exhaustion are the phenomena attending pulmonary collapse, their severity being necessarily in proportion to the extent of the mischief, and the rapidity with which it is set up. Death is a very frequent termination in children, and may take place speedily or gradually. The *physical signs* are:—1. Those of *inspiratory dyspnœa*, the chest falling in more or less during inspiration. 2. *Impaired resonance* over the affected parts. 3. *Weak or bronchial breath-sounds*. In many cases, however, no physical signs whatever can be observed; and they may be obscured by those of emphysema, bronchitis, and other morbid conditions.

Considerable compression of the lung may exist without any symptoms, if it is produced gradually. In this condition a few crepitant râles may sometimes be heard at the close of a deep inspiration—*compression-rhonchus*; and the heart is often unduly exposed.

PROGNOSIS.—Extensive collapse is exceedingly dangerous in very young children, especially if the patient is feeble, and placed in unfavourable hygienic circumstances. This condition adds greatly to the fatality of bronchitis, hooping-cough, measles, and croup.

TREATMENT.—When pulmonary collapse is suspected during an attack of bronchitis in children, the main objects in treatment are to assist the respiratory efforts, and the discharge of the obstructing secretion. Friction with oil over the chest, as recommended by Dr. Graily Hewitt; artificial respiration; the application of sinapisms; an *emetic* of sulphate of zinc or ipecacuanha; and the administration of *stimulant expectorants*, constitute the chief measures which are available. A warm bath is of service occasionally. The diet must be carefully attended to, and much support is often required, this being adapted to the age of the patient; stimulants are also frequently useful. If there are signs of apnoea, the warm bath with cold douche should be tried. As regards the treatment of compression of the lung, the removal of its cause as speedily as possible is the chief indication.

---

## CHAPTER XV.

### PHTHISIS—PULMONARY CONSUMPTION.

UNTIL within a comparatively recent period *pulmonary phthisis* was almost universally regarded as a *specific disease*, a manifestation of the *tubercular diathesis*, and as being due to the formation of tubercles in the lungs, and the destructive processes consequent thereupon. This is still the opinion of many eminent authorities; and it is the view emphatically expressed by Dr. James Andrew in his *Lumleian Lectures* on the “*Ætiology of Phthisis*,” 1883. He regards phthisis as a specific febrile disease, set up by the *bacillus* of Koch or some other micro-organism, and in many most important respects closely resembling ague. I venture, nevertheless, again to express my entire concurrence with those who regard the term as including cases arising from morbid processes essentially distinct from each other, and not merely tubercular, all of which, however, tend to produce similar results, namely, consolidation followed by destruction of the lung-texture; and wasting of the blood and tissues of the body. In this work little more can be done than to give an outline of the main facts and theories relating to this extensive and difficult subject.

ÆTIOLOGY.—Under this head it is proposed to point out the chief causes which may directly or indirectly contribute to the development of phthisis, and these are numerous and varied. It is not practicable to divide them into *predisposing* and *exciting*, as most of them may under different circumstances belong to either class, although Dr. Andrew would regard most of them as merely *predisposing*. 1. *Hereditary or family predisposition*.—There can be no doubt as to the existence of an inherited tendency to phthisis, but the proportion of cases in which this is traceable has been very differently stated by different observers.



Further, many consider that a *specific diathesis* is thus transmitted, whereas others believe that it is merely a *constitutional debility*, and that this may be present in children born of parents in a low state of health from any cause, as well as in those derived from consumptive parents. 2. *Age*. Most cases of phthisis are met with from 20 to 30 years of age. The disease is not often observed during early childhood or in advanced age, but may come on at any period of life. It is usually more rapid in its progress in young subjects. 3. *Constitutional condition*. Persons who are feeble and delicate are most liable to be affected with pulmonary consumption, although the most robust may be attacked. 4. *Occupation*. Phthisis is very common among those whose employment exposes them to various irritant inhalations; to causes originating a cold; or to the influence of certain unfavourable hygienic conditions. 5. *Habits*. Sedentary habits and want of exercise, intemperance, masturbation, excessive sexual indulgence, and debauchery generally, are the chief causes of phthisis coming under this head. 6. *Diet and digestion*. The mal-nutrition resulting from an imperfect supply of nutriment to the system, to whatever cause this may be due, has a powerful influence in developing phthisis, especially in the young. This may be associated with an insufficient amount or improper quality of food; or with a want of power of assimilation, on account of dyspepsia, or of various diseases interfering with digestion. Some writers have laid great stress on a deficiency of fat in the system as a cause of phthisis, either from want of supply of this element, or because it cannot be digested. 7. *Interference with respiratory functions*. Want of ventilation and fresh air, and the consequent breathing of an impure atmosphere, materially assists in the production of phthisis; hence the complaint is common among those whose occupation compels them to remain in a close confined room for many hours during the day, as well as in many instances during the night, such as sempstresses or tailors, especially where gas is burnt constantly or for many hours. It is also frequent in ill-ventilated institutions where many persons are gathered together, especially children, for example, orphan asylums and prisons. Whether interference with the respiratory movements, due to pressure of stays or to posture, has any effect in the production of phthisis is a matter of dispute. Dr. MacCormac attaches great importance to "re-breathed air" as a cause of consumption. 8. *Climate and locality*. Dampness of soil, and abundant moisture in the atmosphere have been proved to be powerful predisposing causes of phthisis. Dr. Buchanan has shown that efficient drainage has materially diminished its prevalence in certain districts. The affection is most prevalent in those climates characterized by rapid changes of temperature, or by prolonged cold with dampness. The tubercular form of consumption is said to be favoured by a high temperature. Elevated regions are remarkably free from phthisis, while those which are situated at a low level present a large number of cases. It has been stated that malarial districts are comparatively exempt from the disease. 9. *Mental causes*. Severe mental depression, as from anxiety, grief, or over-study, certainly seems to have considerable influence in some cases in originating phthisis. The complaint is not uncommon among the inmates of lunatic asylums. 10. *Previous and existing diseases*. Phthisis may follow measles, whooping-cough, croup, typhus, typhoid, scarlatina, and other acute diseases. Repeated attacks of bronchitis greatly favour its development; and it may also result from pneumonia, especially the catarrhal form; from

pleurisy; and probably from laryngitis. Under this head may be mentioned miscarriages; bad confinements; prolonged lactation; continued or excessive discharges, or the suppression of such discharges, all of which certainly increase the tendency to consumption. It is liable to set in during the course of diabetes; as well as in connection with diseases of the alimentary canal and other parts, which interfere with the consumption or assimilation of food. Dr. James Pollock has remarked that young women who are anæmic or chlorotic are peculiarly free from phthisis, but the complaint does sometimes attack such subjects, and may come on very insidiously. 11. *Infection.* The questions relating to the supposed infectious nature of tubercle have already been considered in the chapter on *tuberculosis*, and they are particularly important with reference to phthisis. The notion is gaining ground that this complaint may be communicated from one individual to another, through the medium of the breath or of the expectoration. Dr. Andrew is of opinion that there is not sufficient evidence to prove that the prevalence of phthisis is materially affected by direct contagion. It is maintained by some that the disease may arise from eating the flesh or drinking the milk of animals affected with tubercle. The discovery of the *tubercle-bacillus* by Koch, and especially its presence in phthisical sputum, has given much weight to the infective theory of the origin of pulmonary consumption. Moreover, experiments have been made upon animals, with the view of showing that, if they are made to inhale tuberculous matter, or the sputum of phthisical patients, diffused in the form of spray or small particles in the air they breathe, tubercular disease will become developed. On the other hand, Schottelius has affirmed, as the result of his experiments, that tuberculized nodules in the lungs may be produced by atomized inhalations, not only of tuberculous sputa, but also of purely bronchitic sputa, and of particles of cheese, calf's brain, and other substances. Certain observations seem to indicate that phthisis is in danger of being transmitted from husband to wife, but very rarely from wife to husband.

Such are the principal obvious causes to which phthisis has been attributed. They may be separated into two groups as regards their mode of action, the one tending to affect the system generally; the other to act locally upon the pulmonary organs. In the great majority of cases it will be found that several causes have been at work in originating the disease, and often there is a combination of both classes. Many of those who regard bacilli as the direct cause of phthisis are of opinion that there must be some general condition of the individual essential for the development of the disease by this agency. With regard to the immediate origin of phthisis, it may or may not be traceable to some definite *exciting cause*, such as a cold, or some direct injury or irritation of the respiratory organs.

**PATHOLOGY.**—From a pathological point of view pulmonary phthisis strictly includes all morbid processes which lead to destruction of the lung-tissues, and the formation of cavities. I have already stated my opinion that tubercle is not the only morbid product which induces these results, and in the following observations an attempt will be made to indicate the various ways in which the consumptive process may originate, in accordance with the principal views entertained by different authorities.

1. **Inflammatory forms of phthisis.**—(i.) A comparatively few cases of phthisis result directly from an attack of *acute croupous*



*pneumonia*, especially if this should affect the apex of the lung, the inflammatory products undergoing a process of caseation instead of being absorbed, and ultimately breaking down, thus leading to disintegration of the pulmonary tissue. Acute pneumonia may also originate phthisis by terminating in the formation of abscesses or in gangrene. It must be noticed, however, that Charcot denies that phthisis ever originates in acute lobar pneumonia, and he affirms that none of the reputed cases correspond anatomically or clinically with this disease.

(ii.) *Catarrhal pneumonia*, either *acute* or *chronic*, is the variety of pulmonary inflammation to which Niemeyer attributed the origin of the large majority of cases of phthisis, and he believed that this might arise under the following circumstances:—*a.* As the result of extension of a simple acute or chronic bronchitis into the air-vesicles. He was of opinion that this might occur in a person constitutionally strong, but that it is more liable to happen in the case of those who are debilitated and in a low state of vitality, and that the products are in such subjects more likely to undergo the destructive processes to be presently mentioned. Most cases of *acute* or *galloping* consumption were attributed by him to catarrhal pneumonia complicating extensive acute bronchitis. *b.* From inflammation set up in collapsed lobules associated with bronchial catarrh, as after measles or hooping-cough. *c.* By extension of inflammation due to the inhalation of irritant particles into the air-vesicles, in connection with certain occupations. *d.* As the consequence of the irritation of blood poured out into the bronchial tubes, which, instead of being expectorated, has remained and become coagulated, subsequently setting up catarrhal inflammation. Catarrhal pneumonia may also be set up in lungs which have become partially collapsed or compressed from various causes; and where secretion from the bronchial tubes accumulates in these organs.

Niemeyer's explanation of the destructive changes is as follows:—Cells, the products of inflammation, accumulate in the alveoli and minute bronchi, crowd upon each other, becoming densely packed, and thus by their mutual pressure they bring about their own decay, as well as that of the lung-textures, by interfering with their nutrition, the alveolar walls being also themselves damaged by the inflammatory process. The morbid materials therefore become caseous, and may undergo calcification or absorption, or be ultimately discharged, giving rise to cavities.

Different observers have described special forms of pneumonia as leading to phthisis, which they designate by such terms as albuminous, scrofulous, tubercular, or caseous, but Niemeyer denied that the inflammation has ever any specific characters, and affirmed that all varieties may end in caseous degeneration and consequent phthisis.

(iii.) It is highly probable that some cases of phthisis originate in inflammatory changes chiefly implicating the *walls of the alveoli* and the minute *bronchioles*, with their surrounding tissues—a form of *peri-bronchitis*.

(iv.) *Chronic interstitial pneumonia* leads to destruction of the lung, and, as already mentioned, this morbid condition is termed *fibroid phthisis*. It is observed to a greater or less extent in most phthisical lungs, where the disease is chronic. In the large majority of cases the fibroid condition is secondary, and is an evidence of a disposition towards



healing, but Sir Andrew Clark looks upon it as a special form of phthisis in some cases, the growth of fibroid tissue being primary.

2. **Phthisis from new growths.**—(i.) The usual new growth which originates phthisis is *tubercle*. Niemeyer held that *primary tubercular phthisis* is rare, and that when tubercle is found in the lungs it is as a rule secondary to caseous degeneration of inflammatory products, being formed chiefly in the neighbourhood of these materials; or, should it be primary, some cheesy masses or other sources of infection will be found in other parts of the body. He considered that acute deposit of tubercle in the lungs is more likely to occur as a primary event than chronic; that primary tuberculosis is observed with greater relative frequency in those who are predisposed to inflammation ending in caseous degeneration; that the greatest danger for most consumptives lies in their liability to become tuberculous; and that though tubercle may give rise to pneumonia, this is far less extensive than when the inflammation is the original mischief.

On the other hand, many eminent authorities will not accept these views, but maintain that the formation of tubercle is the first step in the consumptive process in most or in all cases, and that this morbid product undergoes degenerative changes, while at the same time it sets up irritation and excites inflammation; in short, that as a rule phthisis is essentially a tubercular disease.

In this connection Charcot's views demand special notice. As the result of his own investigations, in which he examined phthisical lungs under a high power of the microscope, he ignores entirely the inflammatory origin of phthisis, and holds that the disease, whether acute or chronic, begins in the formation of tubercular nodules or agglomerations. He puts the matter thus strongly:—"Nothing, to my mind, is better established than the existence of infiltrated or discrete tubercle, as a fundamental element in the different forms of pulmonary phthisis. On the other hand, nothing is more doubtful than the existence of caseous pneumonia, independent of tuberculosis, and constituting the prime agent in the phthisical process." This observer denies altogether that the so-called caseous degeneration, ending in yellow consolidation, is the result of the metamorphosis of the products of ordinary inflammation, but that it always begins in the centre of a tubercular nodule or agglomeration, growing at the expense of the "specific embryonic neoplasm" infiltrating the wall of the alveoli, and afterwards invading their cavities. He maintains that the products of common inflammation are only present as a secondary result of the morbid changes, and if they are intermingled with tubercular nodules undergoing caseous degeneration, they will become involved in the process, but their implication is a purely secondary and subsidiary part of it.

It cannot be doubted at any rate that many of the morbid conditions described as *tuberculous infiltration* are not associated with tubercle at all, but are inflammatory in their origin.

(ii.) Under the class of new growths originating phthisis have been included those rare cases of destruction of lung-tissue which apparently result from breaking down of *syphilitic gummata*. Some writers also consider *hydatid-disease* of the lung as a form of phthisis. These morbid conditions will, however, be separately considered in this work.

3. **Vascular conditions.**—Occlusion of branches of the pulmonary artery probably contributes to the destructive process in some cases of phthisis. The decay which occurs in pneumonia or tubercle is usually

believed to be mainly due to compression of the vessels. Dr. Reeves has described a special variety of phthisis, observed in Australia, originating in embolism of the pulmonary branches, and consequent localized gangrene of the lung. It is also affirmed that intensely congested and œdematous lung-tissue may break down and form cavities, thus setting up phthisis; and that hæmorrhagic clots may undergo similar changes.

It is highly probable that phthisis may originate in different cases in either of the ways above indicated. Unquestionably a large number of phthical cases are attributable to causes acting more or less directly upon the pulmonary organs, which tend to excite some form of inflammation, and catarrhal pneumonia certainly does seem to be frequently the primary morbid condition which leads to the destruction of the pulmonary tissue. It must, however, be borne in mind that tubercle may probably result from local irritation or infection, and it may be thus produced along with pneumonia. Moreover, one condition may soon set up another, and so the destructive process may be of a complex nature from the outset, or from a very early period.

Having thus far considered the *ætiology* and *pathology* of phthisis generally, it will be expedient in the subsequent treatment of the subject to give a separate account of the disease, as it occurs in its *acute* and *chronic* forms.

### I.—ACUTE PHTHISIS—GALLOPING CONSUMPTION.

ANATOMICAL CHARACTERS.—Now and then the *post-mortem* examination merely reveals, to all appearance, the remains of an *acute croupous pneumonia*, which has ended in destruction of the lung-tissue. More frequently there are evidences of extensive *bronchitis* with *catarrhal pneumonia*, which may invade large tracts of lung-tissue, the products being soft and caseous, and easily breaking down, or irregular cavities of various sizes having formed here and there. The lower lobes are usually most involved, but acute inflammatory phthisis may begin in the upper lobes, and spread downwards, or be disseminated. Sometimes a whole lobe, or even the greater part of a lung or of both lungs becomes rapidly destroyed. Signs of more or less extensive pleurisy are also observed, generally indicated by deposit of lymph on the pleural surfaces, or by adhesions. In other instances the pulmonary affection is but a part of *acute tuberculosis*, the lungs, in common with other organs, being studded throughout with grey miliary tubercles, at the same time being much congested and œdematous, especially in dependent parts, but not pneumonic. Caseous matter will then generally be found, either in the lungs or elsewhere. It must be mentioned, however, that some writers object to this form of disease being regarded as acute phthisis. Charcot has described tubercle as being present in the lungs in cases of acute phthisis which he examined, and which appeared to be simply of a broncho-pneumonic character. On examining the nodules microscopically under a high power, he found that they consisted of a central region undergoing caseous degeneration; surrounded by a zone mainly composed of a peculiar embryonic tissue, filling the cavities of the alveoli and infiltrating their walls. The outer boundary of this zone was irregular, and in it were habitually found giant-cells, sometimes disposed in regular order, and completely surrounding the central zone.

**SYMPTOMS.**—The clinical history of acute phthisis is that of a febrile disease, which is attended with prominent pulmonary symptoms, and as a rule with signs of consolidation and subsequent destruction of portions of the lungs, either progressively advancing, or assuming a disseminated character. It may attack a person previously healthy to all appearance, but this is not usually the case. Hæmoptysis is sometimes the first symptom noticed. The *course* is in some instances extremely rapid and virulent, but any case of phthisis ending within a few months would be considered acute.

When acute phthisis originates in *croupous pneumonia* it is indicated by a continuance of the chest-symptoms and fever, with abundant sweats and wasting; while the *physical signs* show persistence of the consolidation, followed by softening and the formation of cavities. When associated with *broncho-pneumonia*, the local symptoms include pains about the chest; considerable dyspnoea; frequent cough; and abundant expectoration, which may be “rusty.” There is considerable pyrexia, especially at night; accompanied with much sweating, repeated rigors in many cases, rapid wasting, and great debility. *Physical signs* at first reveal merely the presence of bronchitis. Afterwards there will be indications of consolidation, softening, or excavation in various parts, these being often most marked towards the bases, namely, deficient resonance or dullness; bronchial or hollow breath-sounds; crackling, followed by large, moist, and often ringing or metallic râles; and increased vocal fremitus and resonance. Pleuritic friction-sound is also heard in many cases.

In the *acute tubercular* form the symptoms are those of very high fever, with intense prostration and adynamia, as described under acute tuberculosis; there being also extremely hurried breathing and cough, but no marked *physical signs* in connection with the lungs, only râles significant of pulmonary congestion and subsequently of œdema being observed. There may be evidences of tubercle in other parts.

**DIAGNOSIS.**—This subject will be considered in a future chapter; at present it is only necessary to mention that care must be taken to avoid confounding acute phthisis with certain specific fevers, and especially typhoid.

**PROGNOSIS.**—Acute phthisis is a very grave complaint, and according to the ordinary definition of the disease it invariably ends fatally. At the same time cases do occur in which all the clinical phenomena resemble those which are observed in the pneumonic forms of phthisis, but recovery ensues. Moreover, phthisis may set in very acutely, and afterwards subside into a chronic form of the complaint. Acute pulmonary tuberculosis may be regarded as always fatal in its termination.

**TREATMENT.**—According to the nature of the disease, the treatment of acute phthisis will either be that of ordinary pneumonia; of extensive bronchitis, with catarrhal pneumonia; or of acute tuberculosis. All kinds of lowering measures are to be avoided; and a supporting and stimulating plan of treatment is invariably indicated. If there is high fever, full doses of quinine may be given, and the application of cold employed with due precautions. Various symptoms, such as pain, cough, dyspnoea, hæmoptysis, sweating, and sickness, often need attention. Local applications to the chest, in the form of poultices, sinapisms, turpentine fomentations, or blisters, are frequently serviceable. Dr. McCall Anderson has treated successfully some cases of apparently acute phthisis, by free support and the administration of



brandy; the application of flannels wrung out of iced water over the abdomen at intervals, for half-an-hour at a time; the administration of pills containing quinine, powdered digitalis, and opium; and the subcutaneous injection of atropine, to check the sweating.

## II. CHRONIC PHTHISIS.

ANATOMICAL CHARACTERS.—The appearances observed in the lungs in connection with chronic phthisis vary greatly in different cases, according to the nature of the destructive process, the changes which have taken place during the progress of the disease, and the other morbid conditions with which it is so frequently associated. As a rule, but not always, the mischief begins and is most extensive and advanced at the apex, the entire upper lobe becoming then progressively involved from above downwards, and subsequently the lower lobe, so that the morbid changes are seen in various stages, often retrograding in one part while advancing at another, and being of a different nature in different portions of the lungs. In persons who die of chronic phthisis both lungs are usually implicated to a greater or less extent, though not equally. The disease, however, usually commences in, and may be limited to one lung, or even to a small portion of it; and may undergo curative changes, so that when the patient dies from some other cause evidences of former pulmonary mischief are observed.

The primary morbid condition in the development of phthisis is *consolidation* of some kind. This may originally present the characters of the ordinary grey hepatization of pneumonia, but only in very exceptional cases; of, most commonly, a gelatinous-looking infiltration, greyish, homogeneous, and smooth on section, at first limited to lobules, but afterwards involving the pulmonary tissue extensively, and supposed to be due to catarrhal pneumonia or infiltrated tubercle, according to the view entertained with regard to its pathology; or of grey miliary tubercles, either separate, or, more frequently, collected in groups. The formation of true tubercle is probably in the large majority of cases a secondary process, but may be primary, and it is produced either in the perivascular sheaths; in the walls of the air-vesicles; in the mucous membrane of the bronchi; or in the neighbouring adenoid tissue.

The tendency in all these morbid products is to undergo caseation and subsequent disintegration to a variable degree and extent, either rapidly or gradually. As a result of these changes considerable alterations in their aspect and characters are observed. The affected parts become yellow, opaque, and soft, and give rise to the appearances formerly, and by some authorities even now regarded as characteristic of yellow tubercle. When tubercles become caseous small yellow nodules are seen, but these are frequently simulated by a section of a bronchial division or of alveoli enclosing caseous matter. There is no doubt but that complete liquefaction may take place finally, followed by absorption or expectoration of the morbid product, and ultimate recovery. Frequently calcification ensues, hard calcareous nodules or masses remaining in the lungs. The further course of phthisis, however, is characterized ordinarily by the continued softening of the morbid materials, which finally communicate with the bronchi, and are discharged by expectoration, *cavities*, *excavations*, or *romicæ* being thus

originated in the lungs. A full account of vomicæ is given in the *Gulstonian Lectures* delivered in 1882, by Dr. Ewart; but only the more prominent points can be noticed here. Cavities vary greatly in number, size, form, and other characters. They may be sub-lobular, lobular, lobar, or even involve the whole of one lung ultimately. They increase in size by the gradual necrosis of their inner wall; by the fusion of adjacent excavations; and by their encroachment upon fresh bronchial territories. When lobular, an excavation is roughly spherical; irregular vomicæ may arise from the coalescence of many lobules, simultaneously involved; but the ultimate shape of cavities chiefly depends upon the peculiarities of the bronchial distribution to the district affected. During their active progress their walls are soft, irregular, rough, and ulcerous; subsequently a surrounding fibro-vascular zone forms, the innermost portion constituting a pyogenic layer at first, and subsequently an exfoliating false membrane. A knobbed condition of the internal surface of cavities is not uncommon. Vomicæ generally contain muco-purulent or purulent matter, or sometimes a thin, dirty, shreddy, and fœtid liquid; blood may also be present. Trabeculæ frequently traverse cavities, consisting of blood-vessels and of collapsed or indurated alveolar substance; they are the remains of intervals of spongy tissue, originally separating distinct cavities. The surrounding tissue may be normal alveolar substance, inflamed and thickened tissue, infiltrated with tubercle, or of a fibrous nature. Vomicæ above a very small size always open into one or more bronchi, but the communication may be temporarily or permanently closed. These tubes ulcerate at an early period. Often two sets of bronchial orifices may be observed in cavities, the proximal and the distal. They open abruptly, either directly or slantingly, and presenting circular or oval orifices, indicated by small islets of mucous membrane. With regard to the vessels, the trabeculæ always contain vascular branches, either patent or obliterated. Branches of the pulmonary artery also ramify in the walls. The vessels are more resistant than all the other structures, and thus may be completely exposed, while the circulation through them continues. Moreover, they often become the seat of small aneurisms or "ectasias." As a rule these occur simultaneously in different parts of the same lung; they may undergo spontaneous cure if in very small cavities. The vessels are very liable to rupture, either from mere erosion or at the seat of minute aneurisms; thus fatal hæmorrhage often occurs, but a pulmonary aneurism is sometimes cured by the pressure of extravasated blood. Vomicæ may be found in any part of the lung. With few exceptions they begin in the upper part of the upper lobe, especially the central part of the sub-clavicular region. Almost always the mid-dorsal region is secondarily involved, and Dr. Ewart attributes this to the transmission of irritating matter along the bronchi supplying that district.

In most cases of phthisis more or less *chronic interstitial pneumonia* is set up, which often materially aids in arresting and repairing the mischief resulting from the disease. It arises in the neighbourhood of consolidations and caseous products, sometimes forming dense capsules around them, or originating indurated masses; and also around cavities, which are then said to be *encapsuled*. The vomicæ subsequently may gradually contract and finally close up, leaving only an indurated puckered cicatrix. Complete obliteration, however, is rare. The contraction is chiefly due to shrinking of the capsule, but is aided by other



forces. There is also a retraction towards the root of the lung often observed, partly due to inflammatory thickening and clustering of the bronchi. In some very chronic cases of phthisis the affected portions of the lungs may present nothing but fibroid induration, with cavities in various stages of contraction. It must be remembered that this last condition originates in a different manner from that which is believed to occur in primary *fibroid phthisis*.

In addition to the diverse appearances in the lungs resulting from the combination of conditions already described, these are generally further modified by their association with bronchitis, often with ulceration of the bronchial mucous membrane; dilated bronchi; emphysematous patches; pulmonary collapse; extravasations of blood or their remains; or recent pneumonia. Pleuritic adhesions and thickenings are always evident, especially at the apices, where a dense fibrous cap is often formed, which may be half an inch or more in thickness. In these adhesions new vessels are developed by extension from the intercostals, and thus a communication is formed between the latter and the vessels of the lungs.

Other structures besides the lungs are generally found to be involved in cases of death from phthisis, as will be pointed out when considering its complications.

**SYMPTOMS.**—Chronic phthisis presents considerable variations in its clinical history, both as regards its mode of onset and its subsequent course, but the symptoms bear a general resemblance in the different cases. The disease may commence quite suddenly, as by an attack of hæmoptysis; or it may remain after some acute affection; or may come on acutely, afterwards becoming chronic; or may set in gradually and insidiously. In the latter case the pulmonary symptoms are first observed in some instances, especially those indicative of chronic bronchial catarrh; in others signs of constitutional disturbance, or of derangement of the digestive organs are noticed at the outset. The symptoms may be described as *local* and *general*.

*Local.*—Pains in the chest and sides are common, though not usually severe. They seem generally to be either pleuritic or muscular, the latter being often the result of cough. Dyspnoea is frequently present more or less from various causes, but may be entirely absent. Respirations are usually increased in number, rising somewhat towards evening. Shortness of breath on exertion is very commonly complained of. Of course when the lungs are extensively diseased breathing is much affected. Cough is an essential symptom of phthisis, and may for some time be the only one complained of. In its severity and characters it differs widely, and that by no means necessarily according to the extent of the disease. At first it is often dry and hacking. An abnormal condition of the throat or larynx not infrequently gives rise to cough, which in the latter case is generally of hoarse quality. It is usually worse on first lying down at night, after sleep, and after meals. A paroxysm is often terminated by vomiting, especially after food has been taken. Expectoration soon occurs in most cases, but much of the sputa often comes from the bronchial tubes, which are the seat of catarrh. Their characters and amount alter during the course of a case, and they present much variety in these respects. At first they consist merely of clear mucus, or sometimes small opaque pellets are discharged; subsequently they become muco-purulent; and when cavities of some size form, irregular, opaque, airless masses are often



expectorated, more or less greenish-yellow, which sink in water, and which when discharged on to a flat surface spread out in the form of a coin, hence named "nummulated." This kind of sputum is not characteristic of phthisical cavities, however, as it may be observed in mere bronchitis. The masses are mingled with more or less bronchial mucus. In some cases mere pus is expectorated, and occasionally a quantity of matter is suddenly discharged, owing to the opening of a cavity. The sputa often have an unpleasant odour, and they may be extremely offensive, but this is exceptional. In favourable cases, even after large cavities have formed, expectoration diminishes, and may ultimately cease altogether. Examination of the sputa may reveal evident caseous or calcareous particles. Microscopic examination may discover epithelium; abundant newly-formed, granular, or pus-cells; blood-corpuscles; numerous fat-granules and oil-globules; calcareous granules; vegetable growths not uncommonly; and, in some instances, fragments of the lung-tissues, especially elastic fibres, the presence of these last elements being regarded as of great importance. Sugar may often be detected chemically.

Special consideration must be given to the presence of *tubercle-bacilli* in phthisical sputa, as the detection of these organisms in the expectoration has come to be regarded as an important factor in diagnosis. Different methods of detecting them have been recommended, but it must suffice to mention the following:—

1. *Ehrlich's method.* A thin layer of sputum is placed on a cover-glass, and rubbed between two glasses. They are then dried in the air, and passed three or four times through the flame of a spirit lamp, to coagulate the albumen and fix the sputum. The glasses are now placed with the sputum downwards in a specially-prepared staining-solution, and allowed to remain for half an hour. They are afterwards washed in dilute nitric acid (1 to 2 of distilled water), until all apparent colour has been removed, and then in distilled water. While the cover-glass is still wet, a drop of a saturated solution of methylene blue in distilled water is dropped on to the sputum, and allowed to remain until the whole surface is distinctly stained. It is finally again washed lightly in distilled water, and the specimen is examined while still wet, under a power of 400 or 500 diameters; the bacilli appear as red rods on a blue ground.

The first staining-solution is prepared by dissolving 5 cubic centimetres of commercial anilin in 100 cubic centimetres of distilled water, well shaking and filtering, and adding to this a saturated solution of fuchsin in alcohol, until precipitation commences. It is recommended to add 30 minims of this solution to from 20 to 30 cubic centimetres of the anilin solution, and to continue adding it until a distinct metallic film is produced.

2. *Heneage Gibbes's method.* Dr. Gibbes uses magenta crystals and chrysoidin as colours, and his method is as follows:—Two grammes of magenta crystals, three grammes of pure anilin, twenty cubic centimetres of alcohol, specific gravity .830, twenty cubic centimetres of distilled water. Dissolve the anilin in the spirit, and then rub up the colour with it in a glass mortar, adding the spirit gradually until it is all dissolved, then add the water slowly, while stirring, and keep in a stoppered bottle. Make a saturated solution of chrysoidin in distilled water, and add a crystal of thymol to make it keep; a dilute solution of nitric acid is made, one part of acid to two of distilled water. Spread a thin layer

of sputum on a cover-glass and let it dry ; when quite dry pass it two or three times through the flame of a small Bunsen burner, and let it cool. Filter two or three drops of magenta solution in a watch-glass, place the cover-glass with the sputum downwards on the stain, taking care there are no air-bubbles under it. Let it remain for fifteen or twenty minutes, then wash in the dilute acid until all colour has disappeared, remove the acid with distilled water, when a faint colour will return ; then place the cover-glass in the same manner as before on a few drops of chrysoidin filtered into the bottom of a watch-glass, and let it remain a few minutes until it has taken on the brown stain ; wash off the superfluous colour in distilled water, and place the cover-glass in absolute alcohol for a few minutes ; remove and dry perfectly in the air, place a drop of Canada balsam solution on the cover-glass, and mount. Dr. Gibbes states that an ordinary  $\frac{1}{4}$  inch with daylight will show perfectly bacilli mounted by this process.

Dr. Charnley Smith, of Manchester, has demonstrated the presence of *tubercle-bacilli* in the breath of patients suffering from true tubercular consumption, by making them breathe at frequent intervals during the day, through two thin layers of pyroxilin or fine cotton, placed in the outer compartment of an ordinary "pepper-duster" respirator. The inner layer contains the bacilli. It is converted into gun-collodion by means of a mixture of ether and spirit ; a very thin film of this is placed on a microscope slide, and stained. Dr. Smith affirms that every vestige of cotton-fibre is dissolved by the above menstruum, but other organic particles remain suspended in it.

Hæmoptysis as a symptom of phthisis demands special notice. This symptom is observed to a greater or less degree in the large majority of cases, varying, however, considerably as regards the amount and exact characters of the blood discharged, and the frequency of its occurrence. The amount of blood may range from mere streaks in the sputa to a quantity sufficient to prove immediately fatal, but death directly due to hæmoptysis is not a common event in phthisis. It is more likely to be abundant after cavities have formed. When blood is intimately mixed with muco-purulent matter it has been stated to be pathognomonic of chronic catarrhal pneumonia. The hæmoptysis is frequently, but not necessarily, brought on by some exciting cause, such as a violent cough. In certain cases it tends to be repeated, and may become almost periodic. When not abundant, the loss of blood seems to afford relief sometimes, but usually the effect of hæmoptysis, if in any quantity, or if liable to frequent recurrence, is to induce debility and anæmia, or it may increase the local mischief in the lungs.

It is believed by some authorities that the blood generally comes from the bronchial capillaries, but probably the pulmonary vessels are its usual source. These may be in a state of fatty degeneration ; or, as already remarked, considerable branches may remain unobliterated or be the seat of ectasias, by their rupture giving rise to serious or fatal hæmorrhage.

*General.*—Pyrexia is a very important symptom in cases of phthisis, and one which should always be looked for by the systematic use of the thermometer. This instrument is particularly important in detecting an early stage of the disease, and in indicating its degree of activity. It has been also stated that the thermometer aids in determining the nature of the destructive process, tubercular phthisis being characterized by a more continuous fever than the other forms. This is a very ques-

tionable statement, however. As a rule marked daily variations in temperature are observed, and it increases considerably in the evening. During the progress, but especially towards the close of many cases of phthisis, hectic fever in its most typical form is observed. Dr. Ringer, as the result of his observations upon the temperature in phthisis, has come to the following conclusions:—1. There is probably a daily unnatural elevation of temperature in all cases during the continuance of catarrhal pneumonia, or whilst a deposition of tubercle is taking place in any of the organs of the body. This elevation of temperature is due either to the miliary tuberculosis or catarrhal pneumonia, and not to secondary complications. 3. It is probably due rather to the general than the local conditions existing in phthisis. 4. The temperature may be taken as a measure of the amount of tuberculosis or catarrhal pneumonia, and fluctuations in the temperature indicate corresponding fluctuations in the amount of disease. 5. The temperature is a more accurate indication of the activity of tuberculosis or catarrhal pneumonia than either the physical signs or the symptoms. 6. By means of the temperature we can often diagnose tuberculosis or catarrhal pneumonia long before we can detect any physical signs, and at a period when the symptoms are insufficient to justify such a diagnosis. 7. By means of the temperature we can diagnose tuberculosis, even when during the whole course of the disease there are no physical signs indicative of tubercular deposit in any of the organs of the body, and when the symptoms are inadequate to enable us to arrive at such a diagnosis. 8. It is probable that by means of the thermometer we can decide when the tuberculosis or catarrhal pneumonia has ceased, and that any existing physical signs are due to obsolescent tubercle or to the products of previous catarrhal pneumonia, and the chronic thickening of the lung-tissue around and between the diseased products.

Observations have been made by Dr. Charteris and Dr. McAlldowie with reference to the difference in the axillary temperatures on the two sides in cases of phthisis, and some investigations on the same point were carried out by myself. It is supposed that the temperature is higher on the side which is solely diseased, or which is most affected; but although this is often observed, it is by no means always the case, and the results deduced from the investigation of a number of cases were so indefinite, that they do not warrant any conclusion which can be of service in the diagnosis of phthisis.

Night-sweats are complained of in the large majority of cases of phthisis during some part of their progress. These tend to come on especially towards early morning, but not infrequently they set in as soon as the patient falls asleep, and may be so excessive as to saturate the bed-clothes, causing much distress and exhaustion. Sweating from slight exertion is also often noticed; or it may occur at any time during sleep. This excessive perspiration is due either to fever or to weakness. Loss of flesh is another prominent symptom, being dependent chiefly upon the pyrexia. This must be determined by frequent weighing, and no reliance ought to be placed on the mere statement of the patient. The emaciation is often extreme, and it is a matter of common observation that it is more marked about the body and limbs, and especially the chest, than in the face. The fat disappears, and the muscles feel flabby and wanting in tone. The chest-muscles are sometimes very irritable on percussion. More or less anæmia is frequently observed, and there may be œdema of the legs from this cause. At first the



blood is generally hyperinotic, but soon it deteriorates in quality. In many advanced cases the skin is dry and scaly. Among other external appearances which may be noticed are chloasma over the chest; grey-ness of the hair in this region; lankiness and falling-off of the hair generally; and bulbousness of the finger ends, with incurved or cracked nails.

The patient almost always complains of debility, varying in degree to the most absolute helplessness and exhaustion. The pulse is increased in frequency in most cases, and tends to be quick, sharp, small, and wanting in tone.

The digestive organs are generally out of order. Impairment or loss of appetite, thirst, and dyspeptic symptoms are often complained of. Not uncommonly the mouth, tongue, and throat are red and irritable, this being accompanied with signs of subacute gastritis. The tongue is frequently more or less furred. In some instances the stomach is extremely irritable, retching and vomiting being immediately excited when anything is taken. The breath has in not a few cases of phthisis a very peculiar odour, which has appeared to me to be quite characteristic. At the close of the day is not infrequently observed. It has been stated that phthisical patients have a peculiar dislike to, and difficulty in the digestion of, fatty substances, but there are many exceptions to this statement. Constipation is the rule at first, but later on there is a great tendency to diarrhoea. A red line along the gums, and transverse cracking of the teeth have been described as significant of phthisis, but they are frequently absent, and are not at all characteristic.

Consumptive patients are inclined to be irritable and fretful. As a rule they are remarkably hopeful—*spes phthisica*, and even when near the end cannot realize their condition, but imagine that they will recover.

The urine is more or less febrile in the early stage, and contains excess of the products of tissue-destruction. Finally it becomes watery and deficient in solids. Albumen or sugar may be present. The menstrual functions are often imperfectly performed, or entirely in abeyance.

PHYSICAL SIGNS.—The *physical signs* which may be associated with phthisis are due to:—1. Primary consolidation. 2. Softening of this consolidation. 3. Cavities in the lungs. 4. Secondary consolidation from interstitial pneumonia, which tends to produce much induration and shrinking of lung-tissue. 5. Other pulmonary affections, namely, pleurisy, bronchitis, emphysema, pneumonia, hæmorrhage into the bronchi, and pneumothorax. It has been customary to divide phthisis into three stages, when describing the physical signs, namely, those of *consolidation*, *softening*, and *excavation*, but these are usually more or less combined, while in addition evidences of curative changes are frequently observed. The extent over which the morbid signs are perceived varies considerably, and in most cases they are present in different stages over different parts of the chest. An important character pertaining to chronic phthisis, however, is, that they tend to be localized, the rule being that they are particularly observed over one or both apices, especially in front, though not to the same degree on the two sides. But this is not always the case, and therefore it is essential to examine every portion of the thorax if there is any suspicion of the existence of phthisis; and also to make frequent examinations in order to determine the progress of the disease. It is not intended here to describe the *physical signs* in different stages, but

those characteristic of cavities will be pointed out separately. It must be remembered that they will be greatly influenced by the situation, nature, and amount of the consolidation.

1. As regards *shape* and *size*, the thorax may be congenitally small, being either alar or flattened, but in a large proportion of cases it is originally in every respect well-formed, though subsequently it tends to become more or less generally diminished in size. At first there may be no local depression, or even some degree of bulging, but the tendency is for the chest to sink in some part, especially in the supra- and infra-clavicular regions; a considerable portion of one or both sides may ultimately fall in. There is often lowering of the shoulder when one apex is much involved. 2. *Local movements* are more or less deficient, especially that of expansion. 3. *Vocal fremitus* is usually increased, but may be normal or diminished. 4. *Percussion* reveals deficiency of resonance or a rise in pitch, which may culminate in the most absolute hard wooden dullness; with more or less resistance. Over the clavicles the sound is frequently purely osteal. The area of pulmonary resonance is often diminished towards the neck, showing that the apex of the lung is contracted. The effect of holding the breath after a deep inspiration will sometimes show deficient resonance where previously it could not be detected. The percussion-sound may, however, be perfectly normal in phthisis, or even unusually clear and resonant at the outset. 5. *Respiratory sounds* may be weak to complete extinction; jerky or of "cogged-wheel" rhythm; harsh, with prolonged expiration; or bronchial or blowing. In healthy parts they are often puerile. 6. The *adventitious sounds* which may be heard are those indicative of bronchial catarrh or pneumonia; collapse-rhonchus in the neighbourhood of the consolidation; or dry crackling followed by moist crackling or even somewhat bubbling râles, significant of softening. 7. *Vocal* and *tussive resonance* are usually exaggerated. 8. Localized *pleuritic friction* or creaking is frequently observed. 9. The *heart* may be drawn up considerably, as well as uncovered by lung, so that the impulse is extensive and strong, and the sounds are loud. The better conduction of the latter towards the right infra-clavicular region than the left is not infrequently a very useful sign of disease at the apex of the right lung. Rarely the heart is lowered, or it may be displaced laterally. 10. A *subclavian murmur* is not uncommon, especially on the left side, due to pressure by thickened pleura on the subclavian artery. 11. The *diaphragm* and *liver* or *stomach* are sometimes drawn up, owing to contraction of either lung.

**Signs of cavities.**—These vary considerably, according to the size, shape, number, and situation of the cavities; as well as with the state of their walls, their contents, the condition of the surrounding tissue, and other circumstances. It can be readily understood that vomicae may exist without there being any, or only doubtful evidences of their presence; and on the other hand a careless observer might mistake signs which simulate those associated with cavities; but cavities in the lungs may generally be detected when they have formed, and by careful attention to, and adequate study of the *physical signs* present, a tolerably accurate conclusion may be arrived at as to their exact conditions, while by examination from time to time the progressive changes may be noted, excavations being thus often traced in their formation, enlargement, contraction, and almost complete closure. The following are the important signs of cavities:—



1.—*Percussion-sound* may be tubular, metallic, crack-pot, or very rarely amphoric. A rise in pitch on opening the mouth has been considered a characteristic sign of a cavity. 2. *Breath-sounds* are either blowing, or more or less hollow, ranging from tubular to cavernous or amphoric. Inspiration has a peculiar sucking or hissing character sometimes. 3. The chief significant *adventitious sounds* are large moist râles at the apices, where there are no bronchi of any size; hollow, metallic, or ringing râles, varying in size, amount, and quality, being sometimes gurgling; and very rarely metallic tinkling or amphoric echo. 4. *Vocal resonance* may have a ringing or metallic character, and is often greatly intensified. Pectoriloquy and whispering pectoriloquy are of frequent occurrence. 5. *Tussive resonance* is often painfully strong and metallic, but cough is chiefly useful in that it may cause the breath-sounds to be better heard, by clearing away secretion or emptying a cavity; or that characteristic adventitious sounds are brought out during the act. 6. The *heart-sounds* are sometimes much intensified by transmission through cavities, and may acquire a peculiar hollow quality, or be attended with an echo. The cardiac action occasionally elicits rhonchi in neighbouring cavities. 7. It is said that a *murmur* may be heard in rare instances over a vomica, due to an aneurismal dilatation involving a branch of the pulmonary artery.

COMPLICATIONS.—Numerous symptoms and physical signs which occur in the course of pulmonary phthisis are dependent upon the *complications* so often met with, some of which are due to tubercle in other parts. The chief of these include:—Affections of the larynx and trachea, especially ulceration; bronchitis, pneumonia, or pleurisy; perforation of the pleura, with consequent pneumothorax; enlargement of the external absorbent glands, or of those in the chest and abdomen; tubercular peritonitis; ulceration of the intestines, especially the ileum; fatty or amyloid liver; fistula in ano; various forms of Bright's disease; pyelitis; diabetes; tubercular meningitis or tubercle in the brain; and thrombosis of the veins of the leg.

COURSE—DURATION—TERMINATIONS.—The course and duration of chronic cases of phthisis are subject to much variety. The disease may progress steadily from bad to worse, either rapidly or gradually, but more commonly there are intervals of improvement, followed by exacerbations. Some cases remain apparently in the same state for a long time; while others, even when far advanced, improve and may ultimately become practically cured. It is sometimes quite astonishing what a length of time patients will remain alive, when apparently almost in a moribund condition. Death may take place from gradual asthenia and hectic fever; from hæmoptysis occasionally; from some of the complications already mentioned, which generally aid in bringing about the fatal result; or from some intercurrent attack.

VARIETIES.—It is very difficult, in the present undecided state of opinion and knowledge, to determine upon the adoption of any definite division of cases of pulmonary phthisis into varieties which shall be of practical value from a clinical point of view. As has been already intimated, some eminent authorities refuse to recognize any varieties of this disease, and will only acknowledge the division of cases into *acute* and *chronic*, according to the intensity and duration of the symptoms. Others adopt a pathological classification, but certainly no corresponding clinical arrangement is practicable. Without attempting any discussion on this matter, it may be well to give a summary of the chief varieties of



consumption which have been brought forward by different writers, and to point out the supposed clinical distinctions between certain of them, but the following arrangement can only be regarded as provisional :—

I. **Acute**.—1. *Croupous pneumonic*. 2. *Catarrhal pneumonic*. 3. *Miliary or tubercular*.

II. **Chronic**.—1. *Pneumonic*. 2. *Catarrhal pneumonic*. This tends to come on insidiously, being preceded by one severe and long-continued attack of bronchial catarrh, or by several repeated attacks. The thermometer reveals more or less pyrexia. The disease is prone to be localized, and slow in its progress; while under proper treatment it shows a decided tendency towards cure, with contraction and induration of the affected part. 3. *Pleuritic*. Unquestionably phthisis may originate from simple pleurisy, especially by leading to compression and subsequent destruction of the lung, and I think that this form of the disease deserves a special designation. 4. *Hæmorrhagic*. Two distinct meanings have been given to this term. With some writers it merely implies that the disease has commenced with spitting of blood, or that this is a prominent and frequent symptom in its course; with others that the hæmorrhage into the bronchi or lung-tissue has actually set up phthisis, by exciting inflammation. That phthisis may originate in this manner seems to me absolutely certain. Dr. Reginald Thompson\* has made some important pathological observations bearing upon this subject, and has arrived at the following conclusions:—*a*. That in cases of severe hæmoptysis portions of the blood are driven into the alveoli, which they occupy finally in the form of fibrinous nodules, setting up some irritation in their vicinity. *b*. That in cases of capillary hæmorrhage, with laceration of the pulmonary tissue, the resultant effect produces a calcareous mass, sometimes of considerable size. *c*. That under special circumstances cavities may be formed by the liquefaction of the hæmorrhagic nodules in the first instance, or by the removal of the calcareous masses in the second. *d*. That whether secondary tubercle can result from inhaled blood, without the intervention of secondary processes introducing a new septic condition, is a point that requires further evidence before it can be accepted. 5. *Fibroid*. This variety has already been fully considered. 6. *Mechanical*. Produced as the result of the constant inhalation of irritant particles, this form has several subdivisions, named according to the nature of the occupation or of the irritation; for example, miners', colliers', and knife-grinders' phthisis; carbonaceous phthisis, cotton-phthisis, &c. The progress is slow, the morbid process being due to a combination of chronic bronchitis, with catarrhal and interstitial pneumonia, though at last true tubercle may form. The expectoration contains more or less of the inhaled substances, sometimes in great abundance. Thus in carbonaceous phthisis or *anthrakosis* the sputa may be perfectly black; and the lungs are often observed on *post-mortem* examination to be in the same condition. 7. *Secondary tubercular*, where tubercle is added to some previous morbid condition. Niemeyer has given the following signs as suggestive of the secondary development of tubercle, but justly remarks that the diagnosis is a matter of much difficulty, and it is very questionable whether this ought to be reckoned as a separate variety:—great increase in dyspnoea and frequency of respiration, without any corresponding increase in physical signs; the fever becoming of a more continued type; and symptoms indicative of laryngeal complication, of intestinal ulceration, or of tubercle in other

\* "Medico-Chirurgical Transactions," Vol. lxi. page 253.

parts setting in. 8. *Primary tubercular*. Here there is no preceding bronchial catarrh. The patient may be evidently tuberculous; while the constitution is greatly affected from the outset, pyrexia of continued type, and wasting being marked symptoms. Dyspnœa is often severe, with rapid breathing, but there are no adequate physical signs. Afterwards there may be evidences of inflammatory consolidation and destruction of tissues, but not to the same extent as in other forms. Soon indications of laryngeal phthisis, ulceration of the bowels, tubercular peritonitis or meningitis, or of other complications appear. The progress is generally rapid.

Some writers recognize *scrofulous phthisis*, *drunkard's phthisis*, and other special varieties. Among the cases which have come under my own observation none have occurred indicating any forms of the disease warranting such distinctive appellations.

DIAGNOSIS.—The diagnosis of phthisis involves not only the recognition of the presence of the disease, but also as correct a knowledge as can be obtained of its seat and extent, its stages in different parts of the lungs, and its nature and origin. These questions can only be determined by a careful and thorough consideration in each case of the history, as well as of the existing symptoms, both local and general; and by complete and systematic physical examination. The diagnosis of phthisis will be considered more fully later on.

PROGNOSIS.—Those who desire full information on this important subject will find it in the valuable work of Dr. James Pollock on Consumption. The ability to form a reliable prognosis in phthisis can, however, only be acquired by much experience and observation. There is now ample evidence to prove that phthisis may in certain cases be completely arrested or cured; while in a large proportion its progress may be greatly delayed by appropriate treatment, and life rendered fairly comfortable. It is difficult, and does not serve any useful purpose, to lay down any average *duration* or *mortality*, these varying so much under different conditions. In endeavouring to arrive at a prognosis, the chief circumstances to be taken into account are as follows:—1. *The stage, seat, and extent of the disease*. At an early period a hopeful opinion is warranted as a rule, though at the same time it should be a guarded one. When cavities have formed the prognosis is very much worse. If the disease is limited to one apex, even should there be a cavity, recovery is not unusual; but the prognosis is more serious in proportion to the extent of the mischief, and the number of excavations, especially if both lungs are involved. Basic phthisis seems to be comparatively unfavourable. 2. *The progress of the local lesions*. Signs of rapid progress, either as regards the extension of the disease, or a tendency to softening and destruction of tissues, are very unfavourable; on the other hand, if the disease is chronic or at a standstill, or if, should a cavity have formed, there are indications that it is drying up and contracting, the prognosis is much more hopeful. Signs of considerable local induration from interstitial pneumonia are often favourable, as showing cessation of active disease, and the advance of healing processes. 3. *Origin and nature of the disease*. Tubercular phthisis is extremely serious; when the complaint follows bronchial catarrh, or is due to certain obvious external causes, from the influence of which the patient can be removed, there is a far better chance of recovery. 4. *Constitutional condition and hereditary predisposition*. Phthisis is more dangerous if the patient is feeble and delicate, but especially if

there are evidences of the existence of a tubercular or scrofulous diathesis, or if there is a strong hereditary tendency to phthisis. 5. *Local symptoms.* Continued dyspnoea; harassing cough; profuse expectoration; and severe or repeated hæmoptysis are bad indications. 6. *General symptoms.* Phthisis is dangerous in proportion to the degree and prolonged course of pyrexia; rapidity and weakness of pulse; debility and incapacity for exercise; emaciation and night-sweats. If the general condition shows signs of improvement, the pyrexia ceasing, and flesh and weight being gained, the prospect is much more hopeful. 7. *State of the digestive organs.* Inability to take food or to digest it is a most serious drawback in phthisis. Cases in which vomiting is a prominent symptom are also exceedingly unfavourable. 8. *Diet and hygienic conditions.* Deficient or non-nutritious food, and improper hygienic conditions are most injurious in cases of phthisis. This is frequently exemplified among the out-patients at the Brompton Hospital. Many who become in-patients revive wonderfully, as the result of the improvement in their diet and surrounding circumstances. 9. *Complications.* Several of these morbid conditions seriously increase the gravity of the prognosis in phthisis, and hasten the fatal result, such as intestinal ulceration, laryngeal phthisis, or Bright's disease; and some complication, for instance pneumothorax or intestinal perforation, may be the immediate cause of death.

The question is often asked in advanced cases of phthisis—How long is the patient likely to last? It is useless to attempt to give more than an approximate opinion on this point, there being so much uncertainty. The appearance of thrush is generally a sign of the “approach of the end.” Another question refers to the effects of pregnancy. Usually this condition seems to delay the disease for a time, so far as my experience goes; but after parturition it generally advances with increased rapidity, though there are some remarkable exceptions to this rule. Marriage of persons decidedly phthisical should certainly be opposed.

**TREATMENT.**—The ultimate objects to be kept in view in the treatment of phthisis are:—First, its prevention and arrest; secondly, its cure; or, failing these, thirdly, palliation of symptoms and prolongation of life. Every case requires thoughtful consideration, and it must not be imagined that this is a disease capable of being controlled by any one remedy or class of remedies. An essential part of the treatment, however, is that which has for its end the maintenance and promotion of a state of general good health and constitutional vigour.

1. **General hygienic and dietetic treatment.**—This is of the utmost importance, both for the prevention and cure of phthisis, and if it is neglected all other measures are usually unavailing. The chief things required under this head are a healthy residence, on a dry soil, in a suitable climate, elevated, but well protected from cold winds, with pleasant scenery and sufficient vegetation; free ventilation, especially as regards the sleeping apartments; fresh air and exercise, so far as the powers of the system will permit; the avoidance of crowded places at night, and of all causes which are likely to excite pulmonary affections; the wearing of warm clothing, with flannel next the skin; the employment of cold baths, if they can be borne, with friction afterwards; the administration of as nutritious a diet as can be assimilated, which should contain a good proportion of fatty elements; and the avoidance of all injurious habits, such as intemperance, excessive smoking, or sexual excess. The question of climate will be separately considered.



It is often requisite to inquire into the occupation of the patient, and to change this, should it entail either prolonged confinement in a close room, deficient exercise, or exposure to the exciting causes of lung-diseases. At the same time the patient should, if possible, be relieved from undue mental labour or anxiety. The amount and character of the exercise to be adopted must vary in different cases, but as a rule such exercises as aid in the expansion of the chest are to be recommended, especially in young patients, though they must be kept within proper limits. Walking and riding are useful, and if these cannot be endured, passive exercise is to be enjoined, the patient being driven out daily when the weather permits, so that at least a proper supply of fresh air may be obtained. At the same time over-fatigue must be avoided. Certain acts which call into exercise the muscles of respiration are often beneficial if duly regulated, such as taking deep inspirations, reading aloud, or moderate singing. Anything that interferes with the freedom of the respiratory movements, as the pressure of tight stays, or a bent position, ought to be forbidden. Milk is a most valuable article of diet, and some practitioners consider asses' or goats' milk especially efficacious. Whey has also been well spoken of. In many cases of phthisis a little wine or beer is very beneficial.

**2. Preventive measures.**—In all cases where there is any fear of phthisis setting in attention should be paid to the slightest indication of pulmonary disorder. Should the complaint be acute in its origin, and of an inflammatory nature, the measures already mentioned when treating of the different forms of pulmonary inflammation must be had recourse to. Further, any acute exacerbation should receive immediate attention; but at the same time it is very important in most cases to avoid lowering measures under such circumstances, and to preserve the strength as much as possible, rest in bed being enjoined. Of course everything which is likely to excite irritation in connection with the lungs must be strictly guarded against. Recognizing the possibility of phthisis being conveyed from one individual to another, as well as for other reasons, phthisical patients should sleep alone, if practicable; and if others sleep in the same room, particular attention should be paid to having a good-sized and well-ventilated bedroom.

**3.** Before proceeding to the active treatment of any case of phthisis, it is of the greatest consequence to look to the state of the **digestive organs**. Unless digestion is carried on properly, all other means are of little or no avail; and here it must be mentioned that regularity of meals and other matters upon which healthy digestion depends should receive due consideration. Appetite must be promoted; and if any form of dyspepsia is present, the appropriate remedies must be administered. Should there be signs of gastric irritation, a combination of bismuth with an alkaline carbonate and hydrocyanic acid frequently proves very beneficial; effervescent mixtures are also useful in some cases. In the early period the bowels are often confined, and some mild *aperient* must then be given, so that they may be opened daily.

**4. General medicinal treatment.**—Various *tonic* and other medicines which improve the condition of the general health and the quality of the blood are very serviceable in phthisis. Of these the principal are the dilute mineral acids,—nitric, hydrochloric, sulphuric, or phosphoric; quinine; different preparations of iron, especially if the patient is anæmic; salicine; strychnia; and vegetable bitter infusions or tinctures,

such as those of gentian, calumba, chiretta, quassia, or cascarilla. These may be given in various combinations.

**5. Special medicines.**—Among the many special therapeutic agents recommended for phthisis, cod-liver oil demands the first consideration. Almost universal experience has testified to its good effects in this disease. It is needless to enter here into the question of its mode of action, but certain matters of practical importance as regards its administration must be mentioned. Only a small dose should be given at first, not too often repeated. A teaspoonful once or twice a day is sufficient to commence with, the dose being increased by degrees to a tablespoonful three times daily. It is seldom desirable to exceed this quantity. Most patients take the oil best immediately or soon after meals, and if it tends to disagree, lying down for a short time after taking it will not infrequently prevent any ill effects. Some can manage it best when going to bed at night. It is always well to make use of some vehicle for administering the oil, even when it can be taken alone, but the quantity of this should not be large. It may in many cases be given with the mixture ordinarily taken, if this is of a bitter or acid nature; or with steel wine, or syrup of phosphate or iodide of iron. Milk, orange wine, frothy stout or ale, or a little cold brandy-and-water are among the most useful vehicles. When the oil repeats or causes sickness, it is often well borne when given with lime-water and milk in equal parts, some of which may also be drunk after it. For children it may be made into an emulsion or mixture according to the following formula, which is employed at the Brompton Hospital:—*R* Ol. morrhue ʒ vi, Liq. potassae ℥ xl, Liq. ammon. fort. ℥ ij, Ol. cassiæ ℥ j, Syrupi ʒ ij; dose, two teaspoonfuls. Small doses of strychnine have been found very useful in preventing the nauseating effects of the oil. It is most important to look to its quality, especially at the outset, otherwise a patient may acquire an unsurmountable antipathy to it. Different varieties are preferred by different practitioners. A good pale oil seems to answer best generally; many patients like De Jongh's pale brown oil very well. Regularity and perseverance in the use of the remedy are essential in order to realize the effects which it is capable of producing. During its administration the diet must be carefully attended to, and should not be of too rich a character. If from time to time the oil appears to disagree with the digestive organs, it may be temporarily omitted, especially during the warmer months. It has been recommended to introduce cod-liver oil by inunction or enema, but though necessary under some circumstances, these modes of administration are objectionable as a rule. Inunction is often advantageously employed in the treatment of children. Several preparations containing cod-liver oil have been made, such as the etherized oil, which is a valuable compound, or a combination with quinine, hypophosphite of lime, extract of malt, and numerous other medicines.

Numerous substances have been advocated as substitutes for cod-liver oil, but they are far less efficacious. The chief of these are olive-oil; skate, shark, or dugong oils; cocoa-nut oil; dog's fat; glycerine; and cream. The last two certainly produce good effects in some cases. Chaulmoogra oil has also been particularly recommended in phthisis.

Space will only permit the enumeration of some other special agents recommended in the treatment of phthisis. The principal are pancreatic emulsion; hypophosphites of lime, soda, and iron; phosphate of lime; extract of malt or maltine; iodide of potassium; iodide of iron;



iodoform; sulphurous acid and sulphites; chloride and sulphide of calcium; arsenic; verbascum thapsus or the great mullein plant; and koumiss. Although most of these drugs are useful for certain purposes, they are in no sense to be looked upon as specific remedies.

**6. Local treatment.**—Applications to the chest are decidedly useful in many cases of phthisis, either for the relief of symptoms, or for the subdual of inflammatory processes; or possibly they may have an immediate effect on some forms of the disease. The most useful are sinapisms; small or flying blisters; applications of iodine, more or less powerful; and liniments of croton oil, turpentine, or acetic acid. Local removal of blood is decidedly but seldom desirable. In acute exacerbations fomentations and poultices are often required. In some cases the application of strapping over parts of the chest, in order to procure mechanical rest, has a beneficial effect as regards the progress of phthisis.

**7. Symptoms and complications.**—Various symptoms frequently need attention during the course of a case of phthisis, but it is impossible to do more here than point out their nature, and suggest the indications for their management, most of them being considered in detail in other parts of this work. Pyrexia must be subdued, especially if it is inclined to be high. Quinine in full doses combined with digitalis may be given for this purpose. Sponging the skin, or the employment of cold baths may prove beneficial in some cases. The application of iodoform to the front of the chest has been tried to lower temperature. Debility and wasting will be counteracted by the general treatment already indicated, as well as by subduing the fever. When there is much exhaustion, considerable quantities of alcoholic and other stimulants are required. For night-sweats the chief remedies are oxide of zinc, gr. ij-v, in the form of pill or powder given at night, which may be combined with extract of belladonna or morphia; tincture of belladonna, or subcutaneous injection of atropine; or a full dose of quinine or gallic acid. Subcutaneous injection of ergotone has also been employed with advantage. In some cases I have found that the night-sweats were best checked by the administration for a few days of a mixture containing quinine, alum, and dilute sulphuric acid. Sponging the upper part of the body carefully with vinegar and water is sometimes useful. Dr. Murrell has recommended picrotoxine, gr.  $\frac{1}{60}$ th, in pill; or  $\mathfrak{m}$  v of a one per cent. solution of muscarine, taken at bed-time. Pilocarpin and agaricus or agaricin are also used. Pains about the chest are often relieved by the local applications already mentioned, or by wearing some anodyne or warm plaster; in a considerable number of cases much pain in the side is complained of from time to time, either muscular or pleuritic, and this is almost invariably at once relieved by strapping the side properly, as described under pleurisy, which is the plan of treatment I usually adopt under such circumstances. Cough is often a most troublesome symptom in phthisis. It is by no means always desirable to stop it, but its management must be guided by the amount of expectoration, the discharge of which is to be encouraged, or its amount diminished, by means of remedies mentioned under bronchitis. In most cases cough needs to be relieved, and it is always advisable first to look to the state of the throat and larynx, as cough is very commonly due to some unhealthy condition of these parts. Local applications of tannin or chlorate of potash; or various *astringent* gargles or lozenges are often most beneficial. Ice is often of much service, as well as



simple demulcent lozenges. If the cough is irritable, *anodynes* are valuable, especially opium, morphia, codeia, hydrate of chloral, croton-chloral, bromide of ammonium, conium, belladonna, or chlorodyne, some of which may be combined. These are best given in the form of lozenges, syrups, or linctuses, and it is desirable to make all cough mixtures as small as possible. Among other remedies specially recommended for cough in phthisis are tincture of gelsemium, lactic acid ( $\mathfrak{m}$  x bis die), and prunus virginianus. Anodyne and other inhalations are in many cases extremely serviceable, but not on account of any curative influence upon the disease. They are particularly useful if the larynx is affected. *Antiseptic* inhalations are also very valuable in some conditions, and especially if the expectoration is foetid. Weak iodine inhalations are sometimes decidedly beneficial. Dyspnoea and hæmoptysis must be treated according to the ordinary principles. Vomiting is sometimes a very distressing symptom; if the ordinary remedies fail, small doses of strychnia should be tried, and its effects are in some cases most satisfactory. Koumiss has also been found useful in the treatment of the sickness of phthisis. Diarrhoea, if due to ulceration of the bowels, is frequently very difficult to check. Carbonate of bismuth, gr. v-x, with Dover's powder, gr. iij-v, is often a useful combination; but enemata of starch and opium are most to be relied upon in obstinate cases. Coto bark and its active principles have been specially advocated in the treatment of this symptom. Sulpho-carbolate of soda has been found useful in the treatment of dyspepsia in cases of phthisis. Other symptoms and complications must be attended to as they arise.

8. **Change of climate, and sea-voyages.**—This is a most important subject in connection with the treatment of phthisis, and for detailed information the reader is referred to special works on the subject. In selecting a suitable climate, the chief points to be observed are that it is not liable to either extreme of temperature; that the air is pure and not too moist; that the soil is healthy; and that there is no likelihood of sudden changes, of exposure to cold winds, or of continued unfavourable weather. It is always well also to choose a place rendered attractive by bright sunshine, pretty scenery, and pleasant company. One most important object to be kept in mind in selecting a climate is, that the patient may be enabled to be out in the open air as much as possible. The salutary influence of high altitudes upon phthisis has now been established, and in appropriate cases a residence in mountainous districts proves most beneficial, even during the winter. The aseptic character of the air in these regions is an important element in their usefulness, and this has now assumed special prominence in relation to the antiseptic treatment of phthisis. The exact qualities of the climate which are suitable for any individual case will depend upon its mode of origin, upon the condition of the bronchial mucous membrane, and other circumstances. Those cases of phthisis which are of constitutional origin seem to be particularly benefited by a sojourn in lofty regions. The principal sea-side places to which phthisical invalids resort are the Isle of Wight, especially Ventnor and Undercliff, Bournemouth, Torquay, Hastings, St. Leonards, Eastbourne, Penzance, Worthing, Sidmouth, Cromer, Southport, Grange, Clevedon, Tenby, and Queenstown in this country, where the temperature is moderate, but moisture considerable; Cannes, Nice, Monaco, Mentone, Bordighera, San Remo, Palermo, Malaga, Malta, or Algiers, where there is a high temperature, with but little moisture; Madeira, West Indies, and the Azores, where

both temperature and moisture are considerable. Among inland regions, Pau, Pisa, Upper Egypt, Syria, and parts of Australia, New Zealand, or South Africa are recommended; or if elevated districts are desired, the Alps, Andes, Himalayas, or the Mexican mountain ranges afford the requisite conditions. Among the places which are now in favour, Davos Platz, Wiesen, and St. Moritz, in Switzerland, deserve special notice. Corsica and Sicily are also favourably spoken of as winter resorts.

Long voyages, especially to Australia, or up the Mediterranean, are most useful in many cases, but they should not be recommended if the disease is too far advanced. The purity of the air is doubtless one element in the benefit derived from sea-voyages.

A large number of patients are unable to avail themselves of the benefits to be derived from a suitable climate, although, thanks to the various hospitals established in many sea-side places in this country, these advantages are more widely disseminated at present than they were formerly. If during the winter months patients are prevented by circumstances from residing in a proper climate, they should keep indoors as much as possible in bad weather and at night; and avoid every cause of cold. The use of some covering over the mouth and nostrils is often of great service, and a proper respirator may be required, but these appliances must be employed judiciously. Men suffering from consumption may with advantage allow their beard and moustache to grow.

9. **Special treatment.**—Innumerable special modes of treatment have been advocated for the supposed cure of phthisis, but it need scarcely be said that most of them have no rational foundation, and none of them can be looked upon as having any positive curative influence. Amongst these may be mentioned treatment by mineral waters, especially the sulphur springs of Eaux Bonnes, Caunterets, &c., and the arsenical waters of La Bourboule and Royat; by compressed air; by the inhalation of oxygen; and by electricity. The so-call "grape-cure" may also be alluded to. At the present time *antiseptic* treatment is still the fashion in the treatment of phthisis. This treatment has long been carried out to a certain extent, but it is now especially advocated on account of the presence of bacilli, the growth and development of which antiseptics are supposed to check. These agents are given internally, and also employed in various modes of inhalation. The principal *antiseptics* administered internally are creosote, which is conveniently combined with cod-liver oil or glycerine; carbolic acid or sulpho-carbolates; and benzoate of soda. Inhalation may be effected to some extent by merely impregnating more or less the air which the patient breathes with some antiseptic vapour, such as that of carbolic acid, creosote, tar, turpentine, or iodine. It is a common custom now to have saucers containing carbolic acid in the vicinity of patients suffering from consumption; while instruments have also been devised for the purpose of diffusing antiseptic vapours in rooms or hospital-wards. Moreover, the benefit said to be derived from a residence in districts where there are pine-forests is attributed to the volatile materials diffused in the atmosphere. The more direct methods of application are either by inhalation with steam; by the use of Siegle's spray-inhaler; by inhaling vapours; or by the employment of some form of "antiseptic-respirator," so that the agent used may be inhaled for a considerable time, or even continuously. The chief antiseptics thus employed are carbolic acid, creosote, a two to five

per cent. solution of benzoate of soda in spray; thymol, eucalyptol, camphor, terebene, oleum pini sylvestris, spirit of turpentine (especially useful where there is excessive secretion or a tendency to hæmoptysis), solution of tar in rectified spirit, tincture of iodine, iodoform, chlorine, tincture of benzoin, and sulphurous acid, either derived from burning sulphur or in the form of a spray of the solution. Some of these agents may be used in combination, and they may also be advantageously mixed with spirits of chloroform or ether in some cases. Antiseptic treatment must be practised judiciously, while other appropriate measures are at the same time not neglected, according to the indications present in each individual case. When thus conducted, it may undoubtedly lead to highly beneficial results in appropriate cases. It has been suggested, as the outcome of certain experiments, to inject antiseptics into the foci of disease in the lungs, and in their vicinity, with the view of modifying the phthisical process, and limiting its extension by the formation of cicatricial tissue. Such a method of treatment is of doubtful propriety, and at present, at any rate, would be seldom justifiable. In relation to the antiseptic treatment of phthisis, it may be mentioned that patients should be warned against swallowing their sputa, and that these should be received into a vessel containing some disinfectant, and soon destroyed.

10. **Operations.**—The question of emptying and draining pulmonary cavities through the chest-walls by surgical treatment may in exceptional cases be worthy of consideration, but only under special circumstances. The actual removal of portions of diseased lung has been proposed and practised, but can hardly be recognized as a legitimate method of treatment.

---

## CHAPTER XVI.

### MORBID GROWTHS IN THE LUNGS.

In this chapter it is proposed to discuss briefly the morbid formations met with in the lungs which have not yet been considered, and they may be taken in the following order:—1. CANCER. 2. SYPHILITIC DISEASE. 3. RARE MORBID FORMATIONS.

#### I. CANCER OR CARCINOMA—MALIGNANT DISEASE.

**ÆTIOLOGY.**—Cancer of the lungs, which is exceedingly rare, is by far most common from 40 to 60 years of age; and more males are affected than females. The diathesis may be inherited. In the large majority of cases pulmonary cancer is secondary, especially following cancer of the bones or testicles, but it may result from direct extension, or be primary in its origin. The morbid condition generally extends so as to involve neighbouring parts; but is very rarely followed by secondary cancerous formations in other internal organs.

**ANATOMICAL CHARACTERS.**—*Encephaloid* is the variety of cancer usually found in the lungs, and it is often extremely soft, pulpy, and vascular.



Other forms are occasionally met with, alone or in combination; and considerable deposit of black pigment may be observed, constituting *melanotic cancer*.

*Secondary* cancer assumes almost always the nodular form, and affects both lungs; the nodules vary much in size, and when situated at the surface tend to be depressed; by their union a lung may be involved throughout. *Primary* cancer is particularly prone to be confined to one lung, especially the right, and is often infiltrated. After a time the cancerous material undergoes fatty degeneration and softening, cavities being formed in some cases; and extravasations of blood into the substance of a growth are common. The vessels and bronchi are often either involved in the disease, or obliterated by pressure. The unaffected portions of the lung-texture may be normal, or various morbid changes may be set up in them. A cancerous lung feels remarkably heavy. Extensive pleuritic adhesions are usually observed.

**SYMPTOMS.**—Secondary cancer tends to come on insidiously, without any subjective symptoms. I have seen a case in which the left lung was involved almost throughout, and the right also extensively, the only symptoms being occasional cough, and shortness of breath on exertion. In primary cancer there is usually pain in the chest, which may be extremely severe, of a lancinating character, and accompanied with tenderness. Cough is generally present, attended in some cases with a peculiar expectoration, in the form of a substance resembling red or black-currant jelly, or occasionally containing cancer-elements. Hæmoptysis is very common. Dyspnoea is generally observed, being especially severe if there are projecting nodules pressing on the nerves, or if the cancer is associated with a mediastinal tumour, when other signs of pressure on neighbouring structures are observed.

The *general* symptoms are not usually so marked as might be expected. The cancerous cachexia may or may not be evident. Emaciation, fever, night-sweats, and failure of strength are generally present more or less, but they may be comparatively very slight, especially in cases of secondary cancer. Wasting is sometimes extremely rapid in its progress when it has once commenced.

**PHYSICAL SIGNS.**—These will vary according to the form, seat, and amount of the cancerous accumulation; and whether it is associated with a mediastinal tumour or not. Where there are scattered nodules, there may be no signs, or only slight alterations in the percussion and respiratory sounds. If a lung is extensively involved with *nodular* cancer, being ultimately converted into a mass of encephaloid, the signs are:—1. Enlargement of the chest on the affected side, with widened and flattened spaces, the surface feeling unusually even, but without any sense of fluctuation.—2. Great deficiency or entire absence of movement. 3. Weakened or annulled vocal fremitus. 4. Absolute dullness, unaltered by position, with great sense of resistance. 5. Weakened or absent breath-sounds over a variable area. 6. Deficient vocal resonance. 7. Displacement of heart or diaphragm, the cardiac sounds being frequently conducted with an unusual degree of intensity. In the *infiltrated* form the lung is contracted, and physical examination reveals:—1. Retraction of the side, with depression of the intercostal spaces. 2. Deficient movement, the spaces still acting, however. 3. Increased, diminished, or absent vocal fremitus, according to the amount of consolidation. 4. Hard, wooden, high-pitched, or tubular percussion, which may extend across the middle line. 5. Bronchial, blowing, or feeble respiratory

sounds. 6. Often intensified vocal resonance. 7. Displacement of the heart, either towards the affected or opposite side, with intensification of the sounds; and drawing-up of the diaphragm. Ultimately there may be signs of cavities. In the non-cancerous parts signs of hypertrophy, bronchitis, emphysema, or collapse are usually present.

PROGNOSIS is necessarily fatal in cancer of the lung, death occurring either from local or general causes.

TREATMENT can only be palliative, the usual remedies being employed for the relief of symptoms as they arise.

## II. SYPHILITIC DISEASE.

My own experience leads me to the conclusion that there is an important influence exercised by syphilis in the causation of ordinary phthisis in a certain proportion of cases. Clinical and pathological observations have, however, clearly demonstrated that occasionally the lungs may be the seat of special lesions of a syphilitic nature, which call for brief consideration as constituting one of the varieties of morbid growths found in these organs. These are usually associated with well-marked evidences of the effects of the disease upon other organs and tissues, the clinical results of which are often so prominent that they obscure the pulmonary affection. It has been suggested that a tubercular or scrofulous diathesis may aid in the development of these special manifestations of syphilis in the lungs.

ANATOMICAL CHARACTERS.—In new-born or very young infants who are the subjects of congenital syphilis, a morbid condition of lung has been described under various names,—*syphilitic pneumonia*, *white hepatisation*, *epithelioma of the lungs*, &c., and attributed to the influence of the syphilitic poison. Its extent is variable, and one or both organs may be involved, but the disease presents a more or less diffuse or infiltrated character. In this condition the organ is distended, and in a state of full expansion, so that its surface may be marked by the ribs. The pleura is generally unaffected. The lung is very heavy, and the involved portion of its tissue is dense, firm, hard, and usually resistant, being of a white or yellowish-white colour, uniform and smooth on section. Little or no fluid can be expressed or scraped from the cut surface. The affected parts become bloodless, and the vessels disappear. On close examination of a section minute bands of fibrous tissue may be seen running in all directions. Microscopically most observers have detected increase of the epithelial elements, which fill the small tubes and air-vesicles, but Wagner affirms that the epithelial lining is but little affected. All seem agreed that there is considerable thickening of the alveolar walls and minute bronchi, due to an imperfectly fibrillated and nucleated tissue, which undergoes degenerative changes. Prof. Greenfield has described minute bands of highly-vascular fibrous tissue running in all directions, and enclosing groups of alveoli. The walls of the vessels are also thickened, and in course of time their channels become obliterated.

The most characteristic morbid change recognized as being of a syphilitic nature is the presence of *gummata* in the substance of the lungs. They have been found in syphilitic infants, and also in adults, though rarely. There may be but one of these growths, or a variable number may be found scattered through the organs indiscriminately,

though they are said to be more common in the deeper parts (Wagner). In size gummata in the lungs usually vary from a pea to a walnut, but they may attain the size of a large egg. They are generally rounded, well-defined, and often encapsuled. At first they appear greyish or brownish-red, homogeneous-looking, firm, and dryish. Like other gummatous growths they tend to undergo degenerative changes, becoming yellow and caseous. Occasionally they soften in the centre, so as to form cavities. Microscopically their structure is found to consist of imperfect fibres, abortive nuclei, and a few fibre-cells, infiltrating the lung-structure, and thickening the alveoli; mixed with degenerative products.

Another condition usually believed to be due to syphilis, but about which there has been much dispute, consists in a *chronic interstitial pneumonia*, leading to fibroid infiltration of the lungs, variously distributed, and causing much induration. The bases and roots of these organs seem to be more affected than the apices. Often the growth starts from the surface, the pleura being in most cases thickened or adherent, and penetrates thence into the interior in the form of fibrous bands. The lung consequently presents superficial puckerings and depressions. In other cases the morbid change appears to start from gummata, or from a chronic contracting peri-bronchitis, associated with ulcerative inflammation (Pye-Smith). It does not terminate in caseation, but ulceration or gangrene may occur. The involved bronchi become more or less dilated. Anatomically and histologically syphilitic fibroid infiltration cannot be distinguished from other forms of interstitial pneumonia, but probably the tissue is more vascular in the early stages. Dr. Green thinks that its mode of growth is more valuable in determining the nature of the pulmonary induration than its characters, and that it originates mainly around the small interlobular blood-vessels.

The bronchial tubes may be the seat of a fibro-nucleated growth infiltrating their sub-mucous tissue, or sometimes involving their deeper structures. They are also liable to ulcerations, similar in character to those met with in the larynx and trachea, and when these cicatrize, they cause more or less narrowing and thickening of the tubes, or even complete stenosis.

**SYMPTOMS.**—In most cases syphilitic lesions of the lungs have only been found on *post-mortem* examination, either in newly-born children, or in individuals who have not presented any pulmonary symptoms during life. In some instances they have, however, been recognized clinically, and the data which might lead to the diagnosis of syphilitic disease of the lung would be a history of syphilis, or evidences of constitutional taint or of implication of other organs; accompanied with symptoms of chronic lung-disease, including hæmoptysis at an early period; and *physical signs* indicating marked induration of the lung, especially if limited to one side and involving the middle or lower part of the organ. Signs of excavation may also become developed. The progress is very chronic; and there is little or no fever. Treatment may aid the diagnosis, if it should happen that marked improvement follows the administration of anti-syphilitic remedies. If a main bronchus should be obstructed, serious interference with breathing is liable to arise.

**TREATMENT.**—This consists either in the employment of a mercurial course, or in the administration freely of iodide of potassium, the latter



being the course of treatment usually indicated. Symptoms connected with the respiratory organs may need to be relieved.

### III. RARE MORBID FORMATIONS.

*Hydatids* are occasionally found in the lungs, and they have been noticed in Australasia in these organs in disproportionately large numbers, as compared with other parts of the world. According to the tables prepared by Dr. Davies Thomas, of Adelaide, the ratio of lung to liver cases in Australasia is 1 to 4, while in Europe it is only 1 to 6½. Hydatids may make their way from the liver into the right lung. They present the usual characters of hydatid-cysts, and may rupture and discharge their contents, or become inflamed and suppurate, forming pulmonary cavities. Their presence might be suspected if, along with signs of hydatids in other organs, *physical signs* were observed in connection with the chest, characteristic of localized accumulation of fluid. The expulsion of the hydatid membrane or of echinococci in the expectoration would be the only positive indication of the existence of hydatid disease of the lung, of which I have only met with one example. As the lung-structure becomes destroyed, the symptoms and physical signs assume the characters of phthisis. Treatment must be conducted on general principles.

*Sarcomata*, *enchondromata*, *osteoid*, and *myeloid* tumours have also been found rarely in the lungs, as well as *hæmatomata*, but they have only been discovered usually at *post-mortem* examinations.

## CHAPTER XVII.

### DISEASES OF THE PLEURA.

#### I. PLEURISY—INFLAMMATION OF THE PLEURA.

**ÆTIOLOGY.—Exciting causes.** The causes of pleurisy may be enumerated thus:—1. *Direct irritation* of the pleura, from injury; foreign matters which have gained access into its cavity, for example, pus or air; local deposits in the pleura, such as cancer or tubercle; diseased bone; friction or pressure by tumours. 2. *Cold* or other injurious meteorological influences, though many deny any such mode of causation. 3. Possibly “extreme muscular over-exertion and exertion in continuous public speaking” (Anstie). 4. *Extension* from neighbouring parts, such as the pericardium; under which class of causes may be also mentioned the pleurisy which accompanies pneumonia in most cases, or which complicates other pulmonary affections, especially phthisis. 5. *Blood-poisoning*, in connection with various acute febrile diseases, especially scarlatina, typhoid, puerperal fever, or acute rheumatism; pyæmia and septicæmia; Bright’s disease; or alcoholism.

According to its mode of origin pleurisy has been divided into

*primary* or *idiopathic*, and *secondary*. The former is due to some cause acting immediately on the pleura, the patient having previously been in good health; the latter is the result either of some constitutional affection, or of previous visceral organic disease. In the latter case, however, it is not improbable that the inflammation may in some instances be brought about by a slight exciting cause, acting upon a depraved constitution which greatly predisposes to serous inflammations. Of other *predisposing causes* but little is known. Pleurisy may occur at any age; it is not an uncommon disease in children. Probably it is most prevalent during the colder seasons.

**ANATOMICAL CHARACTERS.**—Like other inflammations of serous membranes, pleurisy, if it is at all extensive and runs a regular course, is characterized by the stages of vascularization; lymph-exudation; fluid-effusion; absorption; and adhesion. The costal pleura seems to be first affected as a rule. At the outset the anatomical characters include bright redness from capillary injection, often with spots of extravasation; dryness and loss of polish of the membrane; with thickening, cloudiness, and diminution in consistence. Then lymph-exudation covers the surface more or less extensively, varying in quantity and characters, and being usually stratified. A sero-fibrinous fluid begins to accumulate in the pleural sac, in some cases from a very early period, in which float fibrinous flocculi, its amount varying exceedingly, and it may be so abundant as to fill the sac completely. More or less blood may be present, as well as gas, the latter being probably due to decomposition. The epithelial cells of the membrane undergo proliferation, and the newly-formed cells are seen in the exudation and fluid. If the termination is favourable, the effusion is absorbed, much of the exudation is also taken up after undergoing degenerative changes, while the remainder becomes organized into adhesions or agglutinations, these also sometimes developing from papillary vascular growths which arise from the sub-epithelial tissue.

In some cases, owing to an unhealthy condition of the system or other causes, the fluid is not absorbed, but remains and becomes more or less purulent. The exudation may also become caseous; or calcification sometimes occurs in it.

The lung, if not previously consolidated, is first floated forwards and relaxed, and afterwards compressed, until ultimately it becomes completely carnified. If the pressure is soon removed, the lung will expand again; otherwise, it is in danger of being rendered permanently useless, or of undergoing further destructive changes.

In a good proportion of cases pleurisy is limited to a very small patch, which is covered with a slight exudation, and an adhesion soon forms. In a few instances I have observed distinct evidence of extensive formation of lymph, with little or no fluid effusion. Sometimes the serum is "loculated" by adhesions. Rarely pleurisy is bilateral, being then generally associated with some constitutional diathesis.

By *chronic pleurisy* is usually meant either extensive adhesion of the pleural surfaces, with falling-in of the side, the result of an acute attack; or a condition in which the effusion remains and cannot be absorbed, being either serous or purulent, and in either case named *empyæma*; or where an opening has been formed, through which there is a permanent discharge, either externally—*fistulous empyæma*, into the bronchi, or, very rarely, into the bowels. Occasionally the disease seems to be chronic in its origin, especially when of a secondary nature;

under this class of cases of pleurisy might also be included those in which there is a tendency to repeated limited attacks.

If there is abundant effusion, the neighbouring organs will be found displaced, especially the heart in cases of left pleurisy. This is partly due to pressure; partly to elastic traction on the part of the lung which is free to act. Dr. Douglas Powell affirms that the axis of the heart can never diverge beyond the vertical line, or only to a very slight degree, so that the apex does not point to the right, which is contradictory of the statements made by other observers. The right side of the heart and general venous system are often overloaded.

**SYMPTOMS.**—In all serous inflammations the symptoms observed are of three kinds, namely, first, those directly due to the affection of the membrane itself, and of the tissues immediately adjoining; secondly, those resulting from the mechanical pressure of the inflammatory products on neighbouring organs and structures; and thirdly, those indicating constitutional disturbance. Much variety is presented in the intensity of the symptoms of pleurisy, and that by no means always in proportion to the gravity of the attack. In many instances, which are common enough in hospital out-patient practice, where the disease is localized in a small patch, the one prominent symptom is a “stitch in the side,” which may be very severe, increased by breathing deeply or coughing, as well as often by pressure, the patient being disposed to lean towards the affected side, which is kept as much at rest as possible. There are no general symptoms.

A typical case of acute primary pleurisy with effusion has the following clinical history. At the outset several chills are generally felt, not of a severe character; accompanied or soon followed by certain *local* and *general* symptoms.

**Local.**—Acute pain is felt, usually in the infra-mammary or infra-axillary region, of a dragging, catching, or stitch-like character, increased by breathing or coughing, and often attended with superficial or deep tenderness. The sharpness of the pain is often evidenced in the expression, posture, and mode of breathing of the patient. Respiration is carried on in a hurried, shallow, and irregular manner, but there is no actual dyspnoea at first, and the number of respirations is rarely above from 30 to 35. Later on evident dyspnoea is observed, should much fluid be poured out, which varies in its degree, being in some cases very severe or even urgent. Cough is generally present, though the patient tries to repress it; it is short and hacking, generally dry, or at least unattended with any particular expectoration. Sometimes a cough is excited by making the patient sit up or bend forward. At first the patient usually prefers to lie on the affected side, but later on there is no uniformity, for it is not uncommon to see patients with one pleura full of fluid habitually rest on the healthy side.

**General.**—Pyrexia is observed, but it is not very marked, and the temperature has no typical course. The pulse is frequent, varying usually from 90 to 120, full and bounding, but deficient in resistance, as evidenced by the sphygmograph (Anstie). The pulse-respiration ratio is altered somewhat, but not to any great degree. There is but little prostration. Disturbance of the digestive organs, headache, and other symptoms associated with the febrile state are present in various degrees. The urine may be slightly albuminous.

**COURSE AND PROGRESS.**—In favourable cases of pleurisy the symptoms subside in a few days, and the effusion is absorbed. Should this not



happen, the only remaining symptom in many instances is a little dyspnoea or shortness of breath, and this may be the case even when there is abundant fluid in the pleura. Ultimately the fluid may in time be taken up, or be discharged through the bronchi or externally, unless it is removed by operation. In cases of *chronic effusion* pyrexia often continues, the skin being hot, dry, and harsh, and the pulse frequent but weak. The patient wastes and becomes much debilitated. Œdema of the affected side, and extreme clubbing of the finger ends are sometimes noticed. *Fistulous empyæma* is generally attended with great weakness, loss of flesh, and a tendency to hectic fever. The hair frequently falls off. Ultimately symptoms of phthisis or tuberculosis may arise. The formation of pus has been supposed to be indicated by repeated rigors, but certainly this is not always the case. Perforation into the bronchi is attended with profuse expectoration of matter; which is usually repeated at intervals. Should extensive adhesions form, with retraction of the side, more or less shortness of breath remains, with a liability to pains on the side affected, and often some degree of debility.

It is very important to notice that extensive effusion may be poured out when there have been no particular symptoms to draw attention to the chest—*latent pleurisy*; and this event is especially liable to happen in secondary pleurisy, or when the complaint occurs in children, but it may be observed in any case. *Bilateral pleurisy* is necessarily a serious condition, and is attended with dangerous dyspnoea. *Diaphragmatic pleurisy* probably gives rise to very severe pain around the lower part of the chest; and also interferes greatly with the act of breathing.

PHYSICAL SIGNS.—In the early stage of pleurisy the only reliable physical signs are:—1. *Diminished movements* on the affected side, on account of pain. 2. *Friction-fremitus*, which is exceedingly rare. 3. *Friction-sound*, at first slight and grazing, but becoming much louder when lymph has been deposited. It may be limited to a small spot; or be heard more or less extensively over the side.

The signs attending the stage of *fluid-effusion* are usually quite characteristic, being, however, considerably modified by its quantity and mode of accumulation. As a rule the evidences of effusion are first observed over the lower part of the chest, and they extend upwards more or less rapidly. 1. The side is *enlarged* to a variable degree, and the spaces are often specially affected, being either flattened or bulged out. It is important to make use of the *cyrtometer* in determining this enlargement, as the measurement may be actually less on the affected than on the healthy side. 2. *Movement* is diminished or almost completely annulled. 3. *Vocal fremitus* is deficient or absent below, in excess above; there being frequently an abrupt transition from the one condition to the other, particularly in front. 4. *Fluctuation* may occasionally be detected. 5. *Percussion-sound* is dull over the area of the fluid. Beginning below, the dullness may ultimately extend over the whole side, and beyond the middle line for some distance. If the patient has assumed the recumbent posture at an early period dullness is sometimes noticed over the whole of the back, before any alteration in percussion-sound is observed in front. It may be movable with a change of posture of the patient, but frequently this is not the case. In many instances an abnormally clear or tubular sound can be elicited under the clavicle at a certain stage, and the transition from dullness to this sound may be quite abrupt; occasionally percussion here

elicits a kind of crack-pot sound. 6. *Breath-sounds* are absent or feeble below; exaggerated or even blowing or tubular above. 7. *Friction-sound* may or may not be heard at the margin of the dulness. 8. *Vocal resonance* is diminished or annulled below, increased above, the change from one to the other being often marked. *Ægophony* is heard in some cases, especially about the angle of the scapula. Dr. Bacelli has affirmed that, in cases of pleuritic effusion, if the whispered voice is conducted well through the thickness of the fluid, and is pectoriloquous in character, the effusion is serous; if it is ill-conducted or inaudible, the fluid is purulent. Dr. Douglas Powell concludes, as the result of his observations, that although of considerable value in association with other signs, Bacelli's sign is by no means pathognomonic. 9. *Displacement of organs* is an important sign of pleuritic effusion, especially of the heart. Its impulse may be noticed far over on the right side in cases of left pleurisy, while its sounds are very loud here. This impulse, however, is probably connected with the right ventricle. The diaphragm, with the liver, spleen, or stomach, may also be depressed. Some cases have come under my notice in which a cardiac murmur seemed to be due to displacement. 10. Rarely *succussion* gives rise to a splashing sensation or sound, owing to the presence of air and fluid in the pleura.

*Absorption* may be traced by the gradual subsidence of the signs described, and their restoration to the normal; often accompanied with the development of a loud *redux friction-sound*, and sometimes with *friction-fremitus*. Dulness may continue for some time. In favourable cases the side resumes its proper form and size, and the lung expands. The heart occasionally remains in its abnormal position, owing to the formation of adhesions; or goes too far in the opposite direction; or lies more or less freely movable in the chest. Should the lung remain unexpanded the signs are:—1. *General retraction* of the side, the ribs being crowded together; the shoulder lowered; all the diameters of the chest diminished, especially the antero-posterior; and the spine curved, usually to the diseased, occasionally to the healthy side. 2. *Movements* null or greatly lessened. 3. *Deficient resonance* on percussion. 4. Feeble *respiratory sounds* over the side generally; or in some parts of bronchial quality. *Fistulous empyæma* is followed by extreme retraction of the side. If the lung is permanently condensed signs may subsequently be noticed indicating that the organ has undergone destructive processes.

The signs of fluid are now and then observed on both sides. On the other hand, they are limited in the *loculated* variety of pleurisy, which may cause local bulging. The fluid sometimes makes its way to the surface, and even points like an abscess; or in rare instances it exhibits pulsation when in the neighbourhood of the heart. Should an empyæma open into the bronchi, râles will be heard over the lung, and sometimes pneumothorax is produced. In diaphragmatic pleurisy there may be no physical signs, except cessation of all abdominal movements during respiration.

In children some important modifications of the ordinary physical signs of pleuritic effusion are noticed. The chest being markedly yielding, is dilated considerably and at a very early period, while the organs are comparatively less displaced than in adults. Bronchial breathing and vocal resonance often persist even when the thorax is apparently full of fluid, as judged by the dulness. This is noticed in adults in rare instances, or the breathing may be actually tubular.

Old adhesions resulting from previous attacks may influence the signs of pleurisy considerably; as well as morbid conditions of the lung with which it may be associated.

TERMINATIONS.—1. *Recovery* takes place in a large proportion of cases of pleurisy, after absorption or removal by operation of any fluid, the lung expanding fairly, but being more or less adherent. 2. *Death* is a rare event in acute cases, unless the pleurisy is bilateral, or is associated with some serious constitutional or local disease. It may happen, however, from the mere mechanical effects of the fluid effusion, accompanied with pulmonary congestion and œdema, and there is a danger of sudden death should there be urgent dyspnoea, or if the heart is much embarrassed. 3. Transition into *chronic pleurisy* is not uncommonly observed. Under this would be included:—*a.* Chronic effusion. *b.* Retraction of the side from extensive adhesions, with permanent binding down of the lung. *c.* Chronic purulent discharge, either by an external opening; through the air-passages; or in some unusual direction, such as into the intestines. In these cases the patient may ultimately either sink from gradual asthenia, or may become phthisical; or sometimes recovery follows, though with more or less permanent loss of the use of the lung on the affected side.

DIAGNOSIS.—This subject will be considered later on, and here it need only be mentioned that pleurisy has not merely to be distinguished from other affections of the lung or pleura, but that it may be simulated at first by painful affections of the chest-walls; or, in the stage of fluid-effusion, by enlargements of the liver or spleen, hydatids of the liver, or a large tumour within the chest. Careful *physical examination* has mainly to be relied upon in diagnosis.

PROGNOSIS.—This involves not only the immediate result of the disease, but also its ultimate issue. Primary pleurisy ought to terminate favourably in the great majority of cases, if properly managed. It is more serious in proportion to the amount of fluid poured out; to the time that it has remained in the pleural cavity; and to its tendency to become purulent. Severe dyspnoea is a dangerous sign. The thermometer and sphygmograph may be of use in aiding towards a prognosis. Pleurisy secondary to constitutional diseases is very grave; also when it occurs in advanced cases of chronic alcoholism. Bilateral pleurisy with effusion is necessarily most dangerous. The different forms of chronic pleurisy are often unfavourable.

Discharge of pleuritic fluid through the lungs is generally regarded as a most untoward termination, but I have known cases do remarkably well after this event. It must be remembered that when pus forms, or when caseous degeneration is proceeding, there is a danger of tuberculosis being set up; and also that the lung may undergo destructive processes, phthisis being thus established.

TREATMENT.—The activity of the measures to be adopted in the management of pleurisy must differ greatly in different cases, and I strongly protest against any routine practice of removing blood, blistering, and administering mercury in this disease, which, whatever may be said to the contrary, is even at the present day by no means an uncommon mode of practice. The ultimate objects to be aimed at are not merely to save the patient's life, but to restore the parts affected to as normal a condition as possible.

The principles to be kept in view in the treatment of pleurisy are:—  
1. To subdue the inflammation, and diminish the amount of lymph and



fluid poured out. 2. To promote the absorption of these morbid products as rapidly as possible. 3. To remove them in some other way if they cannot be absorbed. 4. To relieve symptoms. 5. To support the strength of the patient.

1. The first thing which is attended to in the treatment of inflammation of synovial, as well as of most serous membranes, is to keep the structures affected in as complete a state of *rest* as can be obtained. It appears to me that this should also be the primary object to be kept in view in the management of pleurisy. For some years I have been in the habit of paying special attention to this matter, by mechanically fixing the side affected, and thus limiting or preventing its movements, and am firmly convinced of the beneficial effects resulting therefrom. The following is the method which I now adopt for the purpose of procuring the desired rest:—Strips of a properly-adherent plaster spread on some thick material, from three to four inches wide and of sufficient length, are applied round the affected side from mid-spine to mid-sternum or a little beyond. These are laid on over a variable extent of the chest, according to the requirements of the case, it being sometimes necessary to include the whole side. It is best to make the application from below upwards, and to fix the strips of plaster in an oblique direction rather than horizontally. The patient being directed to expire deeply, a strip is fixed at mid-spine and drawn tightly, firmly, and evenly round the side in the direction of the ribs, that is, a little obliquely from above downwards and forwards; then another strip is laid on across this, also extending from mid-spine to mid-sternum, but in the opposite direction to the first, that is, obliquely upwards and forwards across the course of the ribs; the third is to follow the direction of the first, overlapping about half its width, the fourth that of the second, and so on in alternate directions, until the entire side is included if required. Finally, it is often desirable to apply over the whole two or three strips horizontally, so as to form a superficial supporting layer; and one or two may also be passed from behind forwards over the shoulder, these being kept down by another strip fixed round the side across their ends. The good effects realized by this method of treatment, when efficiently carried out, have been as follows:—1. In cases of limited dry pleurisy, which are very common, especially in connection with phthisis, as well as exceedingly distressing, it gives almost invariably complete and immediate relief, so that patients can breathe and cough comfortably, and are able to follow their occupations without any difficulty, which is particularly important in the case of those who are obliged to work. 2. It is reasonable to suppose that the quantity of inflammatory products poured out will be limited by maintaining the parts in a state of rest. I have every reason to conclude that this result has been attained in several instances which have come under my notice. 3. The rest and pressure may also promote absorption, and I have found this mode of treatment decidedly efficacious in aiding towards the removal of moderate pleuritic effusion. 4. Occasionally cases of pleurisy come under observation, in which there is extensive exudation of lymph, with little or no fluid, and this remains as a chronic condition, causing palpable fremitus, attended with most unpleasant sensations to the patient. The only curative end that can be attained is to bring about adhesion of the surfaces of the pleura, and strapping the chest will most certainly effect this purpose.

Venesection or even local bleeding is, in my opinion, scarcely ever required in pleurisy. Calomel is a drug which had better be avoided,

except as an aperient. Many cases require nothing but rest, but if the attack is severe, the best therapeutic agents at the outset are those which lower the cardiac action, such as aconite, veratrum, or tartar-emetic in small doses, which may be given in some *saline* mixture. Opium is most valuable for the purpose of relieving pain and procuring sleep, Dover's powder being a very useful preparation; or morphia may be injected subcutaneously in severe cases. Tincture of bryony in small doses has been especially recommended in pleurisy, to relieve pain, and subdue cough. The application of cold to the chest has also been advocated, and may be useful in some cases.

2. Should there be much effusion—and this is not infrequently the condition found when the patient comes under observation—counter-irritation over the chest may prove decidedly beneficial for the purpose of promoting absorption. I have often found a small blister produce excellent results, and it may be repeated, if necessary. Applications of iodine are also employed, but in my experience these applications are not nearly so efficient. I have met with several instances in which strapping the side has certainly appeared to aid absorption when the fluid was not abundant. Medicines which act on the skin, bowels, or kidneys are those which are usually relied upon as the most efficient agents for promoting absorption in cases of pleuritic effusion. The administration of full doses of iodide of potassium, with infusion of digitalis and other *diuretics*, is sometimes beneficial. Powerful *purgatives* are of questionable value, and should be employed with caution, if at all, but the bowels should be kept freely opened. Repeated vapour or hot-air baths have proved serviceable in some instances under my care. Jaborandi and pilocarpine have also been employed, on account of their diaphoretic action. Dr. Anstie strongly recommended tincture of iron, and I have found this preparation in full doses a most valuable remedy in many cases of pleuritic effusion. It is well for the patient to avoid liquids as much as possible, and to adhere to a diet of dry food.

3. The removal of pleuritic effusion by *paracentesis thoracis*, which was formerly only adopted as a last resource, is now regarded almost universally, not only as a legitimate, but as a most valuable method of treatment in a considerable number of cases, for the purpose of bringing about a cure. At the same time this mode of treatment ought not to be followed as a routine measure. Unless there should be urgent symptoms calling for immediate removal of the fluid, no case of pleuritic effusion, either acute or even, as judged by the history, of a month or two's duration, should be thus treated until an endeavour has been made to procure absorption, especially if appropriate treatment has not been previously carried out. Several cases have come under my notice in which I fully anticipated that paracentesis would be required, but where the effusion rapidly disappeared under treatment.

Dr. Barlow and Mr. Parker have recommended the use of the hypodermic syringe for diagnostic purposes as a matter of routine in doubtful cases of chest-affections, and have found in their experience that after an exploratory puncture thus made in cases of serous effusion, and the removal of a very small quantity of fluid, absorption has rapidly followed, where medical treatment had already failed. I have also met with instances bearing out this statement.

The subject of operative interference in relation to pleuritic effusions has now become such an extensive one as regards details, that it is impossible to deal with it fully in this work, and only some of the more

general principles can be alluded to. Indeed, in many cases the treatment belongs to the domain of surgery.

It is difficult to lay down any strict rules as to the cases in which paracentesis is indicated, but the following seem to me to be the chief circumstances under which this method of treatment is called for:—

1. Whenever there is a large effusion accompanied with dangerous symptoms, namely, severe dyspnœa, and especially orthopnœa, a tendency to cyanosis, or symptoms indicating serious interference with the functions of the heart. In such cases delay is highly dangerous, as sudden death may occur at any moment, and the relief which follows the removal of the fluid is usually very marked.
2. In cases of considerable effusion, which, after a fair trial, does not yield to treatment, and shows no signs of becoming absorbed. It is not practicable to indicate the exact amount which calls for interference, so much depending upon the age of the patient, the condition of the chest-walls, the effects which the effusion seems to produce, and other circumstances. These are the cases which give most difficulty in forming a judgment as to the proper course to pursue.
3. In all cases of double pleurisy, when the total fluid may be said to occupy a space equal to half the united dimensions of the two pleural cavities (Anstie).
4. When the fluid is known or suspected to be purulent. In doubtful cases an exploratory puncture may be first made.
5. When a spontaneous opening has been formed towards the upper part of the chest. The existence of pyrexia, of some constitutional diathesis, such as tuberculosis, or of general debility, does not contra-indicate paracentesis in pleurisy, and the removal of the fluid often causes marked improvement in the general symptoms.

With regard to the method of operation, as a rule it is best to employ the *aspirateur*, but care must be exercised in its use, the indications for its withdrawal being severe pain and dyspnœa, violent cough, or the escape of blood. Barlow and Parker recommend in recent cases, where the effusion is serous, and small or moderate in amount, merely to use the hypodermic syringe, and thus remove a small quantity of the fluid. Dr. Southey has treated successfully some cases of pleuritic effusion by means of the small trochars and canulæ which he employs in the treatment of dropsy. In some exceptional cases it is necessary to use an ordinary trochar. Air should be excluded, and antiseptic precautions are desirable. The operation may need to be repeated, especially in cases of purulent effusion, in which recovery may take place after several evacuations of the fluid, which becomes gradually less and less. In other instances the pus becomes fœtid, or is so from the first, and then it may be desirable to make a free opening; to remove portions of the ribs; or to introduce a drainage-tube, two openings being made, one in front of the thorax, the second below and internal to the angle of the scapula (Barlow and Parker).

In cases of serous effusion, as has been already pointed out, it is unnecessary to remove the whole of the fluid; but if it is purulent, as much as possible should be taken away. Sometimes it happens that, owing to rigidity of the chest-walls, or binding down of the lung, the fluid cannot be expelled without assistance. Under these circumstances, it has been proposed by Bouchert to forcibly expand the lung through a tube passed into the bronchus; and by Parker to introduce filtered and carbolized air into the pleural cavities by means of a suitable apparatus, in order to displace the fluid. Occasionally it is desirable in cases of purulent pleurisy to use *stimulant* or *antiseptic* injections, such



as a solution of quinine (gr. v to  $\bar{3}$  i), weak carbolic solution, or diluted tincture of iodine (1 part to 4). The plan of washing out the pleural cavity is sometimes resorted to, when the pus is foetid, but Barlow and Parker point out that the double opening dispenses with the need of this painful and sometimes dangerous process in a large proportion of cases in children, and they affirm that if it should be required, equal advantages are derived by placing the patient daily in a warm bath, sufficiently high to cover the upper opening, Condyl's fluid, or a weak solution of carbolic acid previously prepared with boiling water being added to the bath.

The spot usually selected for making the opening in performing paracentesis thoracis is the 6th space, about the mid-axillary line; it may be conveniently made just below the angle of the scapula. In localized effusions the opening must correspond to the centre of maximum dullness, and more than one puncture may be needed when there are separate and distinct purulent accumulations.

4. The chief *symptom* likely to call for special attention in cases of pleurisy is pain in the side. If not relieved by the rest obtained by strapping it, the best plan is to employ a small subcutaneous injection of morphia. If the side is not strapped, the application of hot fomentations, linseed-meal poultices, or sinapisms may be serviceable. Urgent dyspnoea calls for paracentesis usually. Cough must be relieved, if troublesome, by *sedative* remedies.

5. Patients suffering from pleurisy need not be kept low as regards diet, but stimulants are not to be given at first. If the strength fails, and especially in the chronic forms of the disease, abundant nutritious food is required, with wine or beer. Quinine, iron, mineral acids, cod-liver oil, and other remedies of this kind are also very useful at this time.

6. The treatment of *secondary pleurisy* must be guided by the condition with which it is associated. Lowering measures are especially to be avoided in this class of cases.

## II. HYDROTHORAX—DROPSY OF THE PLEURA.

**ÆTIOLOGY.**—Hydrothorax is almost always a part of general dropsy from cardiac or renal disease. The effusion is said to be in rare instances of an active kind, associated with local cancer or tubercle.

**ANATOMICAL CHARACTERS.**—More or less clear serous fluid is found in both pleural sacs, compressing the lungs. No signs of inflammation are present.

**SYMPTOMS.**—Dyspnoea, with signs of deficient blood-aëration, are the only symptoms of hydrothorax, resulting from mechanical interference with the action of the lungs; and there is generally much distress, because this condition is added to some previous serious affection, and because both sides are involved. The *physical signs* are those of fluid in both pleuræ; not excessive in amount; freely movable; without friction-sound or fremitus; while there is no displacement of the heart.

**TREATMENT.**—As a rule this is merely a part of the general treatment for dropsy. Dry-cupping over the chest may be employed with advantage in some instances. In extreme cases paracentesis might perhaps be indicated, in order to afford temporary relief.

### III. HÆMOTHORAX—HÆMORRHAGE INTO THE PLEURA.

**ÆTIOLOGY.**—More or less blood may be mixed with pleuritic effusion of inflammatory origin; or with the ordinary serum, should there be a scorbutic or purpuric condition present. The accumulation of blood in any quantity in the pleura, however, is due to one of the following causes:—1. Rupture of a vessel from injury, or its perforation during operation. 2. Bursting of an aneurism, of which I have seen a most interesting example, in which an aneurism of the aorta between the pillars of the diaphragm ruptured into the left pleural cavity. 3. Carcinoma of the lung giving way into the pleura. 4. Diffuse pulmonary hæmorrhage extending to the surface of the lung. 5. Cancer of the pleura itself.

**SYMPTOMS.**—Dyspnœa is felt, owing to the pressure of the blood upon the lung; this being accompanied with evidences of loss of blood. Death may occur very speedily. The *physical signs* are merely those of pleuritic accumulation, either liquid or solid, according as the blood remains fluid or coagulates.

**TREATMENT.**—In most non-traumatic cases of hæmorthorax nothing can be done but to keep the patient at rest. Of course if the bleeding is due to injury, it is necessary to try to stop it by surgical means. Paracentesis may possibly be demanded.

### IV. PNEUMOTHORAX.—HYDRO- AND PYO-PNEUMOTHORAX.

**ÆTIOLOGY.**—1. Pneumothorax of any clinical importance results in the great majority of cases from *perforation of the lung*, owing to the rupture of a phthisical cavity. In very rare cases the lung gives way in the earlier stages of phthisis, or in connection with emphysema, abscess, gangrene, hydatids, or cancer; or the air-vesicles may rupture from violent cough, especially hooping-cough; or a collection of air or blood under the pulmonary pleura may perforate this membrane. 2. Perforation may take place *from the pleura into the lung*, in connection with empyæma or abscess of the chest-walls. 3. *Injury* may lead to pneumothorax, namely, direct perforation from without, laceration by fractured ribs, or severe contusion. 4. The *stomach* or *œsophagus* has in very rare instances ruptured into the pleura. It is unnecessary to consider those cases in which gas is present in the pleural cavity owing to the decomposition of fluid.

**ANATOMICAL CHARACTERS.**—The gas in the pleura generally consists of oxygen, carbonic anhydride, and nitrogen in variable proportions; with, under some circumstances, foetid constituents, such as sulphuretted hydrogen. It may fill the pleural sac completely, compressing the lung; or is sometimes limited by adhesions. The gas tends to excite inflammation, the resulting effusion being either serous or purulent, and the conditions being termed respectively *hydro-pneumothorax* and *pyo-pneumothorax*.

**SYMPTOMS.**—It is only necessary to consider here those symptoms which are indicative of *perforative pneumothorax*. Usually a sudden,

very intense pain in the side is experienced; as well as occasionally a sensation of something having given way, and of fluid pouring out; followed by urgent dyspnœa, and signs of shock. These symptoms in many instances immediately follow a violent cough. The dyspnœa may temporarily diminish, or it steadily increases in proportion to the amount of air accumulated, until constant or paroxysmal orthopnœa is established. The voice becomes feeble, in some cases to complete aphonia. Cough is often rendered difficult and ineffectual, and expectoration ceases. Occasionally there is much hyperæsthesia of the side. The pulse is frequent, weak, and small, but breathing being hurried out of proportion, the pulse-respiration ratio is altered. The patient generally presents an anxious and distressed aspect, and soon evidences of apnœa become apparent. A common mode of decumbency at first is dorsal, with the head raised, and the body inclined to the sound side; or the patient may assume a kneeling posture, supported on the elbows. In many cases the posture is changed frequently, and when fluid collects there is a tendency to lie on the affected side.

It must be borne in mind that even in severe cases of pneumothorax the symptoms may be by no means marked; and when the escape of air is limited by adhesions, they are usually comparatively slight.

**PHYSICAL SIGNS.**—The amount of air accumulated; the presence and quantity of fluid mixed with it; and the patency or closure, as well as the size of the perforation into the lung, will modify the *physical signs* of pneumothorax. 1. The side is *enlarged*, often to an extreme degree, the intercostal spaces being widened and effaced or even bulged out, so that the surface of the chest feels smooth. 2. *Movements* are deficient or annulled. 3. *Vocal fremitus* is weak or absent. 4. *Percussion* reveals at first increased resonance, the sound being often typically tympanitic, and this may be noticed considerably across the middle line. Sometimes it has an amphoric quality. If the amount of air becomes extreme there is dulness, with much resistance. When effusion of fluid takes place, dulness will be observed in dependent parts, movable with change of posture usually. Occasionally at the line of junction of fluid and air an amphoric note can be elicited, and a quivering sensation is felt by the fingers. 5. *Respiration-sounds* may be weak and distant, or almost suppressed; typically amphoric when the fistula is open, with a metallic echo; or alternately one or the other. A whistling inspiration is heard in rare instances, due to the passage of air through a narrow chink into the pleura. 6. *Vocal resonance* may be feeble or absent; or exaggerated, with a metallic or amphoric echo; while the whisper in some cases is very loud, and has a marked metallic or amphoric character. 7. *Cough* may also have a metallic echo. 8. *Metallic tinkling* is sometimes distinctly produced by breathing, coughing, or speaking; and the *bell-sound* may be elicited. 9. *Succussion* gives rise to a splashing sensation and sound, if both air and fluid are present in the pleural cavity. 10. *Displacement* of the mediastinum, heart, diaphragm, and abdominal organs is observed to a variable degree. 11. The *heart-sounds* are now and then intensified on the affected side, and attended with a metallic echo.

**PROGNOSIS.**—Though a very grave event, pneumothorax is not necessarily fatal, recovery occasionally taking place. It is much less dangerous when localized. In some instances where pneumothorax has occurred in connection with phthisis it seems to have delayed the progress of the lung-disease.



TREATMENT.—I have obtained great relief in some cases of pneumothorax from strapping the side firmly, as described under pleurisy. If the amount of air is considerable, causing urgent dyspnoea, it must be removed by some suitable apparatus, and pressure may be afterwards applied. Dry-cupping of the chest is sometimes useful. *Stimulants* and *antispasmodics* should be given to counteract shock and dyspnoea. Dr. Walshe recommends repeated inhalations of small quantities of chloroform. Pleurisy must be treated should it arise.

## CHAPTER XVIII.

### GENERAL DIAGNOSIS OF AFFECTIONS OF THE LUNGS AND PLEURÆ.

In this chapter it is intended to bring together the chief pulmonary diseases which resemble each other, and to point out their diagnostic marks. In many cases they can only be severally distinguished by a full consideration of the history of the patient; the local and general symptoms; and the physical signs present.

1. ACUTE PULMONARY AFFECTIONS.—The characteristic features of the principal diseases belonging to this group are indicated in the following table:—

DIAGNOSTIC TABLE OF ACUTE PULMONARY DISEASES.

	BRONCHITIS.	CROUPOUS PNEUMONIA.	CATARRHAL PNEUMONIA.	PLEURISY.	ACUTE PHTHISIS.
1. Mode of invasion.	Coryza and other symptoms of "cold." Not marked rigors, but only slight and repeated chills, if any.	A single, severe, prolonged rigor at the outset usually.	Generally occurs after bronchitis or pulmonary collapse, and without distinct rigors.	Several moderate rigors or slight chills, if any. Invasion may be very insidious.	Follows acute pneumonia, bronchitis, or catarrhal pneumonia; or begins with severe rigors, often repeated.
2. Sensations about the chest.	Soreness, heat, or rawness behind the sternum. Muscular pains from cough. Feeling of oppression.	Pain in the side frequently, not stitch-like, but more dull and diffused.	Pains about the chest often, but not specially localized.	Severe stitch-like pain in side.	Generally pains in various parts of the chest.
3. Cough.	In paroxysms, often severe.	Considerable, and in paroxysms.	Short, hacking, and painful.	Slight, and patient tries to repress it.	Frequent and violent fits.
4. Expectoration.	Abundant; changes its characters as the case progresses, from mucous to muco-purulent, &c.	Considerable: viscid, tenacious, and "rusty."	Often less than before; not "rusty."	Absent or very slight, and of no special characters.	Abundant; either bronchitic, or sometimes "rusty," or attended with hæmoptysis.

	BRONCHITIS.	CROUPOUS PNEUMONIA.	CATARRHAL PNEUMONIA.	PLEURISY.	ACUTE PHTHISIS.
5. Disturbance of breathing.	Sense of dyspnoea in proportion to the extent of the disease; may be extreme. Pulse-respiration ratio not proportionately altered.	Very rapid breathing, and much perversion of pulse-respiration ratio, but not proportionate feeling of dyspnoea.	Rapidity of breathing, increased when the complaint follows bronchitis; but feeling of dyspnoea may be less.	Quick shallow breathing at first, but less disturbance of pulse-respiration ratio than in pneumonia. Later on more or less actual dyspnoea.	Marked dyspnoea, and very hurried breathing, especially in the tubercular form.
6. Degree of pyrexia.	Often absent or slight, and temperature rarely above 100° to 102°. Skin moist.	Considerable; temperature usually high, 103°, 104°, 105°, or more, and runs a regular course. Skin acridly hot and dry.	Temperature high, but there are considerable remissions, at irregular intervals.	Not great, and no regularity in course of temperature. Skin not acridly hot.	Often very high, especially in the tubercular form, but no regularity in temperature.
7. Aspect of the patient, and general condition.	Tendency to cyanosis if the disease is extensive. In some cases adynamic symptoms set in.	Marked flushing of face, often unilateral. Not cyanotic. Usually great prostration.	Face is usually flushed. Often much anxiety and restlessness, with loss of flesh and strength.	Nothing special. No particular prostration, or tendency to cyanosis.	Severe prostration and weakness, with profuse perspiration, and rapid wasting. In the tubercular form extreme adynamia.
8. Physical signs.	Various dry rhonchi and mucous râles, with rhonchal fremitus. Signs of obstruction of bronchial tubes in some cases. More or less bilateral. Mucous râles chiefly towards bases; dry rhonchi at upper part of chest.	At first crepitant rhonchus: followed by signs of consolidation, namely, diminished movement; increased vocal fremitus; dullness; bronchial or tubular breathing; increased and metallic vocal resonance: finally, signs of resolution. Usually one base is affected. The side is not notably enlarged; nor is there any displacement of organs.	There may be signs of consolidation, in scattered spots, with râles. Both lungs are usually involved in irregularly scattered patches. When the disease follows extensive pulmonary collapse there may be a peculiar pyramidal form of dullness.	At first friction-sound or fremitus; succeeded by signs of fluid, namely, side often enlarged; movements interfered with; diminished vocal fremitus; dullness, occasionally movable; weak or suppressed breathing and vocal resonance; egophony sometimes; and displacement of organs; finally, signs of absorption, with reduced friction sound or fremitus. Usually on one side.	At first merely signs of bronchitis; followed by consolidation, softening, or excavations in different parts, especially towards the bases. In the tubercular form scattered râles frequently constitute the only physical signs.
9. Course and termination.	Variable. No crisis. Tendency to death by apnoea or adynamia in capillary bronchitis.	Often a marked crisis, and disease ends within a certain period.	No crisis, and course often prolonged.	No crisis, and course very variable.	Generally rapid course, and fatal termination.

It is impossible, in an arrangement like the preceding, to do more than indicate in a general way the main differences between the ordinary acute pulmonary diseases. It must be remembered that non-typical cases are met with; and also that these affections are often presented in various combinations. Usually the chief matters as regards diagnosis are to distinguish bronchitis from pneumonia, especially

catarrhal pneumonia complicating bronchitis; basic pneumonia from pleuritic effusion; acute phthisis from either form of pneumonia or from extensive bronchitis; and the different varieties of acute phthisis from each other.

The diagnosis of *pulmonary congestion* and its consequences, as well as of *abscess* and *gangrene* of the lung, have been sufficiently indicated in the descriptions of these morbid conditions. It is often difficult to diagnose between mere *lobular collapse* and *lobular pneumonia*, but the thermometer will afford important aid in distinguishing these conditions from each other.

2. Occasionally a case comes under observation where *one side is enlarged*, and there is a doubt as to whether the physical signs are due to *fluid*, or to very extensive *solid accumulation*, especially *cancer* of the lung. Under such circumstances the diagnosis must be founded on:—*a.* The history of the case. *b.* Certain physical signs, namely, in consolidation the chest is uneven on its surface; there is no fluctuation, but a marked sense of resistance is experienced on percussion: as a rule also bronchial breathing is heard, with increased vocal resonance and conduction of the heart sounds, though there may be complete absence of breath-sounds and voice, except perhaps in certain spots, such as close to the spine. *c.* The symptoms present, and the general condition. In consolidation pressure-symptoms are frequently noticed; there is more severe cough, with expectoration: and the sputa may have special characters, hæmoptysis being also not uncommon. Should there be any actual uncertainty as to the diagnosis, recourse must be had to the employment of the hypodermic syringe, aspirateur, or a small exploratory suction-trochar, by means of which some of the fluid, if present, may be removed for examination, and no damage is done if there is none.

3. There are certain conditions in which signs of *excess of air* within the chest are observed, namely, *emphysema*; *hypertrophy of the lungs*; and *pneumothorax*. There may be some difficulty in separating the two former, and they are often more or less associated. *Hypertrophy* is generally unilateral, following some affection which evidently interferes with the action of the opposite lung; while the breath-sounds are simply exaggerated; and there are no symptoms. *Emphysema* is usually bilateral; expiration and the accompanying sound are much prolonged; dry rhonchi are often heard; and there may be characteristic dyspnoea. The mode and conditions of onset; severity and nature of the symptoms; almost invariably unilateral character; great enlargement of the side, with typical tympanitic percussion-sound, amphoric breathing, and other marked physical signs, render the diagnosis of *pneumothorax* from the other affections mentioned perfectly easy as a rule.

4. Perhaps as difficult a matter as any in the diagnosis of lung-affections is to distinguish between certain morbid conditions which are attended with *retraction of one side*, namely, *chronic interstitial pneumonia*; retraction after *pleurisy*; certain cases of ordinary *phthisis*; *collapse of the lung*; and *infiltrated cancer*. It will only be practicable here to indicate the main points to be taken into consideration, which are:—*a.* The previous and family history of the patient in all its details; and the duration of the illness. *b.* The nature of the local symptoms, special attention being paid to the presence and character of pain; and to the nature of the sputa, which should be carefully ex-



amined, the occurrence of hæmoptysis, and the characters of any blood expectorated, being points of much importance. *c.* The constitutional and general condition, as indicating tuberculosis or cancer, emaciation, debility, or pyrexia. *d.* The presence of signs of tubercle or cancer in other parts. *e.* The physical signs noted, including their characters; their seat, as to the part of the lungs affected, and whether one or both are involved; and their extent. *Chronic pneumonia, cancer, and phthisis* are often attended with signs of cavities, these being in the last affection usually most marked at the apex, but not so in the others. In cancer dulness frequently extends across the middle line. It is important to examine thoroughly with the view of ascertaining whether a tumour is present in the chest, which might, by pressing upon a bronchus, lead to pulmonary collapse; and also to look for other signs of pressure, which are generally associated with cancer. *f.* The progress and duration of the case, which will usually help considerably when there is any obscurity.

5. Sometimes there is a difficulty in distinguishing between *chronic bronchitis* and *phthisis*, when the former is attended with profuse purulent expectoration and with general wasting. The slow progress and comparatively slight degree of emaciation; absence of fever; non-occurrence of hæmoptysis; and absence of physical signs of consolidation followed by cavities, will serve to characterize mere bronchitis in the majority of cases, but it must be borne in mind that this complaint frequently terminates in phthisis. For the diagnosis of the different forms of phthisis from each other, which is often difficult, reference must be made to what has been stated when discussing its varieties.

6. It may be necessary to determine the *nature of any fluid in the pleura*, and the *cause of its presence*; and here it may be mentioned that fluid in rare instances finds its way into this cavity from the abdomen, as from the bursting of an abscess of the liver or kidney through the diaphragm, or of a hydatid-cyst. There will then have been previous symptoms indicative of these several conditions. With regard to the determination of the nature of the fluid in cases of *pleuritic effusion after inflammation*, it is in doubtful cases impossible to come to any positive conclusion without making use either of the hypodermic syringe, aspirateur, or exploratory trochar, and obtaining some of it for examination. Allusion has already been made to the inadequacy of Bacelli's sign. Barlow and Parker found that in children the signs usually regarded as being distinctive between serous and purulent effusion were quite unreliable; but they regard a peculiar anæmia, with an earthy complexion, and especially clubbing of the finger-ends, as suggestive of empyæma. In children, however, if the fluid has remained in the pleura for some weeks, it will probably be of a purulent character. Mere *hydrothorax* is distinguished from *inflammatory effusion* by the following characters:—*a.* It is usually a part of general dropsy. *b.* Fluid is found on both sides, but not in excessive quantity; it accumulates in the lower part of the pleuræ, pushing down the diaphragm, but not displacing the mediastinum and heart as a rule; and it is freely movable. *c.* There are no friction-phenomena. *d.* Pain and tenderness are absent; but dyspnoea is generally very severe. *e.* There is no pyrexia. *Hæmothorax* is characterized by the circumstances under which it occurs; and the signs of loss of blood. If there is any uncertainty in the diagnosis, the aspirateur should be employed

7. It must be mentioned that symptoms and physical signs may be observed in connection with the lungs, which are due to certain morbid conditions not originally associated with these organs, such as rupture of a hydatid tumour of the liver or of an hepatic abscess into the pleura or lung; a hernia of the stomach through the diaphragm; and other exceptional lesions.

---

## CHAPTER XIX.

### DISEASES OF THE CIRCULATORY ORGANS.

#### I. CLINICAL PHENOMENA CONNECTED WITH THE HEART.

THE evidences of disease in connection with the central organ for the circulation of the blood are necessarily not confined to this part alone, but must be more or less apparent throughout the entire system. It is essential to notice, however, at the outset, that most grave organic cardiac mischief may be unattended with any evident symptoms; and that, on the other hand, apparently serious disturbance of the heart may be observed, which is entirely of a functional character. Moreover, other diseases are often associated with cardiac affections, especially renal and pulmonary, which may greatly modify the symptoms; while these affections tend to set up secondary structural changes in other organs.

1. Various **subjective sensations** are often experienced about the cardiac region, namely, pain, either constant or of a paroxysmal and anginal character, oppression, dragging, sinking, or unpleasant sensations associated with the movements of the heart, such as palpitation, irregularity, jogging, rolling, falling back, jumping into the throat, intermittency, or complete stoppage. These are sometimes attended with extreme distress and dread of death. There may be local tenderness; or, on the other hand, relief may be afforded by pressure.

2. The **action of the heart** is frequently disturbed, being feeble almost to extinction, excited, palpitating, irregular or intermittent, or embarrassed.

3. The most common and important symptoms associated with cardiac diseases are those due to some **derangement of the circulation of the blood**. These require careful consideration in each individual case, as several of the phenomena may result from different causes affecting the circulation. Without entering into details, it must suffice to state that this class of symptoms may arise from:—(1) *Abnormal action of the heart*, which is either *excessive*, *deficient*, or in some way *disordered* and *ineffective*. (2) *Physical or mechanical derangement of the circulation*. This derangement is usually associated with some difficulty in connection with the valves or orifices; but under this head may also be mentioned the presence of an abnormal communication between certain cavities of the heart; the formation of thrombi or clots in these cavities; and the blocking of vessels by thrombosis or embolism, consequent upon cardiac disease. Either the *pulmonary* or the *systemic* circulation, or

both may be thus affected, and it will be necessary to discuss briefly the phenomena which may arise accordingly.

With regard to the *pulmonary circulation*, owing to the absence of vasomotor nerves, and of capillary resistance, mere mechanical influences play a much more important part than they do in relation to the general circulation (Balfour). Should the right ventricle be acting unduly, active pulmonary congestion would be induced. Any disorder of the cardiac action readily disturbs the circulation through the lungs, and thus symptoms connected with these organs may be suddenly or speedily brought on. Mechanical difficulty affecting this circulation is of very common occurrence, either due to impaired action on the right side of the heart, or to some impediment on the left side, especially in connection with the mitral orifice. Portions of clot may be detached within the right cavities, and conveyed into the pulmonary artery, either blocking its trunk or one of its branches.

In consequence of these several conditions there will be more or less imperfect oxygenation of the blood; and the other lesions which may be induced are bronchial catarrh, pulmonary congestion or œdema, infarction or hæmorrhage, pneumonia, gangrene, or pneumothorax. Long-continued congestion will lead to thickening, or atheromatous or calcareous degeneration of the pulmonary vessels; to proliferation of cellular tissue in the lungs, with excessive formation of pigment; or to emphysema.

Symptoms associated with the respiratory organs are of common occurrence in heart diseases, especially disorders of breathing, but there may be also cough, expectoration of different kinds, and hæmoptysis. Allusion must be made to a peculiar form of *cardiac dyspnœa*, or so-called *cardiac asthma*. The difficulty of breathing resembles that of exertion, being more or less hurried, panting or gasping, and noisy. It is subject to much variation, being liable to come on in very severe paroxysms, breathing being quite free and undisturbed in the intervals, the fits occurring particularly after any effort, especially after ascending heights, or when the patient lies down or falls asleep. The act of respiration is not interfered with, hence it is not very frequent, nor is expiration prolonged as in emphysema, while the respiratory movements and sounds are quite free. Of course, if the lungs are involved the characters of the breathing will be modified accordingly, and true bronchial asthma may be observed; while other causes often affect the respiration, such as anæmia, and various complications. A peculiar disturbance of respiration has been observed in fatty disease of the heart—*Cheyne-Stokes respiration*, in which at intervals the act becomes gradually hurried and deeper up to a certain point, and then subsides in the same gradual manner, until at last there is a momentary cessation of breathing, with a dead silence, lasting from a quarter to three-quarters of a minute. Involuntary sighing is occasionally noticed in cardiac diseases, or a frequent tendency to draw in a full breath.

As the result of disturbance of the *general circulation*, numerous symptoms are met with in cases of cardiac disease, affecting the system generally, as well as the more important organs. The blood may be driven into the arteries with undue force; or a deficient supply enters these vessels; or an excessive flow into the arteries occurs, immediately followed by a sudden or rapid emptying in various degrees, the blood running back into the left ventricle. The general venous circulation is frequently impeded more or less, and as a consequence the various



tissues and organs of the body become mechanically congested, and the results of this stagnation follow, namely, serous effusion; permanent enlargement of capillaries and small veins; increase of connective tissue, with thickening and contraction; thrombosis; or rupture of vessels, with consequent hæmorrhage. It is necessary to consider in some detail the symptoms which may follow the different disorders of the general circulation just indicated:—*a.* Patients suffering from heart disease are not uncommonly anæmic. As the result of general venous plethora, they often present a more or less cyanotic appearance, especially about the lips, fingers, and toes; with pallor, from deficient supply of arterial blood. In time the face becomes puffy and bloated, and the finger-ends clubbed. The patient feels chilly; and is deficient in vitality and vigour, being disinclined for any exertion, languid, apathetic, and easily fatigued. Coldness and clamminess of the limbs, especially of the hands and feet, are frequent symptoms. Sooner or later *dropsy* sets in, usually beginning in the feet and ankles and extending upwards, ending in general anasarca, with serous effusions. As a rule it is gradual in its onset and progress, and often subsides temporarily under appropriate treatment; in some instances, however, it is rather rapid in its appearance, and then relief may follow as regards chest-symptoms. If general cardiac dropsy comes on rapidly it is usually more easily got rid of, and less likely to return soon, than when it is gradual in its progress. In rare instances ascites is the earliest form of dropsy observed in heart diseases. In connection with the venous congestion and dropsy, cutaneous lesions are liable to be set up in the legs, namely, erythema, erysipelas, eczema, rupture of the skin, sloughing, or chronic ulceration. *b.* Some very striking symptoms result from disturbance of the circulation in the central nervous system. Among these the chief are dull, heavy headache; a sense of fulness or heat about the head, or rushing of blood, with flushing of the face; throbbing sensations; giddiness and unsteadiness; sleepiness—sleep, however, being disturbed by startings and most unpleasant dreams; mental obscuration, with irritability, want of resolution and stability, indisposition to mental effort, and impairment of the intellectual powers generally; disturbances of vision and hearing, such as flashes and specks before the eyes, noises in the ears, objective changes in connection with the eyes being also ultimately established; and curious sensations or twitchings in the extremities. Attacks of faintness or actual syncope are of common occurrence; or they may assume an apoplectic or epileptiform character. True apoplexy may supervene, due to cerebral hæmorrhage or embolism. Cases of cardiac disease causing general venous congestion may terminate by gradual sopor ending in complete coma. Epistaxis results in some cases from congestion originating in the heart. *c.* The digestive and assimilative organs frequently suffer in cases of cardiac disease. The tongue becomes full, large, congested, and marked with the teeth; the mouth and throat often at the same time presenting venous congestion. The stomach becomes the seat of congestion and catarrh, with increased secretion of mucus, leading to dyspeptic symptoms, such as a sensation of fulness in the epigastrium, flatulence, eructations, and deficient or depraved appetite. Flatulence may be a very prominent symptom. From intestinal congestion results constipation or diarrhœa, or an alternation of these symptoms; and in course of time hæmorrhoids may be originated. The liver is at first congested and enlarged, and a certain amount of jaundice is often evident, this being partly due to congestion

of the mucous membrane lining the bile-ducts. The bile also is liable to be unhealthy, owing to an admixture of mucus from the gall-bladder, and this increases the difficulty of digestion. When jaundice is associated with cyanosis a greenish tint of the skin is produced. Ultimately the liver may become the seat of a form of cirrhosis. After a time the spleen tends to become permanently enlarged. *d.* Undoubtedly the kidneys may be involved, becoming venously congested and probably finally cirrhotic; hence at first the urine is deficient in quantity, dark, concentrated, and of high specific gravity, deposits urates, and contains more or less albumen, as well as casts in some cases. There may be pain and tenderness over the renal region. Catarrh of the bladder occasionally occurs. *e.* From congestion of the genital organs symptoms commonly arise in females, namely, menorrhagia, metrorrhagia, leucorrhoea, and possibly metritis. In males there may be a diminution in sexual power and inclination; while prostatic enlargement and hydrocele have been supposed to be occasionally due to cardiac affections.

6. Very dangerous symptoms may arise in connection with disease of the heart, from the **formation of clots and other matters** in its cavities. As already mentioned, portions of clots are liable to be conveyed into the circulation as *emboli*, inducing local symptoms associated with obstructed arteries; but occasionally septic products are produced within the cardiac cavities, which become mixed with the blood, contaminating it, and leading to pyrexia or more serious conditions.

7. In exceptional instances certain conditions of the heart or pericardium originate symptoms by causing **pressure on neighbouring structures**.

8. Cardiac affections will necessarily influence materially the state of the **pulse**, from which most important information may be gained. In all cases, therefore, the pulse ought to be thoroughly investigated in all the particulars to be presently described; and it is requisite also to examine carefully in order to determine whether the arteries are in a condition of degeneration or not.

9. In rare instances **rupture of the heart** occurs, with consequent escape of blood, necessarily leading to grave symptoms.

## II. CLINICAL PHENOMENA CONNECTED WITH THE ARTERIES.

1. Occasionally there may be pain, throbbing, tension, or other **subjective sensations** associated directly with some diseased condition of an artery; and tenderness is not uncommon.

2. **Pressure on neighbouring structures** gives rise to an important class of symptoms in connection with aneurismal dilatation of arteries. Only aneurisms within the chest or abdomen, however, come specially under the notice of the physician. At present the symptoms due to pressure within the chest will alone be considered. They may result from the pressure of any mediastinal tumour, and therefore the description here given will apply to all forms of mediastinal enlargement, it being borne in mind that the exact symptoms must necessarily depend upon the situation, shape, size, direction and rate of growth, and other characters of the tumour; that it is rare for the whole of those mentioned to be observed in the same case; and that they are liable to change, owing to an alteration in the direction of growth, or other causes.

The modes in which pressure contributes to the production of symptoms may be summed up generally as follows:—*a.* By causing displacement, as of the heart, trachea, or large vessels; and altering the relation of orifices. *b.* By pressing upon hollow tubes or organs, and obstructing them to a greater or less degree, for example, the air-tubes, œsophagus, great vessels, thoracic duct, heart. *c.* By compressing the substance of organs, and thus preventing them from performing their functions, for instance, the lungs. *d.* By leading to actual destruction of tissues, as of the chest-walls, spinal cord, walls of hollow tubes, pericardium or heart, lungs, nerves. *e.* By irritating or paralyzing nerves, symptoms being often thus set up at a distance from the seat of mischief. *f.* By exciting local inflammation, ending in exudation, adhesions, or suppuration.

Such being the general effects of pressure, the special symptoms may be considered according as the pressure tends in an outward or *centrifugal*, or an inward or *centripetal* direction.

(i.) *Centrifugal symptoms.*—In addition to obvious physical signs, pressure on the parietes of the thorax will excite pain, either neuralgic; or due to inflammation of various structures; or to destruction of bone, when it tends to be heavy, grinding, or gnawing in character. When neuralgic, the pain often shoots in various directions, as up along the neck or down the arm. There may be merely a sense of weight and oppression, or heat; or indefinable feelings may be complained of. Tenderness is frequently observed, and sometimes extreme hyperæsthesia. Actual paralysis of nerves may ultimately be caused. If the vertebral column is eaten through, symptoms associated with the spinal cord are set up, first indicative of irritation, and subsequently of destruction.

(ii.) *Centripetal symptoms.*—*a.* Pressure on the right side of the heart or pulmonary artery will interfere with the supply of blood to the lungs, and thus aid in causing dyspnoea, while it leads to general overloading of the venous system. The action of the heart is very liable to be disturbed when this organ is pressed upon. *b.* Obstruction of the main arteries—innominate, carotid, or subclavian—will alter the characters of the corresponding carotid or radial pulse, diminishing its fulness and force, delaying it, or even obliterating it. *c.* Most important symptoms result from pressure on the large systemic veins, usually the superior vena cava, either innominate, or the vena azygos major. Very rarely is the inferior cava interfered with. Venous congestion, œdema, enlargement of capillaries and veins, the formation of coagula, or actual rupture of vessels may follow, the nature and extent of the symptoms necessarily depending upon the vein which is obstructed, being usually confined to the head, face, neck, chest, and arms, and either bilateral or unilateral. The face, especially about the lips, is often puffed and livid, presenting distended capillaries. The neck may be full, thickened, and tumid-looking, having a peculiar spongy or elastic feel, somewhat resembling that of erectile tissue. The throat is often congested, and forms abundant secretion. More or less severe cerebral symptoms may result from venous congestion of the brain, and deafness is sometimes complained of. If the vena azygos is pressed upon, there are signs of spinal congestion, namely, sensory and motor disturbances in the lower part of the body. Should the inferior cava be interfered with, there will be anasarca of the legs and abdominal walls, accompanied with ascites and other signs of obstruction involving the abdominal circulation. *d.* Rarely the pulmonary veins are compressed, causing



pulmonary congestion and its consequences. *e.* The various morbid conditions set up in connection with the main air-tubes or lungs will cause more or less severe dyspnœa, cough, hæmoptysis, alterations in voice, and other symptoms. Frequently marked laryngeal or tracheal symptoms are present, either due to direct pressure upon the air-tube; to chronic laryngitis and ulceration, which may be the result of mere irritation of the nerves; or to functional nervous disturbance. When hæmoptysis occurs the blood sometimes resembles "currant jelly." *f.* From œsophageal obstruction dysphagia may result; and if food cannot be taken, emaciation necessarily follows. Rarely hæmatemesis takes place. *g.* Extreme emaciation is said to be the consequence of obstruction of the thoracic duct. *h.* Pressure on nerves originates numerous clinical phenomena, some of which have been already noticed. Interference with the vagus nerves or pulmonary plexuses disturbs breathing and cardiac action. The recurrent nerves, especially the left, are peculiarly liable to be pressed upon, severe laryngeal symptoms and dysphagia being thus excited. Pressure on the phrenic nerve will affect the action of the diaphragm. Diminution in the size of the pupil of the eye, or, more rarely, dilatation, depends upon more or less disturbance of the sympathetic; this also may affect the temperature and nutrition of one side of the head and face. Some of the nerves forming the brachial plexus are in exceptional instances so pressed upon as to lead to various disorders of sensation, especially pain, or even to paralysis of the arm; and pressure on the intercostal nerves may cause pain in, or paralysis of the corresponding muscles.

I may be allowed to digress here to draw attention to the absolute necessity of an intelligent knowledge of the medical anatomy of the thoracic contents, as well as of the functions of the various structures, before the symptoms due to pressure can be at all comprehended.

**3. Obstruction of an artery** will be followed by symptoms dependent upon the want of a proper supply of arterial blood in the part to which the obstructed vessel normally conveys it. These will vary not only with the organ or part which is thus deprived of blood, but also according to the degree and rapidity of obstruction. If sudden and complete, it will lead to immediate abolition of functions, and thus may induce serious symptoms, as in the case of the brain, in connection with which sudden loss of consciousness and hemiplegia may follow obstruction of an artery; or when the main artery of a limb is blocked up, which is followed by local paralysis. If the obstruction is more gradual it causes anæmia, diminution of temperature, depression of functions, and deficient nutrition, which may end in softening or actual gangrene. The pulse also is more or less weakened to complete extinction in the arteries which receive their blood from that which is obstructed; while in the portion of the obstructed artery nearer the heart there is increased pulsation.

**4.** Diseased conditions of arteries may originate **emboli or substances which contaminate the blood**, thus giving rise to symptoms of obstruction in distant parts, or to general symptoms indicating septicæmia.

**5.** Serious phenomena, both local and general, will necessarily attend the **rupture of an artery**, if it is of any size.

**6. The Pulse.**—To *feel the pulse* has always been justly looked upon as one of the first duties of a medical practitioner. This gives invaluable information in general diseases, and in various affections connected

with other organs which influence the heart and vessels, as well as with regard to special morbid conditions of these structures. The subject will be considered in detail under **PHYSICAL EXAMINATION**.

### III. CLINICAL PHENOMENA CONNECTED WITH THE VEINS.

1. There may be **pain, tenderness, or cutaneous redness** in the course of veins. 2. When veins are **obstructed** in any way, the local formation of a clot being the most frequent cause, there will be the signs of venous congestion already described, varying in extent and situation according to the vessel or vessels involved. 3. **Emboli** may originate from clots in veins, and be conveyed to various parts of the body. Septic matters may also be formed.

### IV. PHYSICAL EXAMINATION OF THE CIRCULATORY ORGANS.

The chief modes of physical examination available in the investigation of the circulatory system are similar to those already described in connection with the lungs. In addition, certain special methods are employed. For observing accurately the difference in time between the movements of different parts of the heart the plan is adopted of fixing bristles over corresponding parts of the chest, by means of pellets of bees' wax, each bristle carrying a small paper flag. Special instruments are also in use for graphically recording movements, namely, the **cardiograph**, to record those of the heart, and the **sphygmograph**, those of the pulse; Dr. Gowers has invented a combined **cardio-sphygmograph**. Another apparatus, named a **sphygmomanometer**, has been introduced by Professor S. von Basch, for the purpose of estimating the blood-pressure in the radial artery.

The nature of the information afforded by the different modes of examination in connection with the heart and vessels may be thus summarized. **Inspection** reveals:—1. Any alteration in the shape and size of the chest over the cardiac region; or any bulging corresponding to an aneurism. 2. Certain points about the impulse of the heart. 3. The amount of visible pulsation in the great arteries of the neck; the existence of abnormal pulsation; and certain conditions of the arteries of the limbs. 4. The state of the superficial veins, as well as of the large veins in the neck, especially the right external jugular. **Palpation** indicates:—1. Any local change in size and shape. 2. The precise characters of the cardiac impulse. 3. The presence of any cardiac thrill or pericardial friction-fremitus. 4. The condition of the large arteries of the neck; the characters of any abnormal pulsation, whether visible or not; and the state of the arteries of the limbs. 5. Certain signs connected with the veins of the neck. **Mensuration** merely gives more accurate information with regard to form and size. **Percussion** discloses:—1. Any alteration affecting the cardiac dulness; and the amount of resistance felt over this region. 2. Abnormal dulness due to an aneurism. **Auscultation** is mainly useful for investigating certain sounds, namely:—*a.* **Sounds connected with the heart.** (i) The ordinary *cardiac sounds*. (ii.) Abnormal sounds originating within the heart, named *endocardial murmurs*, usually depending upon some morbid condition in connection with the orifices and valves. (iii.)

*Pericardial murmurs* or *friction-sounds*, due to roughness of the surfaces of the pericardium. *b. Arterial sounds and murmurs*, especially in the large arteries of the chest and neck, but which may also be observed in the smaller arteries. *c. Venous murmurs*. It may be mentioned that the stethoscope may also prove very serviceable in realizing certain characters of the cardiac impulse or of an aneurismal pulsation, through the sensations thus conveyed to the head. Vocal fremitus and resonance may also be made use of with advantage in some cases, as aids in determining the limits of the heart.

I proceed now to consider specially the PHYSICAL EXAMINATION of the several parts of the circulatory system.

### A. EXAMINATION OF THE HEART.

Before discussing the several points relating to the physical examination of the heart, attention may be called to the fact that in rare instances the viscera are transposed, and then the signs associated with the heart will be transferred from the left to the right side of the chest.

#### 1. CHANGES IN THE FORM AND SIZE OF THE CARDIAC REGION.

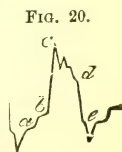
**1. Bulging.** This varies much in degree, but may extend from the second to the seventh or eighth rib, while the sternum may also be partly involved. The intercostal spaces are either normal or unduly prominent. Measurement shows that there is a greater distance from the nipple to mid-sternum on the left than on the right side. Bulging is most liable to occur in young persons. *Causes.* (i.) Enlargement of the heart, especially hypertrophy. (ii.) Pericardial effusion.

**2. Depression.** There may be a general falling-in over the cardiac region; or the spaces are sometimes chiefly affected. *Cause.* Pericardial agglutination, with adhesion of its outer surface to the chest-wall.

### II. CARDIAC MOVEMENTS.

The cardiac movements are usually investigated by *inspection* and *palpation*, assisted in some cases by the use of the *stethoscope*. The instruments already mentioned may be required to give accurate information, and it will be convenient to offer here a few remarks regarding the **cardiograph**. This consists of an apparatus which registers graphically the movements of the different parts of the heart, either upon a sphygmograph-plate, or on a revolving cylinder. The description of the different instruments employed will be found in physiological works. The tracing is named a *cardiogram*.

**DESCRIPTION OF CARDIOGRAM.**—The accompanying diagram (Fig. 20) represents a normal cardiogram. In the line of ascent two waves are seen; that opposite *a* represents the commencement of the ventricular diastole; that at *b* is due to the auricular systole. From *b* to *d* corresponds to the ventricular impulse; the wave *c* indicating the closure of the auriculo-ventricular valves, but it is not always at the extreme summit. In the line of descent *e* corresponds to the closure of the sigmoid valves. The waves between *c* and *d* have been attributed to oscillations originated by the closure of the auriculo-





ventricular valves; but some observers suppose that they are produced by the cardiograph.

A *cardiographic tracing* gives very accurate indications of the absolute and relative duration of the different parts of a cardiac revolution; and also affords information with respect to the force of the ventricular contraction, and as to variations in the force and frequency of the heart's action. Clinically the cardiograph has been found useful in cases of aortic regurgitation, mitral regurgitation or obstruction, adherent pericardium, and reduplication of the heart-sounds. Apart from the heart, the instrument has also been employed to record the movements of pulsating tumours and aneurisms.

**IMPULSE IN HEALTH.**—In health the impulse practically corresponds with the apex-beat, which is usually felt in the fifth left interspace, about  $1\frac{1}{2}$  inch below and  $\frac{3}{4}$  inch inside the nipple, or about 2 inches from the left margin of the sternum, over an area of about a square inch. It may be a space higher or lower, according to the shape of the chest; and also tends to be raised in children, and depressed in old people. It is single, and systolic in time; slightly heaving and gliding down towards the left; gradual and not abrupt in its development.

**IMPULSE IN DISEASE.**—When examining the cardiac impulse the chief points to be noticed are:—1. Its exact position, and whether this is constant or varies with different beats of the heart. 2. Its area as seen and felt; and if it is well-defined or not. 3. Its force. 4. The characters it presents to the sight or touch. 5. Its rhythm. 6. The effects of change of posture upon it.

**1. Position.** The impulse may be displaced by conditions external to the heart; morbid changes in the pericardium; alterations in the size of the heart itself; or a combination of these causes. (i.) *Elevation.* The apex-beat is often raised to the fourth space or higher. *Causes.* *a.* Pushing up of the heart by some abdominal accumulation, such as ascites or an enlarged liver. *b.* Upward traction, owing to contraction of the lung from phthisis involving the left apex, or sometimes the right, with the formation of adhesions. *c.* Pericardial effusion, or adhesions after pericarditis. *d.* Diminution in the size of the heart, from atrophy or great loss of blood. (ii.) *Depression.* The impulse is often lowered, and may reach as low as the seventh or eighth rib. *Causes.* *a.* Cardiac enlargements, especially hypertrophy, either general or affecting the left side. *b.* An aneurism or other tumour above the heart, pushing it down. *c.* Pericardial effusion in some cases. *d.* Weakness of the great vessels, owing to some acute or long continued illness, allowing the heart to sink down. (iii.) *Lateral displacement,* either to the right or left, is very common, being often combined with elevation or depression. *Causes.* *a.* Pushing aside of the heart by a collection of fluid or gas in either pleural cavity, especially the left; by an enlarged lung, due to emphysema, hypertrophy, or cancer; or by an aneurism or other tumour. After such displacement the heart may remain permanently adherent in its new position. *b.* Cardiac enlargements. According to the nature of the enlargement, and the part of the heart affected, the impulse will be carried more to one side or the other. As a rule, it may be stated that hypertrophy tends to displace the impulse towards the left; dilatation towards the right. *c.* Pericardial effusion, which always carries the apex-beat to the left. *d.* Contraction of the right lung in cases of phthisis, which may draw the heart more or less towards the right.

(iv.) Occasionally the impulse *alters its position* with each beat of the heart, when this organ is greatly dilated.

**2. Area, and degree of definition.** (i.) The area of the cardiac impulse is often *increased* to a variable extent, being either well-defined or the reverse. *Causes.* *a.* Cardiac enlargements, especially if associated with pericardial agglutination. *b.* Excited action of the heart. *c.* Undue contact of the heart with the chest-walls, either from retraction of the left lung; adhesion between the pericardium and costal pleura; pressure on the heart from behind by an enlarged liver or spleen, or a tumour; or falling-in of the chest-walls. *d.* Pericardial effusion, in which the impulse appears to be very extensive and ill-defined. (ii.) *Diminished* area is observed in most of the conditions which weaken the impulse, but it is not of much practical importance.

**3. Force.** This may be :—(i.) *Increased.* *Causes.* *a.* Hypertrophy of the heart. *b.* Undue contact with the chest-walls. *c.* Excited action. (ii.) *Diminished*, sometimes to complete extinction. *Causes.* *a.* Functional weak action of the heart from any cause. *b.* Certain cardiac diseases, such as dilatation, fatty degeneration or infiltration, and atrophy. *c.* Fluid or air in the pericardial sac; or, in exceptional cases, pericardial adhesions, the movement of the apex being completely interfered with. *d.* Distension of the lungs, especially the left, from emphysema or hypertrophy, in consequence of which they come between the heart and the chest-walls.

**4. Characters.** The impulse often presents unusual characters, the following being the most important:—(i.) *Undulatory* or *wave-like*. This may be only visible, or felt as well. *Causes.* *a.* Pericardial effusion. *b.* Dilatation of the heart, with thin, weak, and degenerate walls. *c.* Uncovering of the heart, with adhesion of the pericardium to the chest-walls. (ii.) *Heaving* or *pushing*. It is for the purpose of observing this character that the stethoscope is useful, through which the movement becomes often very obvious, both to the auscultator and to bystanders. A distinctly heaving impulse is characteristic of cardiac hypertrophy. (iii.) In dilatation the impulse is often *quick, sharp, and slapping*. (iv.) When the heart is very feeble the action may be *jerking* or *fluttering*. (v.) If pericardial agglutination exists along with hypertrophy or dilatation, and with or without valvular disease, the impulse frequently acquires very peculiar characters, differing in different cases, and it may be impossible to say what parts of the movement are systolic, and what diastolic. In some instances a recession or indrawing is observed at the apex after the impulse; and rarely, in cases of extensive agglutination, a curious systolic recession of the epigastrium is noticed.

**5. Rhythm.** (i.) *Irregularity* is often observed, both as regards force and time; or the beat may be *intermittent*. *Causes.* *a.* Functional disturbance of the heart's action. *b.* Cardiac diseases, namely, marked dilatation, fatty disease, and some cases of mitral or aortic disease. *c.* Malformations of the heart. *d.* Occasionally pericardial effusion or adhesions. (ii.) In pericardial effusion the impulse sometimes seems to *lag behind the ventricular systole*, as if it took some time to be conveyed to the surface. (iii.) The systolic impulse may appear to be *double* or even *treble*; or a *diastolic* impulse may likewise be present. This is observed in some cases of dilatation and hypertrophy with adhesions.

**6. Effects of change of posture.**—(i.) *Increased mobility* of the apex-beat has been considered a sign of pericardial effusion, but it is not of

much importance. (ii.) The fact that the impulse *does not alter in different postures* is sometimes of much aid in determining the existence of adhesions—pericardial and pleuritic.

**BASIC AND EPIGASTRIC IMPULSE.**—It is necessary to allude briefly to the impulse not uncommonly seen or felt towards the base of the heart; and to that in the epigastrium. *Basic impulse* is chiefly observed in cases where a cavity in the apex of the left lung has contracted, drawing up the heart and bringing it into close contact with the chest-walls, adhesions probably forming; but it may be due to hypertrophy about the base, affecting the auricle, or to aneurism of the heart. Of course a pulsation in the neighbourhood of the base of the heart may be associated with the aorta or pulmonary artery. *Epigastric impulse* is generally cardiac in origin; sometimes it is associated with the aorta; or is the result of regurgitation of blood into the inferior vena cava or hepatic vein, which may even give rise to expansile pulsation of the liver. Cardiac epigastric impulse is either due to displacement of the heart, or enlargement of the right ventricle; or it may be the natural consequence of a short thorax.

### III. PECULIAR SENSATIONS FELT OVER THE CARDIAC REGION.

**1. Thrill or purring tremor.** These terms sufficiently indicate the special character of a peculiar vibratory sensation conveyed to the fingers, which is indicative of certain conditions of the orifices and valves of the heart. In order to determine the origin of a thrill, it is necessary to observe its *situation* and *synchronism*. It may be requisite to excite the heart by brisk movement before it can be felt. The different thrills which may be met with are as follows, and more than one may be present in the same case:—(i.) At the left apex—*a.* Systolic, indicating mitral regurgitation, especially if accompanied with hypertrophy and mitral obstruction. *b.* Præsystolic, associated with mitral obstruction. (ii.) Systolic in the second right interspace near the sternum, due to aortic obstruction; or more extensively if the aorta is at the same time diseased or dilated. (iii.) Diastolic, felt down the sternum, occasionally observed in aortic regurgitation. (iv.) Very rarely systolic over the inner part of the second left space or opposite the third cartilage, indicative of pulmonary obstruction. (v.) Præsystolic in the fourth left space or opposite the fourth cartilage. This is a mere curiosity, but has been said to accompany tricuspid obstruction.

**2. Pericardial friction-fremitus** is very exceptionally observed in pericarditis, but it may be felt over more or less of the cardiac region. Differing in its characters entirely from a thrill, it gives the impression of being quite superficial and rubbing; is movable, and irregular as regards its site and rhythm, though usually felt chiefly during the systole; and seldom lasts for any length of time. It may be simulated by pleuritic fremitus caused by the action of the heart. A curious sensation is sometimes felt, due to the morbid changes remaining after an attack of pericarditis.

### IV. CARDIAC PERCUSSION.

**(A.) CARDIAC DULNESS.**—This is described as being *superficial* and *deep*. The former corresponds to the part of the heart uncovered by lung, and is triangular in shape, being bounded towards the right by a line along the middle of the sternum from between the fourth cartilages; and



towards the left by a line extending obliquely from the same point to the apex. The deep cardiac dulness extends as far the limits of the heart, but requires much practice in order to mark it out.

**CARDIAC DULNESS IN DISEASE.**—The points requisite to be noticed are:—  
1. Position. 2. Extent and directions of increase. 3. Shape. 4. Degree and quality. 5. Effects of change of posture.

1. **Position.** This may be entirely abnormal, as, for instance, when the heart is displaced to the right by pleuritic effusion.

2. **Extent and directions of increase.** (i.) The area of cardiac dulness may be *increased* more or less, this being usually associated with some change in shape. *Causes.* *a.* Abnormal contact of the heart with the chest-walls, especially when due to retraction of the lung. *b.* Enlargements of the heart, the extent and direction of the increased dulness depending upon the part of the heart involved, and the nature of the enlargement. *c.* Accumulation or clotting of blood within the cavities, or congestion of the walls of the heart, especially as the result of some pulmonary obstruction. *d.* Any liquid or solid collection within the pericardium, but especially effusion from inflammation, the dulness then increasing chiefly in an upward direction; and excess of fat. *e.* Increase of cardiac dulness may be simulated by conditions external to the heart, for instance, consolidation of the margin of the lung; accumulation of fat; a solid tumour; or aneurism of the aorta. (ii.) *Diminution* of cardiac dulness is not reliable in determining the condition of the heart, but is often most useful in indicating distension of the lungs, especially the left. *Causes.* *a.* Atrophy of the heart. *b.* Great loss of blood and consequent emptiness of the cavities. *c.* Accumulation of air in the pericardium. *d.* Hypertrophy or emphysema of the lungs.

3. **Shape.** The form of the cardiac dulness often affords important evidence as to the cause of any increase in its extent. In pericardial effusion it tends to be triangular, with the base down and the apex upwards. In hypertrophy it becomes elongated vertically; in dilatation lateral enlargement takes place, especially towards the right, and the outline is square or circular. The form of dulness, however, will be modified according to the part of the heart involved, and the degree in which the two kinds of enlargement are combined.

4. **Degree and quality.** The degree of dulness sometimes affords a distinction between pericardial effusion and cardiac enlargement, being more marked in the former. If the pericardium or heart is calcified, the percussion-note may become somewhat osteal in quality.

5. With **change of posture** the dulness due to pericardial effusion may be made to alter in extent and form, but this test is rarely called for, and it may be very dangerous to carry it out.

(B.) **RESISTANCE.**—The sensations conveyed to the fingers on percussion are not very reliable in the diagnosis of cardiac affections; but the sense of resistance is likely to be more marked in pericardial effusion than in hypertrophy.

## V. AUSCULTATION OF THE HEART.

### (A.) SOUNDS OF THE HEART.

It is essential to have a clear comprehension of the mode of action of the heart, and of the sounds associated therewith, before auscultation can be of any value in the investigation of the morbid conditions of this

organ. With regard to the normal sounds, it is requisite to know the characters of each: how these differ as examination is made over different parts of the thorax; and the mechanism of their production.

During each action of the heart, on listening over the apex-beat, there may be noticed in succession:—1. A systolic sound, synchronous with the contraction of the ventricles. 2. A short silence. 3. A diastolic sound at the moment when the ventricles cease to contract, and the aortic and pulmonary valves close. 4. A longer silence, which is again followed by the systolic sound. As regards duration, they bear about the following proportion to each other, dividing an entire cardiac action into tenths:—

<i>Systolic sound.</i>	<i>1st interval.</i>	<i>Diastolic sound.</i>	<i>2nd interval.</i>
$\frac{4}{10}$	$\frac{1}{10}$	$\frac{2}{10}$	$\frac{3}{10}$

At the *left apex*, that is, just within and below the nipple, the systolic sound is prolonged and well-defined; much accentuated; it seems muffled and rather deep; and is of rather low pitch. The diastolic is much shorter, sharper, and more abrupt; clearer; more superficial; and higher-pitched. At the *right apex*, namely, over the base of the ensiform cartilage, both sounds are clearer, and higher-pitched than at the left, and the systolic is less accentuated, shorter, and sharper. Comparing the sounds at the *base* and *apex*, it will be found that at the base the diastolic sound becomes relatively the more marked. It is loud and distinct; well-accentuated; clear and often ringing; while the systolic sound is dull and indefinite; shorter; and without any accent. At the *right base*, that is, opposite the second right interspace or third cartilage close to the sternum, the sounds are usually louder than at the corresponding point on the left side, especially the diastolic. Finally, it must be noticed that the sounds are generally better heard under the left clavicle and over the left side posteriorly than over the corresponding regions on the right side. It is now generally acknowledged that the *systolic* sound is due chiefly to the tension of the mitral and tricuspid valves, and the muscular contraction of the ventricles; though it is supposed by some writers to be modified by the impact of the apex of the heart against the chest-walls, the rush of blood through the aortic and pulmonary orifices, and the vibrations thus set up in the blood-currents. The *diastolic* sound results mainly from tension of the aortic and pulmonary valves; but partly, according to Sibson, to tension of the whole root of the aorta.

In auscultating the heart in order to detect abnormal conditions it may be necessary to make the patient stop breathing for a moment; to excite the heart by a little brisk movement; or to examine in different postures. In order to compare the sounds at the base and apex, some authorities recommend the use of a double stethoscope, so that they may be heard simultaneously, but the ordinary instrument answers perfectly well.

**HEART-SOUNDS IN DISEASE.**—It is highly important to attend to the ordinary cardiac sounds when investigating for morbid conditions, as they frequently afford most valuable information.

1. **EXAMINATION OF THE SOUNDS AT THE LEFT APEX.**—The stethoscope should first be applied over the apex-beat; and the following are the deviations from the normal which may be met with:—

1. **Changes in intensity and apparent depth.**—(i.) *Intensity increased.* *Causes.* (a.) Excited action of the heart. (b.) Approximation of the heart to the chest-walls, when the sounds also appear to be

superficial. (c.) Combined hypertrophy and dilatation, particularly if the valves are somewhat hypertrophied at the same time. (d.) Deficient quantity or a watery condition of the blood. (ii.) *Intensity diminished.* Causes. a. Feeble action of the heart. (b.) Certain organic cardiac affections, namely, atrophy; simple or concentric hypertrophy; dilatation, with thinning of the walls; changes in the muscular walls, especially fatty disease, but also softening associated with fevers, and fibroid or cancerous infiltration. (c.) Collections of fluid, air, or much solid material in the pericardial sac. (d.) Distension of the left lung by emphysema or hypertrophy. In the last two conditions the sounds appear to be deep, in consequence of imperfectly-conducting materials intervening between the heart and the parietes of the chest.

2. **Changes in characters.**—The *pitch, quality, and degree of clearness* of the *systolic sound* may give important information as to the condition of the valves and walls of the heart; and also as to the quality of the blood. In marked hypertrophy without dilatation the sound becomes toneless, dull, obscure, muffled, and of very low pitch. In dilated hypertrophy with some thickening of the valves it may be booming, clanging, or musical. When the heart is merely dilated it is often high-pitched, abrupt, and clicking or slapping. Anæmia frequently causes the systolic sound to become unusually sharp, clear, and high-pitched. The heart-sounds are said to be *impure* when they are wanting in clearness and definition. This may be due to thickening of the valves, irregular tension, or non-synchronous closure of the different cusps.

3. It is sometimes important to notice the **length of the systolic sound**; and to compare the **relative lengths of the sounds and intervals**. For instance, in dilated hypertrophy the systolic sound is very prolonged; there may be hardly any diastolic sound; and the intervals are shortened. In mere dilatation the diastolic sound often becomes the longer one, so as to simulate the systolic, which is much shortened. The first sound at the left apex is also usually remarkably short and abrupt in mitral obstructive disease.

II. COMPARISON OF THE SOUNDS IN DIFFERENT PARTS OF THE CHEST.—It is often of advantage to compare the sounds over different parts of the thorax, but especially at the apex and base of the heart; and at the right and left apex or base. As illustrations of the knowledge thus to be gained the following are important:—1. If the sounds, being weak at the apex, are louder at the base, this serves to distinguish pericardial effusion from dilatation or fatty heart. 2. Greater intensity at the right apex than the left shows either displacement or right enlargement of the heart; or that this organ is unusually covered by some imperfectly-conducting material, especially an emphysematous lung. 3. Marked loudness and accentuation of the second aortic sound at the base often indicates obstruction in the general circulation, either due to degenerative changes in the vessels, or to renal disease, especially the granular kidney, giving rise to increased arterial tension. This may be associated with a very weak first sound, owing to degeneration of the cardiac walls. A change in the aortic sound may also depend on commencing changes in the valves, a dilated aorta, or aortic aneurism. 4. Louder sounds at the left base than the right, particularly the diastolic, indicate that there is some disease, affecting the passage of blood through the mitral orifice, so that the pulmonary circulation is overloaded and the pulmonary artery distended. 5. Any condition, either in con-



nection with the heart itself or external to it, which alters the position of this organ, will correspondingly modify the sounds. For example, in left pleuritic effusion they are transferred to the right side of the chest. 6. The extent and direction of conduction of the sounds may be useful in determining the presence of disease in other organs. Thus, in consolidation at the apex of the right lung they are very often decidedly louder under the right clavicle than the left. In right basic pneumonia they are frequently very marked over the corresponding part of the chest. Cavities in the lungs may intensify the sounds considerably, or sometimes impart to them unusual characters, such as a peculiar hollowness or a metallic quality.

III. REDUPLICATION.—This term implies a *doubling* of either of the sounds of the heart. The second sound is more often affected than the first, but both may be reduplicated in the same case. The slower the action of the heart, the more easily is the doubling appreciated, and very rapid action may abolish it. This phenomenon has received much attention of late years, and has been particularly discussed by Dr. James Barr, of Liverpool.

With regard to the cause of reduplication of the *first sound*, it is generally considered to be due to asynchronism in the action of the two sides of the heart. Dr. Barr refers it definitely to "want of synchronicity in the closure and tension of the tricuspid and mitral valves, or in the initial stages of contraction of the right and left ventricles." Other views are, however, entertained, namely, that it is the result of the resolution of the first sound into its component elements; of non-synchronism in the tension of the different segments of the auriculo-ventricular valves, owing to absence of perfect uniformity in the contraction of the papillary muscles (Guttmaun); of a double click of the left or right auriculo-ventricular valves (D'Espine); while some maintain that the first part of the reduplicated sound is caused by the auricular systole, when the auricle is hypertrophied and acting energetically—in short, that it is *præsystolic*. The auricle is thus supposed to produce a sound by its own contraction (Johnson); or by the effect of its action upon the valves (Sansom, Guttmaun). Doubling of the *second sound* is almost universally regarded as being due to asynchronism in the closure and tension of the aortic and pulmonary valves; but Guttmaun has also attributed this phenomenon to asynchronous closure of the individual segments of the semilunar valves, and to auricular action.

With respect to the causes of the asynchronism on the two sides, which is generally assumed to account for reduplication, there is also a difference of opinion. Usually it is supposed that, in the case of the first sound, it is due to excess of blood-pressure in one or other ventricle, retarding the closure of its auriculo-ventricular valve; in the case of the second sound, to excess of blood-pressure in the aorta or pulmonary artery, accelerating the occlusion of the respective valves. Dr. Barr, however, maintains "that relatively greater blood-supply to one or other ventricle does not retard the closure of its auriculo-ventricular valve, but more quickly overcomes the inhibitory action of the vagus, stimulates that ventricle to initiate contraction, and first apply tension to its auriculo-ventricular valve, which perhaps may be more readily effected on account of the hyper-distension of the ventricle, and thus produces the first element of a duplex first sound." Regarding the second sound, he holds "that duplication does not directly depend on high tension in

one or other artery, but is owing to asynchronism at the end of ventricular contraction and the consecutive reaction of the pulmonary artery and aorta, with tension of their respective valves."

Reduplication is often physiological, being observed in health, especially in relation to the act of respiration. Doubling of the first sound occurs at the end of expiration or commencement of inspiration; of the second sound, at the end of inspiration or commencement of expiration. This phenomenon is also present in certain cases of Bright's disease; and doubling of the second sound has been noticed in connection with mitral constriction, aortic stenosis, and certain pulmonary affections which obstruct the circulation.

The exact characters presented by reduplicated sounds vary, and there is sometimes a danger of mistaking them for a murmur. One form of reduplication is that which has been called *bruit de galop* by Potain, who noticed it in cases of hypertrophy of the heart associated with granular kidneys, but it may be present in other conditions. As Dr. Barr rightly observes, this *bruit* is not a galop but a canter.

### (B.) ENDOCARDIAL MURMURS.

An *endocardial murmur* is usually associated with one of the cardiac orifices, being either one of the ordinary sounds altered in its characters, or altogether a new sound. With regard to the immediate cause of a cardiac murmur, it does not depend upon the friction of blood against rough or irregular surfaces, as was at one time supposed, for a thin layer of motionless fluid exists between the blood current and any such surface. It is due to the passage of the blood through a narrow orifice into a wider space beyond, and has been attributed either to certain "fluid veins" which produce sonorous vibrations, or to friction between the fluid particles of the blood. In order to determine the site of production and the immediate cause of any murmur, it is necessary to observe:—1. The seat of its greatest intensity. 2. The directions in which it is conducted. 3. Its time, whether systolic, diastolic, præ-systolic, or post-diastolic. These being the essential characters, it is always advisable, however—4. To attend to certain other particulars, especially the duration, loudness, quality, and pitch of a murmur; and its effect upon the ordinary cardiac sounds. Thus a tolerably accurate conclusion may be arrived at with regard to the actual conditions of the valves and orifices upon which the murmur depends; the state of the heart's walls, and the manner in which this organ is acting; and the quality of the blood.

GENERAL OUTLINE OF CAUSES OF MURMURS.—1. In the large majority of cases a murmur depends upon some morbid condition in connection with one of the cardiac orifices, which either causes *obstruction* to the onward passage of the blood, or permits *regurgitation* owing to imperfect closure of the valves. (i.) *Obstruction* may arise from:—*a.* Constriction at or about an orifice, its margins being generally thickened at the same time. *b.* Some direct impediment, as from much enlarged and nodulated or adherent valves, which cannot fall back. *c.* External pressure by a tumour, fibrous thickening, or other morbid condition, or by the stethoscope. *d.* Twisting of an orifice, with a wrong direction of the current of blood, consequent upon displacement of the heart. (ii.) *Regurgitation* may be due to:—*a.* Mere widening of an orifice, the valves

not enlarging in proportion. *b.* Organic changes in the valves, which prevent them from performing their functions properly, such as actual destruction or rupture, perforation, contraction, thickening and rigidity, or adhesion to the walls of the heart. *c.* Organic changes in the appendages of the valves, namely, the chordæ tendinæ or muscoli papillares, interfering with their closure. *d.* Mere irregular action or altered position of the muscoli papillares, which prevents the valves from falling into their places at the proper time or in the proper manner. *e.* Degeneration at the root of one of the great arteries, interfering with the adaptation of its valves. 2. Mere roughness of the endocardium may cause a murmur, especially when due to endocarditis, and particularly if in the vicinity of an orifice. 3. Fibrinous coagula among the columnæ carnæ or upon the surface of the valves occasionally give rise to a murmur. 4. Cardiac murmurs may depend upon certain rare morbid conditions, such as sacculated aneurism of the heart; abnormal communications between the cavities of the heart, or between either of these and one of the great vessels; or dilatation of the aorta at its commencement, the orifice being unaltered. 5. An abnormal condition of the blood is liable to cause a murmur, for example, anæmia. 6. Excited cardiac action may render the sounds rough and murmur-like. Murmurs have been appropriately divided into *organic* and *inorganic*, according as they are associated or not with positive organic mischief. The latter will be presently alluded to separately.

CHARACTERS OF MURMURS AT THE SEVERAL ORIFICES.—Theoretically there may be two murmurs in connection with each of the four chief orifices of the heart, one indicating *obstruction*, the other *regurgitation*; but only *mitral* and *aortic* murmurs are usually met with, those associated with the *tricuspid* and *pulmonary* orifices being exceptional.

I. **Mitral Murmurs.**—These are loudest over or just above the apex-beat, being conducted more or less round the left side in an outward direction, and also heard to a variable extent upwards towards the base. 1. *Regurgitant.* Systolic in time, this murmur is generally of medium or low pitch, but varies much in its other characters; it is not uncommonly more or less blowing. It may be so loud as to be heard extensively over the chest, but is not often distinct at the base of the heart, and in many cases becomes abruptly fainter on passing the stethoscope in this direction. It is said, however, that a mitral regurgitant murmur is occasionally loudest a little to the outside of the pulmonary area, or may even only be heard here. This has been explained by Naunyn by the fact that the appendix of the left auricle impinges upon the chest at this point, and he attributes it to the better conduction of the murmur along the course of the regurgitating blood, the fluid veins producing sonorous vibrations louder at the point of impingement than at that of origin; and the dilated auricle being closer than usual to the surface of the chest, and therefore nearer to the ear. The murmur is commonly well-conducted directly backwards round the left side, being perceptible behind in the left vertebral groove, or even sometimes in the right, especially between the sixth and ninth dorsal vertebræ. The exact direction of conduction of this murmur has been supposed to indicate which flap of the valve is mainly involved, the aortic flap being said to be implicated if the murmur takes the direction of the axilla, the outer flap if it is conducted away to the left of the nipple. 2. *Obstructive or constrictive.* Usually an obstructive mitral murmur is post-diastolic or præ-systolic, being entirely a new sound, and having no connection with



the normal diastolic sound. In some cases, however, it seems to begin almost simultaneously with this sound, and to continue through the entire interval. Some observers have described a separate diastolic murmur in mitral obstructive disease, either existing alone or separated by a very short silence from the præ-systolic murmur. Dr. George Balfour describes it as always slightly musical, and as heard either over the mitral area, or where the fourth rib joins the sternum on the left side. The explanation of the mitral obstruction murmur is as follows:—As soon as the ventricle ceases to contract the mitral valves fall back, and the orifice becomes patent. The blood which has collected in the auricle quietly passes through for a while; but finally the auricle, being distended, suddenly contracts, and drives on the blood with some force through the mitral opening, this being immediately followed by the ventricular systole. It is at the time of this auricular contraction that the murmur is usually perceived, and hence it has been called “auricular-systolic.” It may in some cases, however, be heard during the whole of the period that the blood is passing through the orifice. Hence the length of this murmur varies, but it is usually rather short. Its intensity is not very great as a rule, but even when it is loud the extent of its conduction towards the axilla is much less than that of the regurgitant murmur, it being, indeed, often confined to a very limited area, while it is only very exceptionally heard in the back. It seems, however, to be more conducted towards the right than the systolic murmur. Mitral obstructive murmur may only be heard in the recumbent or erect posture respectively, and is often brought out or made more evident by exercise. The pitch of this murmur is low, and quality almost always harsh, sometimes very much so, being almost grating. The late Dr. Hilton Fagge described it as churning or grinding. It is followed by a very short and sharp systolic sound, often mistaken for the second sound. Mitral obstructive murmur may disappear for a time or even permanently.

**II. Aortic Murmurs.** 1. *Obstructive.* Most marked at the base of the heart, generally over the sternum and in the contiguous portion of the second right space, an aortic obstructive murmur is conducted mainly upwards and to the right, but also to some extent down along the sternum and towards the left apex, though it is not often heard at this point. Behind it is often audible in the left vertebral groove, usually from about the second or third to the sixth or seventh dorsal vertebra, but sometimes it can be heard all along the dorsal region and even on the right side. I have met with several instances in which the murmur was so loud as to be heard over the chest and back extensively, as well as for a considerable distance along the main arteries. It is generally prolonged and of moderate pitch, occasionally musical; it may be very harsh or even rasping. 2. *Regurgitant.* This murmur is usually loudest over the sternum, opposite the third space or fourth cartilage, being conducted chiefly downwards along the sternum, so that it is very distinct at its lower end, where it generally abruptly ceases. Towards the right infra-clavicular region it is not nearly so well-conducted as the obstructive murmur, and it is rarely heard in the back. Occasionally it is audible at the apex of the heart, and it is said that this murmur may be loudest here. This has been supposed by Dr. Balthazar Foster to indicate that the posterior or mitral segment of the aortic valve is incompetent. Its rhythm is diastolic, it being in fact an altered second sound; but the murmur is

always prolonged more or less into the interval, and may fill it completely. Indeed, the duration of this murmur is often so considerable as to lead to its being mistaken for a systolic murmur, even when both exist together. Commonly it is of blowing quality, not harsh, and of medium or high pitch; but its characters are variable.

**III. Tricuspid Murmurs.** These are heard at the right apex, that is, over the junction of the xiphoid cartilage and sternum, being conducted a little upwards and to either side. 1. *Regurgitant*. Regurgitation is common at the tricuspid orifice, but as this is due to mere enlargement of the opening, and as the right ventricle does not act powerfully, a murmur is only heard in exceptional instances. When present it is systolic, usually faint, but sometimes loud, and of low pitch. 2. *Obstructive*. This is of rare occurrence, but some cases have been brought forward of late years in which a tricuspid obstructive murmur was present. It is præ-systolic in time.

**IV. Pulmonary Artery Murmurs.** These are chiefly audible at the left base, about the second space and third cartilage or space near the sternum, and are conducted upwards and to the left, so that they are well heard under the left clavicle. As in the case of the aorta, two murmurs may be met with, namely:—1. *Obstructive* or *systolic*. 2. *Regurgitant* or *diastolic*. Of the former several instances have come under my notice; the latter is extremely rare, and is always associated with a systolic murmur.

**CONDITIONS INFLUENCING MURMURS.**—Without entering into particulars, it must suffice to state that murmurs may be modified as regards their intensity, seat, direction of conduction, or other characters by:—1. Deformities of the chest. 2. Posture. 3. Morbid conditions external to the heart, for example, emphysema, pleuritic effusion, lung-consolidation. 4. The state of the walls and cavities of the heart, as regards hypertrophy, dilatation, or degeneration. 5. The force and regularity of the cardiac action. 6. The presence of two murmurs at the same orifice. 7. The existence of two synchronous murmurs at different orifices. 8. The state of the blood.

**INORGANIC MURMURS.**—A brief summary may be given here of the inorganic cardiac murmurs which may be met with. 1. *Anæmic*. This has usually the position of a pulmonary systolic murmur, somewhat blowing or whiffing in quality. It may, however, be situated over the aorta, or be heard at all the orifices. Excited action of the heart, pressure with the stethoscope, and the erect posture intensify an anæmic murmur. The anæmic cardiac murmur has been commonly supposed to be produced at the pulmonary orifice, and to be due either to the watery state of the blood; to pressure upon the pulmonary artery with the stethoscope; or to unusual vibration of the walls of the artery or its valves, in consequence of their being in a relaxed condition. Some authorities, however, among whom is Dr. George Balfour, maintain that the murmur is that of mitral regurgitation, conducted to the left auricular appendix, as already explained, and due to dilatation of the heart, associated with the anæmic condition. Parrot's theory is that the murmur is of tricuspid origin; while others have localized it in the aorta. An aortic systolic murmur is said to be developed late in an anæmic case, and to be due to the large blood-wave sent on by the dilated and hypertrophied heart (Bean). 2. It is generally believed that a murmur may result from *irregular action of the muscoli papillares* in the left ventricle; being of the nature of a slight, or occasionally of a tolerably marked,

though inconstant, mitral regurgitant murmur. It is usually associated with chorea, but may depend upon a very feeble or irregularly-acting heart. Many, however, deny the possibility of a murmur being originated in this way, and attribute the inorganic murmur in chorea to dilatation of the heart and consequent mitral regurgitation. 3. *Excited cardiac action* or *irregular palpitation*, especially if associated with enlargement of the heart, may cause the first sound to become rough and murmur-like, particularly at the base. 4. *Twisting of the heart* may give rise to a basic systolic murmur. 5. *External pressure* generally leads to an aortic obstructive murmur, but occasionally the murmur is seated at the pulmonary orifice. 6. Murmurs due to *clots in the heart* are usually systolic, and connected with the right orifices.

### (C.) PERICARDIAL SOUNDS.

1. **Pericardial murmurs, or Friction-sounds.** A pericardial friction-sound depends upon the rubbing together of roughened surfaces of the pericardium during the cardiac action. The roughness may be due to excessive vascularization; exudation or its remains; coagulated blood; or tubercle or cancer.

CHARACTERS.—In the following description of a *pericardial friction-sound*, the differences between this sound and an *endocardial murmur* will be evident. 1. Its *seat* and *extent* are very variable, but frequently its point of greatest intensity does not correspond to that of any endocardial murmur; while it is usually abruptly limited even when loud, and is not conducted in the directions characteristic of endocardial sounds. It is more frequent towards the base than at the apex. 2. It appears to be distinctly *superficial* as a rule. 3. Great variety is observed as regards the *intensity*, *quality*, and *pitch* of a friction-sound. Usually it is more or less *rubbing* and *rough* in quality, but may be clicking, creaking, or grating, and Walshe describes churning and continuous-rumbling varieties, due to the presence of fluid. It may differ over different parts of the cardiac region. 4. The *rhythm* may be systolic, diastolic, or both, but very often it is irregular, not corresponding exactly to either, and varying with each beat of the heart. A double murmur of maximum intensity at the same spot is considered very characteristic of pericardial origin. In many cases the heart-sounds may be heard quite distinctly through the friction. 5. Pressure with the stethoscope frequently materially modifies a pericardial murmur, by increasing its area or intensity; altering its rhythm; raising its pitch; or rendering it rougher in quality. 6. Bending the body forwards is said to intensify pericardial friction, but this is not reliable. It may disappear in the sitting posture; and a change in position may affect that of the murmur, should fluid be present in the pericardium. 7. A quick inspiration in some cases intensifies friction-sound, and raises its pitch. 8. Rapid changes are liable to take place during the progress of the case, as regards the site, extent, rhythm, and characters of a pericardial murmur.

It is necessary to mention that pericardial rubbing may be simulated by pleuritic friction, modified by the cardiac action. Its situation, which is generally about the left border of the heart; marked irregularity; and cessation when the breath is held, will usually serve to distinguish the latter.



II. A **pericardial splashing-sound** has been described, developed by *succussion*, and due to the presence of air and fluid in the sac, but it is extremely rare.

## B. EXAMINATION OF THE ARTERIES.

In directing physical examination to the arterial system, it is well to attend first to the great vessels of the chest and neck; and afterwards to the arteries of the limbs, especially the brachial and radial.

(A.) **EXAMINATION OF THE ARTERIES OF THE CHEST AND NECK.**—The chief abnormal conditions which may be observed in connection with these vessels may be thus summarized:—

I. **Local bulging**, which may be caused by aneurism.

II. **Changes in the amount and characters of pulsation.** These are usually determined by *inspection* and *palpation*, but the *stethoscope* gives material aid in some cases, while the paper-flag apparatus or the cardiograph may be sometimes used with advantage.

1. *Excessive pulsation* may be associated with:—*a.* Excited action of the heart. *b.* Hypertrophy of the left ventricle. *c.* Aortic regurgitation, which is also characterized by an immediate collapse of the arteries. *d.* An atheromatous condition of the vessels. *e.* Aneurisms of various kinds, which present a limited impulse, usually *expansile* and *heaving*. 2. In cases of mitral regurgitation there is sometimes almost an entire *absence of pulsation* in the carotids and subclavians, even when the heart is much hypertrophied, and is acting powerfully.

III. **Thrill.** Arterial thrill may depend upon:—1. Anæmia. 2. External pressure. 3. Diseased vessels and aneurisms, especially general dilatation accompanied with atheroma or calcification. A thrill may be felt in the suprasternal notch, owing to the aorta being thus affected.

IV. **Abnormal dulness and resistance.** The only morbid condition of an artery which can give rise to this physical sign is an aneurism.

V. **Sounds and murmurs.** 1. Two sounds are said to be usually heard in health in connection with the carotid, and often with the subclavian, being the conducted sounds from the aortic orifice, though Guttmann attributes the sound accompanying arterial expansion partly to the vibrations in its wall. This sound is also sometimes heard in the brachial, the abdominal aorta, and the femoral. Pressure over an artery with the stethoscope will readily produce a murmur at the time of the arterial expansion. This is well observed in connection with the third part of the subclavian artery, especially if the heart is acting forcibly or is hypertrophied, or if there is anæmia. The anæmic murmur is usually very easily produced; of high pitch, and blowing, whiffing, or whizzing quality; and may be heard extensively along the arteries. Tripier has attributed to the arteries a cephalic murmur heard in anæmia over the mastoid process, the occiput, and the eyeball. A double murmur is sometimes produced in anæmia by pressure over the femoral artery. 2. Murmurs in the arteries may be associated with diseases affecting the aortic orifice, and it is affirmed that occasionally mitral murmurs are faintly heard in the carotids. Aortic murmurs are conducted more or less along the arteries. In aortic incompetence the sudden tension of the arterial walls with each cardiac systole gives rise to a sound, and a double sound or murmur may be sometimes present, or is brought out by pressure over the femoral artery in such cases. 3. Pressure by a

tumour, enlarged glands, or fibrous thickening and adhesions may cause an arterial murmur. One of the best examples of this mode of causation is the subclavian murmur heard above or below the left clavicle in some cases of phthisis. 4. Arterial murmurs may be associated with morbid conditions affecting the vessels themselves, including—*a.* Roughness of the inner surface of an artery, due to atheroma, calcification, erosion, exudation, or fibrinous coagula. *b.* Change in the form of an artery, namely, aneurism, in which the murmur may be systolic, diastolic, or both; and coarctation. *c.* Abnormal communication between a large artery and vein, such as between the aorta and superior vena cava. *d.* Vascular tumours.

(B.) EXAMINATION OF THE ARTERIES OF THE LIMBS.—The brachial artery, just above the bend of the elbow, affords the best indications as to morbid states of the arterial system generally, especially atheroma and calcification. On bending the elbow the vessel is then distinctly visible and tortuous, presenting a vermicular motion with each pulsation; while it feels more or less hard and rigid, full, incompressible, and rolls like a cord under the finger.

**The pulse.**—Usually the radial artery at the wrist is made use of for observing the characters of the pulse, but it is often advantageous to attend to other arteries, such as the brachial, temporal, or carotid, and when investigating local morbid conditions special vessels must of course be examined. The methods of examination are by *inspection*, *palpation*, and the use of the *sphygmograph*; and the points to be noticed with regard to the pulse include:—*a.* Its *visibility* or *invisibility*; *b.* *Frequency*; *c.* *Quickness* (sharp, abrupt, slow); *d.* *Volume* (large, full, small, thready); *e.* *Force*, and *degree of resistance* or *tension* (strong, weak, extinct; soft, hard; compressible, incompressible: equal, unequal; *f.* *Rhythm* (regular, irregular, intermittent, lagging behind cardiac systole, continuous); *g.* *Special characters*, both to sight and touch (rigid, tortuous, bounding, hammering, jerky, undulating, with sense of sudden subsidence, vibrating or thrilling, tremulous, dicrotic, or reduplicate). The term *dicrotic*, when applied to the pulse as felt by the finger, implies that this has a sensation of being doubled; now, however, it possesses a special significance, as indicating a peculiar character of the pulse brought out by the sphygmograph. *h.* *Sphygmographic tracings.* *i.* The effects of *change of posture*; and comparison of the characters of the pulse *on opposite sides*, in particular cases. It is in some instances very useful to notice whether there is any difference in the pulse when the arm is raised vertically.

**THE SPHYGMOGRAPH.**—For more complete information respecting this instrument reference must be made to the standard physiological works, and to the writings of Marey, Burdon-Sanderson, Anstie, Balthazar Foster, Mahomed, and others on the subject. The sphygmograph must be seen in order to be properly understood, but it may be stated here what it essentially consists of. An elastic steel spring, of sufficient strength, is provided on the under-surface of one end with a convex piece of ivory, which is placed over the artery, the other end being fixed to the framework of the instrument. By a certain arrangement the movements produced in this spring by the pulsation of the artery are transmitted to a narrow lever moving on a pivot, and long enough to amplify them considerably. At the free extremity of this lever is a little pen, made of flexible metal, which traces the motion, either on a piece of glazed paper by means of ink, or on smoked glass. This paper

or glass is made to travel quickly and steadily in a definite direction, by the aid of an apparatus with clock-work, which is wound up, and the plate can be started or stopped at will by a regulator. As it passes along, the pen traces upon its surface the movements communicated from the pulse through the spring.

A sphygmographic tracing is generally taken over the radial artery, the apparatus being fixed on the front of the fore-arm, with the end of the spring over the artery near the wrist, and being kept in its place by elastic bands passing round the fore-arm, the back of which rests on a pad. It is no easy task at first to fix the instrument so that the pulsations are rendered evident, and to regulate the pressure on the artery so that it shall not be too great or the reverse, and that thus the movements may be made visible in their maximum degree; this regulation is effected by means of a screw, and is a matter of much importance.

*Description of a sphygmographic tracing.* The entire tracing, of which Fig. 21 is intended to give a general idea, is made up of a series of

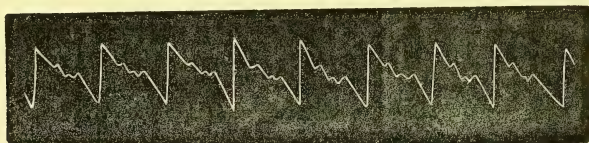


FIG. 21.—Sphygmographic Tracing.

curves or pulsations, each of which corresponds with a complete revolution of the heart's action. It is necessary first to study the characters of an individual typical curve. It may be described as consisting of a *systolic* and *diastolic* part, corresponding respectively to the period of contraction and dilatation of the ventricle; or it may be divided into:—*a. Line of ascent*; *b. Summit*; *c. Line of descent*, in which may be observed

two or sometimes three secondary waves with intervening notches, named, *first* or *distension wave*; *second* or *great wave*, or *true dicrotism*; and *third wave*, which lies between the other two, but is usually absent. In order to explain these different parts, it is necessary to point out certain facts in the physiology of the circulation, of which the sphygmograph has given far more accurate knowledge than was previously possessed; and to indicate their relation to the various parts of a pulse-curve. It will be well to take them in the order in which they occur, illustrating them by Fig. 22.

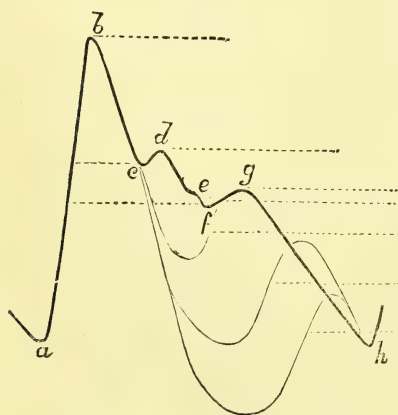


FIG. 22.—Enlarged Sphygmographic Curve.

1. The left ventricle contracts more or less suddenly, opening the aortic valves, which give an impulse to the blood in the arteries; thus is produced the *line of ascent*, *summit-wave*, or *percussion-impulse* (*a* to *b*). 2. After



this sudden vibration the arterial walls partially collapse, which is indicated by the first part of the line of descent, ending in the *first notch* (*b* to *c*). 3. A wave of blood next passes out of the heart into the aorta, and this gives rise to the *first secondary wave*, *wave of distension* or *systolic pressure* (*c* to *d*). 4. After this there is a reflux of blood towards the heart, by which the aortic valves are closed, which corresponds to the portion of the line of descent from *d* to *f*, ending in the *great or aortic notch* (*f*). 5. During this reflux a vibration may occur, originating the *third secondary wave* (*e*), which is placed as it were in the aortic notch, and which, as already stated, is generally wanting. 6. The aortic valves are then suddenly closed by the pressure of the reflux current of blood, and this accounts for the *great secondary wave* or *true diastolic* (*f* to *g*). 7. Finally, the blood flows onward in the vessels, this corresponding to the remainder of the line of descent (*g* to *h*); after which the ventricle again contracts, and the same series of phenomena is repeated.

It will be evident on studying this description, that the systolic portion of the curve extends from the beginning of the line of ascent to the bottom of the aortic notch (*a* to *f*), during which the ventricle is either contracting or contracted; the rest of the line of descent corresponding to the diastole.

In observing a sphygmographic pulse-tracing, the following are the particulars to be noted:—1. The number of the pulsations on the tracing, which gives the exact frequency of the pulse. 2. With regard to each curve:—*a.* the length of the line of ascent, and whether it is vertical or more or less oblique; *b.* the shape of the summit, whether acute, rounded, or square; *c.* the number, size, and position of the secondary waves; *d.* the direction and length of the part of the line of descent beyond the aortic wave, and if there are any undulations in it. 3. The relative characters of the curves in a tracing, especially their height and depth, observing whether their summits and bases are on the same level or not, which may be determined by drawing a horizontal line along the top and bottom of the tracing; these being respectively the lines of the greatest and least arterial tension. In this way the regularity or irregularity of the pulse is determined with precision.

The conditions which chiefly modify the sphygmographic tracing are:—1. The rapidity and force of the ventricular contraction. 2. The degree of arterial tension or resistance, which is influenced by the condition of the walls of the vessels; the controlling effect of the nerves upon them; and the degree of difficulty in the onward passage of the blood, either in the distal part of the artery itself, or through the capillary circulation. 3. The quantity of blood sent into the vessels, which is to some extent dependent upon the duration of the interval between the pulsations, as, if this is long, the blood flows onward, and thus the quantity in the arteries is diminished, and the pressure lessened. 4. The volume of the artery. 5. The condition of the aortic valves.

The more rapidly the ventricle contracts, the more vertical will be the line of ascent, while the height of this line is in proportion to the force of contraction. If the ventricle is acting feebly the summit assumes a rounded form. High arterial tension tends to diminish the height of the line of ascent, and to render it more sloping; to make the first secondary wave proportionately more developed, and to raise it until it ultimately becomes blended with the apex, making this round or square; to obliterate all minor waves; to lessen the aortic wave; and, if there is obstruction to the onward passage of the blood, to make

the remainder of the line of descent slightly convex upwards, and to shorten it. Low tension produces the opposite effects, and is often attended with vibratory undulations in the line of descent; it being only when this condition exists that the third secondary wave is observed.

A healthy pulse-curve presents a line of ascent nearly vertical and of moderate height; an acute summit; and a gradual descent, usually only interrupted by the distension and aortic secondary waves. This form of pulsation is sometimes called *tricrotous*, because it has three waves. It must be remembered that physiological variations of the pulse-tracing will arise from taking food or alcohol, over-exertion, external heat, strong emotion, and other causes.

Certain terms are used in describing sphygmographic curves, which it is requisite to notice. When the first secondary wave is absent or nearly so; the aortic notch deep (owing to the closure of the valves being delayed), so that it is on a level with the base of the curve; and the aortic wave prominent, the pulse is called *dicrotous*. (See Fig. 22.) It indicates very low arterial tension. A minor degree of this variety is named *hypo-* or *sub-dicrotous*. A greater degree, so that the aortic notch sinks below the level of the curve basis, the aortic wave forming part of the line of ascent of the next pulsation, is termed *hyper-dicrotous*. *Monocrotous* signifies that there is only the primary wave; and *poly-crotous*, that there are a number of undulatory vibrations.

*Uses of the Sphygmograph in disease.* Undoubtedly the sphygmograph gives much more exact and accurate information with regard to the circulation than can be obtained by merely feeling the pulse, especially with respect to the action of the heart, and the degree of arterial tension; while it reveals irregularities and inequalities which cannot otherwise be detected. The tracing can also be preserved for future reference. The sphygmograph is employed for purposes of diagnosis and prognosis, and for indicating treatment. Its *diagnostic* value has, by different observers, been advocated in aortic disease, especially regurgitation; cardiac hypertrophy; degeneration of arteries; capillary disease associated with degenerative processes in tissues; renal disease; and aneurisms, in connection with which it is necessary to compare the pulses on the two sides. The characteristic differences in the tracings will be pointed out under the several diseases.

For *prognostic* and *therapeutic* indications the tracings obtained by the sphygmograph are useful in fevers and other acute diseases, such as delirium tremens, pericarditis, or pleurisy, especially by comparing them with the temperature. Among the principal dangerous signs are a marked dicrotous, hyper-dicrotous, or monocrotous pulse; great inequality and irregularity of the tracings; or a small curve, the ascent being short and not vertical, with a rounded or square summit.

### C. EXAMINATION OF THE VEINS.

The veins, from the examination of which most information may be gained, are those of the neck, and the superficial veins of the chest. In many cases it is also useful to observe the superficial veins of other parts, especially those of the abdomen and legs. When examining the veins of the neck, special attention should be paid to the right external



jugular, and to the venous sinus at the junction of the subclavian and internal jugular. The following are the important abnormal signs which may be noticed in connection with these vessels:—

**I. Enlargement.** It is necessary to observe the degree of the dilatation; whether it is permanent or variable; and if the vessels are knotted or varicose. *Causes.* 1. Engorgement and dilatation of the right cavities of the heart. 2. Tricuspid regurgitation. 3. Obstruction of the superior vena cava, innominate, or a more local vein, owing to pressure by a tumour or some other morbid condition; or internal plugging by a thrombus. 4. An aneurism communicating with a large vein within the thorax.

**II. Excessive distension of the veins of the neck after a cough.** During the act of coughing the veins of the neck always fill more or less, but when they are dilated and their valves are inefficient, they become much more distended than usual, and the degree of imperfection in the valves may often be thus indicated.

**III. Pulsation, and filling from below.** Some observers affirm that these characters can be seen in the large veins of the neck even in healthy persons, but at any rate they are then scarcely appreciable. In order to realize them satisfactorily, the patient should assume a recumbent posture, with the head low. Generally pulsation can only be *seen*, but when very powerful it may also be *felt*. Care must be taken not to mistake transmitted arterial impulse for venous pulsation. In order to detect *filling from below*, it is requisite to press upon the *right external jugular vein* near the clavicle, and then draw the finger up the neck along its course, keeping up the pressure; the vein usually fills in jerks. *Causes.* 1. When the veins of the neck are distended, a certain degree of pulsatory movement attends the act of respiration, each inspiration diminishing the distension, and each expiration increasing it. 2. When the right cavities of the heart are over-filled, the systole of the ventricle may cause a vibration to be transmitted through the tricuspid valve to the blood in the auricle, and thence to the veins. 3. Tricuspid regurgitation often leads to venous pulsation, but usually this is soon associated with—4. Inefficiency of the valves of the veins, which renders the pulsation more marked, and which must exist before there can be any filling from below. 5. Right hypertrophy considerably intensifies venous pulsation. Venous pulsation in connection with the liver is occasionally noticed, and of an expansile character. Sudden collapse of the jugular veins during the ventricular diastole is said by Friedreich to be a sign of pericardial adhesions.

**IV. Venous thrill.** A thrill is in very exceptional instances felt in connection with the veins of the neck. It may accompany the pulsation just mentioned; or may be due to extreme anæmia.

**V. Venous murmurs.** 1. *Venous hum*—“*Bruit du diable*.” This is the only venous murmur which is at all likely to be met with, being often present in health, but being particularly common in cases of marked anæmia. It is best heard at the junction of the right internal jugular and subclavian veins, especially on twisting the neck a little to the left; this murmur may, however, be extensively diffused along the veins, being audible sometimes over the thorax and over the cranium. It is continuous and uninterrupted, though not uniform in its intensity; and of variable quality, such as humming or musical, buzzing, rushing, or whistling. Inspiration, pressure, and the erect posture intensify the venous hum; while it is loudest during the ventricular systole, being



in proportion to the force and rapidity of the current of blood. 2. *Intermittent venous murmurs* have been described, associated with anæmia, tricuspid regurgitation, hypertrophy of the right heart, and other morbid conditions, but they must be extremely exceptional.

---

## CHAPTER XX.

### ON CERTAIN SYMPTOMS AND FUNCTIONAL DISORDERS CONNECTED WITH THE HEART.

#### I. ANGINA PECTORIS—SUFFOCATIVE BREAST-PANG.

**ÆTIOLOGY AND PATHOLOGY.**—There has always been much difference of opinion as to the explanation of the symptoms which characterize an attack of angina pectoris, and as to the condition of the heart during the paroxysm. Formerly the complaint was regarded as of neurotic origin, due to some disturbance of the cardiac plexus, leading, according to one view to spasm, according to another view to paralysis of the walls of the heart. That some cases of angina are due to such disturbance is unquestionable, and this is borne out by experiment, as well as by clinical and pathological observation. It may thus arise from intrinsic disorder of the excito-motor cardiac nervous system; from direct irritation of the cardiac branches of the vagus; or from reflex excitement, especially set up by irritation in connection with the abdominal organs. The pathology of the majority of cases of true angina pectoris is at present, however, differently explained. It is supposed that it arises from general vaso-motor spasm, due to increased stimulation of the vaso-motor nerve-centre, which leads to spasmodic contraction of the vessels, and this causes acute distension of the left cavities of the heart, which consequently are embarrassed and act with difficulty, being unable to empty themselves perfectly. During a paroxysm there is marked rise in blood-pressure, with increased arterial tension, as was first shown by Dr. Lauder Brunton by the aid of sphygmographic tracings. Should an attack prove fatal, the heart will stop acting in a state of full distension, and at *post-mortem* examinations it is generally found flaccid rather than rigidly contracted. Dr. Gairdner suggests that the condition is aggravated by cardiac anæmia, due to vaso-motor spasm of the smaller arteries in the heart itself. Therefore true angina pectoris is part of a morbid process, and not a disease of the heart *per se* (Fothergill).

In the large majority of cases angina pectoris is associated with some previous disease of the heart or pericardium, and some change in the nutrition of the organ has always been found in fatal cases. The morbid conditions which have been most commonly observed are extensive atheroma or calcification of the coronary arteries; fatty degeneration of the heart; and flabby dilatation. Anginal attacks do, however, no doubt occur in persons whose heart is healthy, and these are the cases in which there is comparatively little danger, the organ being capable of recovering from the sudden distension to which

it is subjected; the danger increases in proportion to the degree and extent of structural degeneration of the cardiac walls.

With regard to the *exciting causes* of an anginal attack, the original one can seldom be attributed to any obvious cause, except undue bodily exertion. The first paroxysm has been said to set in generally while the patient is walking up a hill, against the wind, after a meal, and especially after breakfast. Not uncommonly subsequent attacks come on after the first sleep at night. Sudden or powerful agitation, and various forms of emotion may bring on a paroxysm. Exposure to external cold is not an uncommon cause, owing, as it is supposed, to its producing contraction of the cutaneous capillaries. Anginal seizures may also arise from reflex disturbance in connection with the abdominal organs, as from heavy meals, indigestible food, or dyspepsia. One form may depend upon direct irritation of the cardiac nerves by growths.

Certain distinct *predisposing causes* of angina pectoris have been made out, namely, the male sex, probably because men are more subject to lithiasis; advanced life, true angina being rarely observed under 45 to 50 years of age; and a high social position. Several eminent men have died from this complaint. It is believed to be connected with the gouty diathesis, and imperfect anginal attacks are frequently observed in gouty persons, also occurring comparatively early in life in individuals whose fathers have died of angina (Fothergill).

**SYMPTOMS.**—An attack of angina pectoris comes on as a rule with abrupt suddenness, but warnings of its approach are occasionally present, in the way of curious sensations or slight pain about the cardiac region.

The chief symptom is an intense pain in some part of the præcordial region, generally referred to mid-sternum, which may amount to the most excruciating torture. In character it is described as shooting, plunging, tearing, aching, gnawing, sickening, or burning, but it is often indescribable. At the same time a feeling of oppression or constriction is experienced across the chest, as if it were being forcibly compressed and could not be expanded, attended with a sense of suffocation and inability to breathe, though this act is not really interfered with, and there is not the least indication of cyanosis. If a deep breath can be taken and held, this may relieve the pain. Usually no tenderness is felt, but pressure rather gives relief, though occasionally tenderness over the sternum and adjoining spaces is complained of. In most cases painful sensations shoot from the cardiac region in various directions, especially down the left arm, or, in exceptional instances, the right, even to the fingers, in which there may be sensations of tingling or numbness; upwards along the left side of the neck; directly backwards; or round the side. This is due to the connection of the cardiac with the cervical and first dorsal nerves.

These symptoms are accompanied with signs of grave general disturbance. The face becomes pale, sunken, and covered with cold sweat; while the expression is indicative of the intense anxiety, alarm, and dread of impending death which the patient feels. In most cases the pulse tends to become feeble, and even fluttering or irregular if the attack is prolonged, though at first there is increased tension. The general surface is often pale, cold, and dry; and the patient may feel general chilliness, with chattering of the teeth. Much will depend on the condition of the heart with which the angina is associated, which also necessarily influences the *physical signs*. Occasionally

vomiting and eructations accompany the attack. The patient is quite conscious at first, but in prolonged or fatal cases may fall into a state of syncope, and spasmodic movements or even general convulsions may be observed.

Usually an entire attack of angina is made up of several brief paroxysms with intermissions, but there may be only one; the morbid sensations generally cease suddenly, this being attended with a sense of extreme relief, though a feeling of exhaustion is afterwards experienced, which may last for some time. Very rarely does the first attack prove fatal, but it may thus terminate, either suddenly or gradually. Probably some cases of sudden death are due to angina. A marked character of the complaint is its great tendency to recur under the influence of very slight exciting causes.

A form of angina pectoris is described, which is not attended with pain—*angina sine dolore*. Here also an affection may be alluded to, named *pseudo-angina pectoris*, which is probably of a neuralgic character, and is met with mainly among young persons, being characterized by sudden pain and unpleasant sensations about the heart, palpitation, disturbance of breathing, faintness and giddiness, pallor of the face, and feeble pulse. The condition of the patient may appear to be really serious, but very rarely does a fatal termination happen. This complaint is chiefly observed in connection with anæmia; various nervous disorders, especially hysteria; or blood-diseases, such as gout. Females are most commonly affected, and it is not infrequent in connection with the menopause. It may also arise from undue exercise after a full meal.

**PROGNOSIS.**—True angina pectoris is a very dangerous affection, but the false variety is not, therefore it becomes important to distinguish between them. The presence and nature of any organic cardiac lesion will necessarily influence the prognosis materially, and this can only be determined by physical examination. Age has also an important bearing on the prognosis, the complaint being more serious the older the patient is.

**TREATMENT.**—1. In order to *prevent attacks*, any one who is subject to angina pectoris should avoid every possible exciting cause, and it is desirable that he should carry some remedy in his pocket, especially nitrite of amyl, nitro-glycerine, or laudanum, so that it may be made use of immediately there is the least indication of the approach of a paroxysm.

2. *During an attack* any obvious source of reflex disturbance, such as indigestible food or flatulence, must be at once got rid of. The internal remedies usually given for relieving the symptoms are *sedatives*, *anti-spasmodics*, and *stimulants*, especially opium or morphia in full doses, hydrate of chloral, the various ethers, chloroform, spirits of ammonia, musk, camphor, and hot brandy-and-water. Digitalis and belladonna are very useful in some instances, when the cardiac action is much disturbed. *Inhalations* of chloroform or ether, or, still better, of nitrite of amyl should be resorted to with due care in severe cases. Nitrite of amyl has been found especially valuable, giving marked relief, and speedily cutting short paroxysms of angina. Patients are in the habit of carrying this remedy about with them, so that it may be used on the first threatening of an attack. The internal administration of nitro-glycerine in minute doses has been found very serviceable, and this remedy is made into suitable tablets; more recently nitrite of sodium



has been used. Subcutaneous injection of morphia may be found of great benefit. *Local applications*, such as dry heat with friction, sinapisms, or friction with chloroform or belladonna liniment, may at the same time be employed. The use of the constant current has also been recommended. Dr. Gairdner advocates warm mustard pediluvia, with heat applied to the arms and thorax. Hot applications are particularly indicated if cold has induced an anginal attack. In a gouty person the joints of the feet should be irritated.

3. *During the intervals* the treatment indicated is that which applies to cardiac affections in general, in the way of regulating the diet and digestive organs; attending to the general and constitutional condition, as well as to the heart and the state of the blood; and to all hygienic matters. Gout must be especially recognized and treated. Tepid or cold baths followed by friction, and change of air and scene, are often beneficial. A belladonna plaster should be worn constantly over the cardiac region.

For *pseudo-angina* similar remedies are indicated during a paroxysm, but they need not be so powerful. At other times the treatment must be directed to the cause of the complaint, and the condition of the patient.

## II. SYNCOPE.—FAINTING.

The phenomena associated with syncope are due primarily to failure in the action of the heart; which is speedily followed by symptoms resulting from anæmia of the nervous centres; these being succeeded by failing pulmonary functions.

**ÆTIOLOGY.**—The chief *predisposing causes* of syncope are early adult age; the female sex; a nervous temperament; and general weakness, with an impoverished condition of the blood.

*Exciting causes.*—Some of the causes now to be mentioned seem to lead to a condition allied to “shock,” in which the three chief systems appear to be almost simultaneously affected, though probably the nervous centres are first disturbed; and it is not always easy to determine whether a case should be regarded as one of syncope or shock. They may be arranged under the following heads:—1. *Want of blood in the cavities of the heart*, from rupture of its walls or of a great vessel, or any form of severe hæmorrhage; obstruction in the principal veins; or sudden removal of pressure from the great vessels, as when syncope follows tapping for ascites. 2. *Inadequate supply of blood to the cardiac walls*, as from obstruction of the coronary arteries; or a supply of impure blood, as in low fevers, or when a syncopal attack comes on in a hot and crowded room. 3. *Partial or complete paralysis of the muscular tissue of the heart*, either from some organic change; or from nervous disturbance, either centric, reflex, or intrinsic in origin. Numerous causes of syncope act in this way, such as fatty and other degenerations of the heart, flabby dilatation, or simply a weak state of this organ in certain chronic diseases, for example, cancer or phthisis; sudden reflux of blood in aortic regurgitation; various poisonous substances, for example, aconite, tobacco, prussic acid, antimony; violent emotions; severe cerebral lesions; long continuance in a warm bath; reflex disturbance from bad smells or unpleasant sounds; pain of any kind; extensive burns; the passage of a catheter; a shock to the sympathetic trunk, as

from a blow in the epigastrium; drinking cold water when the body is heated; taking indigestible food; or over-eating after fasting. Lightning sometimes kills in this way. 4. *Continued spasmodic contraction of the heart.* 5. *Mechanical pressure upon the heart from without*, as in some cases of extreme pericardial effusion.

**ANATOMICAL CHARACTERS.**—The state of the heart varies considerably according to the cause of the syncope. After great loss of blood it is usually contracted and empty. When the walls are paralyzed, the cavities are dilated, and contain more or less fluid or coagulated blood. The lungs are usually anæmic, and the nervous centres markedly so.

**SYMPTOMS.**—Syncope may come on quite suddenly, or may even cause instantaneous death. In many cases, however, it is gradual in its onset, there being *premonitory* symptoms, differing in different cases, before actual insensibility occurs. These are a sense of faintness, giddiness, and trembling, with sinking in the epigastrium, nausea, or sometimes vomiting; pallor, especially of the face, with drawn features; chilliness and shivering, or in some cases a sense of heat, there being at the same time cold, clammy perspirations; a very rapid, small, and weak pulse, tending to become irregular and slow, though the large arteries may throb; hurried, irregular, or gasping breathing, often attended with sighing; great restlessness, and occasionally slight convulsive movements; mental confusion; and disturbance of the senses of sight and hearing, indicated by more or less dim vision, extreme sensibility to light, and noises in the ears. When the syncopal state is established, the symptoms are absolute insensibility, with dilatation of the pupils; death-like pallor, with cold and clammy sweats; a slow and extremely weak, irregular, or actually imperceptible pulse; infrequent and irregular respiration, which may ultimately cease altogether. Not uncommonly convulsive movements are observed; and the sphincters may be relaxed, with involuntary discharge of fæces and urine. Examination of the heart reveals feebleness or complete absence of the cardiac impulse and sounds, especially the systolic sound.

The syncopal condition lasts a variable time, and either ends in death or recovery. In the latter case very uncomfortable sensations are usually experienced during the restoration of the patient to consciousness, this being often attended with palpitation, vomiting, or convulsive movements.

**TREATMENT.**—1. Any obvious reflex cause of syncope should be at once removed. 2. It is most important to attend to the posture of the patient, which should usually be horizontal, with the head low. Fainting may not uncommonly be prevented by bending forwards, and hanging the head down between the knees as far as possible. 3. All clothing should be loosened about the neck and chest, and plenty of fresh air admitted. 4. The application of ammonia to the nostrils; dashing cold water in the face; or friction along the limbs and over the heart, either with the hand alone or with some stimulating liniment, will often restore vitality. 5. The internal administration of *stimulants*, such as brandy, wine, ammonia, ether, or musk, is most useful, and if these agents cannot be swallowed and there is evident danger, stimulant enemata should be employed, or in grave cases ether might be injected subcutaneously. 6. Attempts may be made to confine the blood to the central organs, by making pressure on the arteries of the limbs by the aid of the fingers or of tourniquets, warmth being maintained in the parts thus deprived of blood by means of hot bottles and friction. 7. Sina-

pisms or turpentine stupes over the heart should be employed, if necessary; and in dangerous cases of syncope the judicious employment of galvanism, artificial respiration, and transfusion are the most potent remedial means available. The last is particularly valuable if the syncope is due to great loss of blood. Gentle tapping over the cardiac region has been found useful in rousing the heart to action.

### III. DISORDERED CARDIAC ACTION.—PALPITATION.

**ÆTIOLOGY.**—The cardiac action may be disordered in various ways, but it will only be practicable to refer here to the more prominent disturbances met with. Palpitation implies more or less excessive action. It occurs under a variety of circumstances, but is always a sign of one or other of the following conditions, which affect the action of the heart, namely:—1. Muscular failure. 2. Laborious effort to overcome some impediment; or in consequence of the organ having to act under a physical disadvantage. 3. Nervous excitement. 4. Nervous exhaustion. The nervous disturbance may be intrinsic in the heart itself, or of centric or reflex origin.

**Exciting causes.**—These may be arranged as follows:—1. *Acute or chronic organic diseases of the heart or pericardium.* These may induce palpitation either because the muscular tissue is involved, the cardiac action being thus more or less impaired; or because there is some obstruction to the circulation at one or other of the orifices, which the heart cannot overcome. When palpitation accompanies hypertrophy, it is presumed to be due to the fact that this is insufficiently compensatory, and it may indicate commencing degeneration of the heart-substance. 2. *Mechanical interference with the cardiac action*, as from tight lacing; distorted chest; displacement by pleuritic effusion; or abdominal enlargements, of which flatulent distension of the stomach is a common form. 3. *Obstruction in the vessels*, either from a diseased condition of their walls or high arterial tension, in connection with vascular spasm, atheroma or calcification, or Bright's disease. 4. *Chronic affections of the lungs which interfere with the circulation*, such as bronchitis and emphysema. 5. *Some abnormal state of the blood*, either as regards its quantity or quality, for example, plethora or anæmia; the condition associated with gout, renal disease, or fevers; and, probably, the admixture of materials introduced into the system from without. There is a difficulty in driving on the blood under these circumstances, and thus the heart becomes easily disturbed, while at the same time its own tissue is supplied with impure blood. 6. *Causes acting through the nervous system*, for example, prolonged cerebral excitement or undue mental labour; emotion; functional nervous disorders (hysteria, epilepsy, chorea); the abuse of tea, alcohol, or tobacco; reflex disturbance, originating in the alimentary canal, as from eating indigestible food, or in connection with the genital organs. Palpitation is a prominent symptom in exophthalmic goitre. The view has been advanced that nervous palpitation is chiefly due to spasmodic contraction of the arterioles, whereby a difficulty in the passage of the blood is induced; no doubt, however, the innervation of the heart itself is disturbed.

In some cases palpitation is present at all times more or less, though in such instances it tends to be aggravated by anything which



throws extra work upon the heart, such as slight exertion. In other instances it occurs only in paroxysms, which are brought on by some evident exciting cause, or are independent of any such cause. Palpitation due to muscular failure is most marked after effort.

**Predisposing causes.**—Occupation, habits, and numerous other causes often predispose to palpitation. The individuals most frequently affected are young adults, and persons beyond middle age; females; nervous subjects; and fat, flabby people, who live highly, and are habitually dyspeptic.

**Irregularity** is a form of cardiac disturbance often existing alone or accompanying palpitation, and it is frequently a serious indication of deficient heart-power. It may affect only the rhythm or force of the heart's action, or both. Rhythmical irregularity is due to a halting, hesitation, or partial arrest of the ventricular contraction, which may be brought about by a disturbance of the balance of power between the vagus and cardiac ganglia; or, more commonly, between the opposition offered to the blood to be driven and the power to drive it (Fothergill). It is often, but by no means necessarily, associated with grave organic disease, such as dilatation, fatty degeneration, or mitral disease, especially mitral stenosis according to some observers; or with low conditions of the system, such as malignant fevers. The irregularity may appear to be of a hesitating or anticipating character; sometimes it passes through regular cycles, but in other cases the cardiac action seems altogether confused.

**Intermittency** is the most advanced evidence of cardiac failure, and signifies that there is a complete arrest in the ventricular contractions, until two or sometimes more auricular contractions have occurred, which are required before sufficient blood is sent into the ventricle to rouse its walls into activity. The conditions with which it may be associated are fatty degeneration of the heart; aortic obstruction; hypertrophy and dilatation; irritation of the vagus nerve, either at its root from cerebral disease, or in its course from pressure by a tumour; the advanced stages of severe fevers; diseases of the lungs causing great obstruction to the circulation, the left ventricle being disturbed under these circumstances along with the right; or mere nervous disorder of the heart. Intermittency may even be brought on voluntarily, by holding the breath.

**SYMPTOMS.**—Palpitation is generally accompanied with increased frequency and quickness of the heart's beats, as well as with augmented force, especially when a severe paroxysm comes on. The action may be quite regular; or attended with various forms of irregularity, or with intermittency. There is also frequently some inequality in the force of the cardiac pulsations. Various unpleasant subjective sensations are usually experienced over the cardiac region, the patient being conscious of the heart's action, and this may be associated with a sense of rolling, jogging, sudden falling back, jumping into the throat, and other indefinable feelings—*precordial distress* or *anxiety*. There is occasionally considerable pain, which may be almost anginal, and this is relieved in some cases by pressure. Severe paroxysms are often attended with very serious symptoms, namely, faintness, occasionally ending in actual syncope, especially in cases of palpitation due to nervous exhaustion; dyspnoea, with hurried breathing, and an inability to "catch the breath;" flushing of the face, with a sense of heat, headache, giddiness, disturbed vision, and noises in the ears; and cold, clammy extremities. Sometimes there is much anxiety and fear of dissolution. The radial pulse usually corre-

sponds to the heart's beats, but not always; it is in many cases small and weak, even when the heart is acting violently and the large arteries throb, being generally also quick and sharp.

A form of cardiac disturbance has been described under the term "irritable heart," first applied by Dr. Da Costa to cases observed by him in the American War, and due to hard field service, diarrhœa, fevers, and other depressing causes, especially acting upon persons of nervous temperament, under circumstances of intense excitement. Similar cases have been recognized in our own army, and also in civil life, being in the latter case particularly induced by over-work combined with worry and anxiety, though it may be brought on by other causes. Those instances in which cardiac disturbance occurs in high altitudes, especially as the result of some over-exertion, may be mentioned in this connection. Irritable heart is characterized by a variable degree of palpitation, often brought on by exertion, or occurring when the patient is in bed, especially when lying on the left side, and frequently attended with much distress; a small and rapid pulse, easily compressible, and much influenced by posture; often pain about the cardiac region and left shoulder; embarrassed breathing; and nervous symptoms. It is often very intractable.

The *duration* and *severity* of a fit of palpitation vary greatly, the symptoms being usually more serious when there is irregularity of the heart. The paroxysm is often terminated by profuse diuresis of light-coloured urine; or a sense of much exhaustion follows, which calls for a prolonged sleep. The palpitation induced by drinking strong tea is sometimes of a very distressing character. Occasionally this symptom is constantly present in great intensity, apart from any organic cardiac disease, of which I have met with some well-marked examples in young women, generally, but not always, associated with Graves's disease.

*Physical signs* will of course depend upon whether the heart is organically affected or not. The signs which may be due to mere palpitation are as follows:—1. *Impulse* is too extensive; often strong, but not heaving; and it may be irregular in rhythm and force, jogging, fluttering, or intermittent. 2. *Dulness* is occasionally increased towards the right in prolonged cases, from over-distension of the right cavities with blood. 3. *Heart-sounds* are often louder than natural, with a marked tendency to reduplication; and may be irregular. 4. Occasionally a *temporary systolic murmur* may be heard at the base of the heart, or at the left apex, the latter being supposed to arise from irregular action of the muscoli papillares.

Intermittent action of the heart is sometimes attended with the most distressing and horrible sensations, there being an intense dread of impending dissolution.

**DIAGNOSIS.**—The important matter as regards diagnosis to be determined is to find out the cause of the disturbances of the heart's action included under the term palpitation, and particularly to ascertain whether they are due to organic disease or not. A satisfactory conclusion can only be arrived at by making a thorough physical examination, and by taking into consideration all the circumstances of each case. The impulse of palpitation differs from that of hypertrophy in not being heaving in quality.

**PROGNOSIS.**—This will depend greatly upon the cause of the disturbed action, and especially upon the presence and nature of any organic disease. It must not be thought, however, that simple palpitation is

harmless, for it may prove very serious. Irregularity or even intermittency is by no means a certain sign of organic disease, as both these conditions may be associated with mere functional disorder.

TREATMENT.—1. *During a paroxysm* of palpitation the chief measures to be adopted are to get rid of every source of reflex irritation, or of any other obvious cause; to enjoin perfect quiet, and calm the patient as much as possible; to administer *antispasmodics*, *sedatives*, or *stimulants*, as may be required, such as brandy, ether, ammonia, opium or morphia, hydrocyanic acid, bromide of potassium, tincture of henbane, musk, camphor, tincture of lavender, galbanum, or assafoetida, as well as medicines which act upon the heart directly, especially digitalis; and to apply dry heat or sinapisms over the præcordial region, with heat to the extremities, if necessary.

2. *During the intervals*, as well as in cases of a *chronic* nature, it is necessary to look to the state of the heart, digitalis being often most valuable for improving its action when there is muscular failure; to avert every possible cause of fits of palpitation, by removing mechanical pressure, getting rid of reflex irritation, attending to the diet and digestive organs, and regulating the habits generally, especially avoiding excess in the use of alcohol, tobacco, or tea, as well as over-study and other forms of mental disturbance, and venereal excess; to treat any constitutional diathesis or local complaint, such as gout or renal disease; and, if requisite, to improve the condition of the system generally, but especially that of the nervous system and blood, by giving mineral tonics, mineral acids, quinine, strychnine or tincture of nux vomica, or various preparations of iron; aided by the employment of cold or tepid baths, douches with friction, a proper amount of exercise, and change of air and scene. A mixture containing tincture of steel, nux vomica, and digitalis is often most beneficial. A belladonna plaster may be worn from time to time over the cardiac region.

The principles of treatment applicable to the other forms of cardiac disturbance are similar to those just indicated.

## CHAPTER XXI.

### DISEASES OF THE PERICARDIUM.

#### I. ACUTE PERICARDITIS.

ÆTIOLOGY. — Cases of pericarditis may be classed as *primary* and *secondary*, according to their mode of origin; the great majority belong to the latter class. The affection may arise under the following circumstances:—1. In connection with certain *blood-diseases*, especially rheumatic fever and Bright's disease, and occasionally pyæmia, typhoid, typhus, variola, scarlatina, puerperal fever, gout, scurvy, or purpura. It has been stated to result from the condition of the blood in cyanosis; or after the cure of cutaneous diseases of long continuance. 2. From *injury*, such as a wound of the pericar-



dium, or its laceration by fractured ribs — *traumatic pericarditis*. 3. From *perforation*; for example, a neighbouring abscess opening into the pericardium — *perforative pericarditis*. 4. From *extension* of adjoining inflammation; or irritation set up by neighbouring disease, as pleurisy, pneumonia, chronic cardiac disease, aneurism of the aorta, abscesses in the vicinity, carious ribs, tumours. In these cases the pericarditis tends to be localized. 5. From *irritation* by some new formation in the pericardium, as cancer or tubercle. 6. *Idiopathic* pericarditis has been described, resulting from “cold”; but probably cases of this kind are of a rheumatic nature.

ANATOMICAL CHARACTERS.—The morbid appearances in pericarditis are similar to those observed in other serous inflammations, and they run a similar course. The exudation is generally deposited on both surfaces, but is usually most abundant on the visceral portion of the pericardium; it is rarely observed over the whole extent of the sac, being generally in patches, and it may be confined to a small area, especially about the great vessels. The thickness and mode of deposit are very variable, the lymph being either stratified, or presenting little elevations, ridges, bands, masses, and numerous other arrangements. Usually the material is tolerably consistent, and sometimes quite tough, adhering fairly to the surface. In low conditions it may be soft and granular. The effusion is generally sero-fibrinous, with flocculi floating in it; in exceptional cases it may contain an admixture of blood or pus, and in extremely rare instances is actually purulent. The quantity is not usually above from eight to twelve ounces, but may range from an ounce or two to three pints or more. Gas is sometimes present, arising from decomposition of the fluid. Sloughing of the pericardium is said to occur occasionally.

The processes of absorption and adhesion are precisely identical with those described under pleurisy. The adhesions may be merely in the form of loose bands, or of more or less extensive agglutinations of the two surfaces, and the inflammatory process sometimes extends through the pericardium, so as to cause its union with the chest-walls. When seated about the great vessels, the lymph often leads to their adhesion to one another; or it may remain as a hard mass of considerable thickness.

Allusion may here be made to the so-called *white patches* sometimes observed on the pericardium. As a rule these are decidedly merely due to friction; but they may be the remnants of inflammation.

SYMPTOMS.—It is in the course of acute rheumatism or Bright's disease that pericarditis almost always comes under notice in ordinary practice, and it should be particularly looked for in these affections. Pericarditis may set in without any evident symptoms, but this is not usually the case, though it must be noted that the clinical phenomena observed will be modified considerably by the condition with which the disease is associated; as well as by its combination with other cardiac inflammations, or with distinct complications, such as pneumonia.

At the outset *local* symptoms are generally present, namely, pain, tenderness, and disturbed action of the heart. The pain is generally felt over a part or the whole of the præcordial region, occasionally in the epigastrium, while sometimes it shoots in different directions; its severity and characters vary widely, it being described as mere uneasiness, or dull aching, shooting, stabbing, burning, or tearing, and it may amount to the most intense suffering. On the other hand, pain

may be entirely absent. Tenderness is experienced as a rule over the corresponding intercostal spaces, as well as in some instances when upward pressure is made over the epigastrium. The disturbed cardiac action is indicated by palpitation, sometimes violent.

An attack of pericarditis may be ushered in by slight rigors, followed by pyrexia. When it occurs in connection with acute rheumatism, however, such phenomena are commonly absent, there being no increase of fever previously existing. The pulse is necessarily hurried, and may be very frequent.

When fluid accumulates in the pericardium the pain generally subsides, but the action of the heart is interfered with, as well as the functions of neighbouring structures, in proportion to the quantity of the effusion, and the rapidity with which it collects. Hence serious symptoms are liable to arise, indicating either a tendency to syncope; overloading of the right heart and venous system; interference with the respiratory functions; or grave nervous disturbance. The pulse often becomes very frequent, feeble, small, and in bad cases irregular; sometimes it is slow and laboured. Dyspnoea is present, and may be extremely severe, even amounting to constant or paroxysmal orthopnoea, and accompanied with a sense of great oppression across the chest. A dry, irritable, spasmodic cough is not uncommonly observed. In grave cases the face assumes a very anxious and distressed expression, and becomes pale or more or less cyanotic; the expired air is cool; and the extremities feel cold. The mode of decumbency is generally on the back, with the head high; some patients prefer lying on the left side, others on the right; while occasionally they are obliged to be propped up, or even to bend forwards. Frequently there is much restlessness, provided the patient is not prevented from moving, on account of the pain which accompanies rheumatic fever. Headache and sleeplessness are common symptoms, and among occasional serious nervous phenomena may be mentioned delirium, occasionally almost maniacal; stupor; subsultus tendinum and jactitation; clonic or tonic spasms; choreiform or epileptiform symptoms; and dysphagia. In most cases, however, these probably depend rather on the general condition with which the pericarditis is associated. Vomiting is sometimes a prominent symptom. Should death take place, it usually results either from failure of the heart's action and consequent syncope, which may be sudden, from making the patient assume a sitting posture when the pericardium is very full of fluid; from interference with the aëration of the blood and with the circulation, the lungs becoming œdematous, and dropsy setting in; or from nervous disturbance.

**PHYSICAL SIGNS.**—In the **early stage** the only reliable signs of pericarditis are:—1. *Excited action* of the heart, as evidenced by the impulse. 2. *Pericardial friction-fremitus*, which is extremely rare. 3. *Pericardial friction-sound*. It must be mentioned, however, that friction-sound may be absent, either on account of the soft consistence of the lymph; or from its being deposited only on one surface, or at the back of the heart.

In the **effusion-stage** the *physical signs* are more or less marked in proportion to the quantity of the fluid, being of the following nature:—1. There is usually *bulging* of the cardiac region, especially in young persons. This may extend from the 2nd to the 6th or 7th cartilage, the spaces being widened or even protruded, and sometimes the left edge of the sternum is pushed forwards. Local measurements are increased.

2. The *impulse* presents several important changes. *a.* It is displaced, usually upwards and to the left, but sometimes downwards; while its position alters with change of posture. *b.* Its force is much diminished, and the impulse may be visible when not perceptible to the touch. Often it can be felt in the erect or sitting posture, when absent in the lying posture. There is sometimes great irregularity in its force. *c.* In rhythm the impulse tends to be delayed slightly after the systole; it may also be extremely irregular. *d.* The character is undulatory when there is much fluid, this being observed over a variable area, and being modified by position; the undulations usually appear to pass from below up and from left to right, but they may have a horizontal direction.

3. *Cardiac dulness* is materially altered in extent, degree, and shape. It increases first about the base of the heart, extending upwards and then laterally. Usually it does not pass below the 6th rib, but in extreme cases may reach considerably lower than this, the fluid pushing down the diaphragm and causing protrusion of the epigastrium. In an upward direction it may extend as high as, or even above the clavicle; and transversely from the right border of the sternum to beyond the left nipple. A very important character may be observed when the fluid is abundant, namely, that dulness extends to the left beyond the apex-beat. The shape of the dulness is more or less triangular, with the apex upwards. Its intensity is unusually marked. Change of posture will modify it; the area is larger in the lying than the sitting position, but if the amount of fluid is not very great, it extends higher in the latter posture.

4. The *heart-sounds* are more or less feeble at the apex, and appear to be deep and distant; but on passing the stethoscope upwards towards the base, they become louder and more superficial. Change of posture may influence the sounds.

5. It is said that a *basic systolic murmur* is heard occasionally, due to pressure of the fluid on the aorta.

6. *Friction-phenomena* often persist for a variable time while the fluid is accumulating, becoming by degrees less marked, or being only observed in certain positions.

7. Pericardial effusion will necessarily affect *neighbouring structures*, especially the lungs. Vocal fremitus and resonance, as well as breath-sounds, are diminished in area over the cardiac region; ægophony is occasionally heard above and to the left, while vocal resonance is intensified at the borders of the dulness. Impaired resonance at the base of the left lung may possibly be observed, indicative of partial collapse, the result of pressure by the distended pericardial sac on the bronchus or lung. The liver and spleen are in some cases depressed, along with the diaphragm.

Should **absorption** take place, the signs become gradually normal in favourable cases, and it is only requisite to notice that the *dulness* diminishes from above and laterally; and that the *friction-signs* return, usually in an increased degree, the sound also assuming more of the "churning" and "clicking" characters. The phenomena indicating chronic adhesions will be hereafter considered.

**DIAGNOSIS.**—In the early period the chief matter in diagnosis is to distinguish *pericarditis* from *endocarditis*. Symptoms are by no means reliable, but severe local pain would be in favour of pericarditis. The diagnosis, however, must be founded on the different characters of the morbid sounds present in each disease, aided occasionally by the existence of friction-fremitus. When any difficulty is experienced, which is not uncommon at first, the case must be thoroughly watched in its further progress. Pericardial friction might be mistaken for pleuritic



friction of cardiac rhythm; or it might be simulated by oedematous integuments, fluid in the mediastinum, or the friction of a cirrhotic liver.

*Pericardial effusion* is most liable to be mistaken for *cardiac enlargement*, but the circumstances under which the effusion arises, coupled with the symptoms and physical signs which characterize the several conditions, ought rarely to leave any doubt as to the diagnosis. The dulness of pericardial effusion might be confounded with certain extrinsic conditions, which will be presently pointed out. The distinctions between *inflammatory effusion* and mere *hydropericardium* will be indicated after the latter affection has been considered.

TERMINATIONS.—Pericarditis may end in practical recovery, generally, however, more or less adhesions remaining behind; in death; or by becoming chronic, either the effusion remaining, and in rare instances becoming purulent, or even pointing and opening externally; or extensive agglutinations forming, which greatly disturb the cardiac action. As a consequence hypertrophy or dilatation of the heart may follow; or, in exceptional cases, atrophy or fatty degeneration of its structure, owing to pressure on the coronary arteries.

PROGNOSIS.—The immediate prognosis will depend upon the condition with which acute pericarditis is associated, it being, for instance, extremely dangerous in Bright's disease; the amount and nature of the effusion; the previous condition of the heart; the state of the pulse, as indicated especially by the sphygmograph; whether the disease is complicated with other inflammations; and the severity and character of the symptoms. Marked nervous symptoms are of very serious import. The ultimate prognosis must be entirely governed by the conditions remaining behind; extensive agglutination, and permanent displacement of the heart in consequence of adhesions, are untoward events.

TREATMENT.—The principles of treatment in pericarditis are similar to those mentioned as applicable to pleurisy, but as the former occurs almost always in the course of some other complaint, its management must be modified accordingly. When it occurs in connection with acute rheumatism, the treatment for this affection must be persevered in, and opium given freely, for the purpose of calming the excited action of the heart, care being taken, however, to avoid narcotizing the patient, especially should there be any tendency to cyanosis. Venesection and mercurialization are never admissible. A few leeches may sometimes be applied over the cardiac region with advantage in robust subjects, but as a rule the persistent application of heat and moisture over this region, by means of linseed-meal poultices or fomentations, is the most serviceable local treatment. Great care must be taken that the applications are frequently changed; and that the chest is not unduly exposed or chilled. Cold is strongly recommended by some writers, as strongly condemned by others; it certainly should only be tried very cautiously, in my opinion. Aconite, veratrum, and tartar emetic have also been given, with the view of calming the heart's action, but these are dangerous remedies in pericarditis.

For the *removal of effusion*, if this is not readily absorbed, the application of blisters or strong iodine may be tried, if necessary; with *diuretics* and iodide of potassium internally. Tincture of iron in full doses is decidedly a valuable drug to aid absorption; and *tonics* are often useful.

As a rule a good quantity of nourishment is required, and in many

cases a little stimulant is indicated; if there is much depression, with failing cardiac action and pulse, a considerable amount of brandy may be necessary, and digitalis is indicated under these circumstances. The patient must be kept at rest, and should not be examined unnecessarily, or be made to sit up, if there is any danger of syncope.

*Paracentesis* is called for in rare instances, in order to relieve dangerous symptoms, or to remove purulent fluid. Some advocate early recourse to this operation, but, for obvious reasons, it ought not to be rashly practised. The fluid is best removed by means of the aspirateur.

In pericarditis complicating Bright's disease or low fevers, free stimulation is usually required; while opium is contra-indicated, or must be employed with exceeding caution.

The symptoms which are liable to arise in the course of pericarditis must be treated by the usual remedies.

## II. CHRONIC PERICARDITIS—ADHERENT PERICARDIUM.

**ANATOMICAL CHARACTERS.**—Chronic effusion may remain after acute pericarditis; or sometimes the surfaces of the membrane become universally agglutinated, and calcareous matter may be deposited in the adhesions; or the outer surface of the pericardium may further unite with the chest-wall.

**SYMPTOMS.**—Subjective symptoms are often absent in cases of chronic pericarditis, but there may be uneasy sensations, or even a dull pain over the cardiac region, and, in exceptional cases, anginal attacks have been noticed. Disturbed action of the heart; palpitation easily induced; and shortness of breath on exertion are the chief symptoms complained of, if any. From the effects of extensive adhesions upon the heart, grave interference with its action and with the circulation may ultimately arise, and serious organic changes may be set up. In some cases of this kind ascites is a prominent symptom, coming on before, or being out of proportion to, dropsy in the legs. An agglutinated pericardium will seriously increase the danger from an attack of any pulmonary inflammation.

*Physical examination* may reveal the presence of fluid in the pericardium; or, when adhesions have been formed between its surfaces, as well as with the chest-wall, the signs may be more or less of the following character:—1. *Depression* of the præcordial region, with narrowing of the spaces. 2. Increase in extent, or permanent displacement of the *impulse*, especially elevation, there being no other obvious cause for this; the apex-beat being unaltered by change of posture, or by a deep inspiration; or the impulse having altogether unusual characters, being markedly undulatory, or attended with recession of the spaces or of the epigastrium, or with an irregular jogging movement, both systolic and diastolic. An indrawing of the apex may be noticed after the beat in some cases; while in others there is no impulse at all. 3. Usually increased area of *dulness*, which is not altered after a deep inspiration, there being other signs that the lungs do not expand over the cardiac region. When there is extensive calcification, the percussion-sound is said occasionally to have an osteal character. 4. A rough *friction-sound* may be heard over some part of the cardiac region. 5. Sudden collapse of the jugular veins during the ventricular diastole has been described by Friedreich as a sign of pericardial adhesions.

## III. HYDROPERICARDIUM—PERICARDIAL DROPSY.

The important facts relating to dropsy of the pericardium may be thus summed up, and it will at once be seen in what respects it differs from inflammatory effusion:—1. In the great majority of cases this condition is a part of chronic general dropsy; it may set in acutely in Bright's disease; and rare instances have been met with in which it owned a mechanical origin, having resulted from the pressure of an aneurism or other mediastinal tumour, disease or thrombosis of the cardiac veins, or sudden extreme pneumothorax. 2. There are no severe initiatory symptoms; pyrexia is absent; and there is no marked disturbance of the heart's action. 3. The effusion, which is simply serous, is not abundant. Hence there is no bulging; the physical signs indicative of fluid are less marked than in pericarditis; and the dulness is more liable to be altered by posture. 4. Friction-signs are absent throughout. 5. Hydropericardium generally follows hydrothorax, and hence it is preceded by the symptoms and physical signs of the latter condition, the effects of which it necessarily tends to aggravate.

TREATMENT.—This is merely a part of the ordinary treatment for dropsy. Paracentesis might possibly be warranted in extreme cases.

## IV. PERICARDIAL HÆMORRHAGE.

Blood may be found in the pericardium as the result of:—1. *Spontaneous rupture*, either of the heart or a cardiac aneurism; of an aortic aneurism; of one of the coronary vessels; or of vessels in cancerous deposits. 2. *Injury*. 3. *Pericarditis*, the effusion being more or less hæmorrhagic. 4. *Diseased conditions of the blood*, such as scurvy and purpura.

SYMPTOMS.—The symptoms are generally severe in connection with actual hæmorrhage into the pericardium, indicating loss of blood, and interference with the heart's action; but they will necessarily vary with the amount of blood present, and the rapidity of its accumulation. Sudden death may occur. The *physical signs* are those of an accumulation of fluid in the pericardial sac.

## V. PNEUMO-PERICARDIUM.

Gas is occasionally found in the pericardium, either having entered from without, or resulting from decomposition of fluid in its cavity. It might give rise to *tympanitic resonance* over the cardiac region; and to *succussion-plash*, if mixed with fluid.

---



## CHAPTER XXII.

## ACUTE DISEASES OF THE HEART.

## I. ACUTE ENDOCARDITIS AND VALVULITIS.

**ÆTIOLOGY AND PATHOLOGY.**—In ordinary practice acute endocarditis is by far most frequently associated with rheumatic fever, and it is generally supposed that the inflammation is set up by the direct irritation of the endocardium by the poisoned blood; but other views have been advanced, which regard the endocarditis as primary. These views have already been sufficiently discussed in the chapter on Acute Rheumatism. Endocarditis may also be met with in connection with acute or chronic Bright's disease, scarlatina, measles, small-pox, typhoid fever, puerperal fever, pyæmia, septicæmia, pregnancy, parturition, and syphilis. Cases of this disease occasionally arise which can be traced to no definite cause, and are then said to be *idiopathic*. I have already called special attention to the fact that endocarditis may occur without the presence of any of the usual joint-symptoms of rheumatic fever; and that the cardiac affection may be met with in cases of all grades of severity. *Local* endocarditis may result from wounds and injuries, such as the rupture of a valve or of one of the chordæ tendineæ; from irritation by growths; abscess in the walls of the heart; or, it is said, from unusual blood-currents. Endocarditis in chorea has been attributed to the cause last-mentioned. The disease may occur in the fœtus.

There is a special form of the disease, named *ulcerative endocarditis*, which is usually of septic origin, and may be traceable to some foul ulcer or wound, usually seated on the female genital organs, and following parturition. The inflammation is supposed to be due to organisms, and micrococci have been described by Koch, not only in this, but also in other forms of endocarditis. Ulcerative endocarditis may also arise in connection with chronic valvular disease, or with acute rheumatism.

**ANATOMICAL CHARACTERS.**—It rarely happens that the endocardium is seen in the early period of inflammation, when this membrane merely presents bright redness, usually with distinct points of increased vascularization. Soon it becomes less smooth than normal, swollen, and clouded; diminishes in consistence; and loses its polish. Numerous young cells form in the sub-epithelial tissue, causing it to become thickened; while little vascular villi or granulations also project on the surface. In some cases the endocardium assumes the appearance of a soft, velvety membrane. Fibrinous vegetations are soon formed, which are derived directly from the blood, its fibrin being deposited on the inflamed membrane, either in strata or in masses of considerable size. There is also exudation into the substance of the valves, the free edges of which are much thickened, especially those parts which are exposed to considerable friction and irritation, and the fibrinous vegetations are chiefly deposited on the surface which is opposed to the current of the circulation. Only the left cavities of the heart are usually involved

in endocarditis, the orifices and the membrane in their vicinity, with the valves and their appendages, being especially affected. During intra-uterine life the disease chiefly attacks the right side of the organ.

As occasional consequences of acute endocarditis there may be observed fissuring of the membrane; actual ulceration; formation of pus in the deeper layers, which ultimately reaches the surface; perforation, rupture, or extensive destruction of a valve; rupture of one or more chordæ tendineæ, the free ends of which may float in the blood, and lead to the deposit of vegetations; or the rapid formation of an aneurism of the heart. *Ulcerative endocarditis* is a destructive form of the disease, in which the valves present on their surfaces or borders eroded or ulcerated spots or patches, irregular, covered with granular material, and fringed by vegetations. In the granular material organisms have been found. This form of endocarditis may lead to any of the destructive effects just mentioned. Other organs are affected at the same time; and organisms have been found in the capillaries.

Emboli are very liable to be detached from the fibrinous deposits in endocarditis, and evidences of these may be discovered in distant organs; or some of the products of inflammation may be conveyed into the circulation, and give rise to septicæmia.

If the inflammatory process subsides, the young tissue develops into an imperfect fibrous structure, and proliferation with subsequent organization may go on for some time, thus leading to most serious permanent organic changes, fatty and calcareous degeneration often finally occurring, which increase the damage. This is one of the conditions known as *chronic endocarditis*, but the affection may be chronic from the outset, there being a slow growth of new tissue, which tends to develop into a fibroid material. The ultimate changes are similar in both cases, and the chief morbid conditions resulting therefrom are as follows:—1. Thickening, induration, and puckering of some part of the general tract of the endocardium. 2. Thickening of the valves, with opacity, rigidity, and more or less shrinking, by which they may be greatly narrowed and rendered incompetent. 3. Adhesion of the tongues of a valve to each other, or to the walls of the heart. 4. Thickening, induration, and contraction of the chordæ tendineæ or muscoli papillares. 5. Narrowing of the orifices, usually accompanied with irregularity, roughness, and hardening. 6. Formation of firm warty growths, either sessile or pedunculated.

When atheroma and calcification take place, the structures present the usual characters indicative of these forms of degeneration.

**SYMPTOMS.**—Many cases of endocarditis are only revealed by *physical examination*. *Local* symptoms are always very indefinite, there being little or no pain or tenderness, but palpitation is frequently observed. The characters of the pulse have been variously stated by different observers; at first it is usually frequent, full, and excited; afterwards it may become feeble, small, and irregular in force and rhythm, but numerous circumstances influence it. Increased pyrexia may accompany endocarditis, the fever often tending to assume an adynamic type. The chief symptoms which are liable to arise in the course of the disease, however, are those dependent upon:—1. Interference with the circulation at one or more of the orifices. 2. Formation of extensive clots in the heart, embarrassing its action, and causing obstruction to the flow of blood, indicated by extremely frequent and irregular cardiac

action and pulse; tendency to syncope; great dyspnoea, amounting to orthopnoea in paroxysms, attended with extreme anxiety, and followed by asphyxial symptoms; and severe disturbance of the nervous system. 3. The convection of emboli to the spleen, kidneys, brain, and other organs; or of deleterious inflammatory products, the latter giving rise to symptoms of septicæmia.

*Ulcerative endocarditis* is attended with grave symptoms, but they are general rather than local, or associated with other organs than the heart. At the outset there is usually a sudden rigor. Subsequently the symptoms may be merely those of high fever, often assuming a typhoid character, with marked symptoms associated with the stomach and intestines, and enlarged spleen. Or they may be indicative of septicæmia, with high irregular fever, jaundice, vomiting and diarrhoea, and albuminuria. The termination is said to be always fatal.

PHYSICAL SIGNS.—The only positive signs which may be associated with endocarditis are those indicating excited action of the heart; some derangement of an orifice or extensive coagulation of blood. 1. The *impulse* is often forcible and increased in area; if coagulation takes place, it tends to become irregular in rhythm and force. 2. *Increased dullness*, especially towards the right, may arise from stagnation and clotting of blood in the cavities of the heart. 3. The *sounds* are often altered in character, but this is not to be relied upon. 4. The great sign of endocarditis is the presence of one or more *endocardial murmurs*, but it must be remembered that these may have previously existed. Different observers have given different statements as to the valvular lesions most frequently present. In my own experience *mitral regurgitation* has been decidedly the most common condition in acute endocarditis, but this may in some cases result from irregular action of the muscoli papillares. *Aortic obstruction* is not uncommon. *Pulmonary obstruction* murmur may be observed as the result of coagulation in the right cavities; but on the left side clotting of blood sometimes interferes with the production of a murmur.

DIAGNOSIS.—Not only must endocarditis be distinguished from pericarditis, but it must be remembered that the symptoms arising in its course render it liable to be mistaken for certain low fevers, and it is particularly difficult to diagnose the ulcerative form. Physical examination should be made at frequent intervals in those diseases in which it is apt to supervene, so that the morbid condition may be detected in its earliest development.

PROGNOSIS.—There are always immediate dangers in connection with acute endocarditis, which have been sufficiently indicated. The remote prognosis will depend on the permanent organic changes which the disease originates; and on the orifice which is affected.

TREATMENT.—The treatment of endocarditis is mainly that of the disease in the course of which it occurs, and no direct remedies are known for this complaint. Such measures as bleeding, mercurialization, and the local application of cold are decidedly to be deprecated. As a rule *stimulants* are required, in some cases in large quantities, along with abundant nutriment. Digitalis is indicated, should the heart's action be failing. Should there be signs of obstruction from coagulation of blood in the cavities of the heart, alkalies and carbonate of ammonia must be given freely, along with alcoholic and other stimulants. Septicæmic symptoms may call for special treatment, especially in ulcerative endocarditis, but seldom can much good be done in such cases. Other



symptoms must also be treated as they arise. It is exceedingly important, after an attack of acute endocarditis in which the valves are involved, to keep the patient in bed for some days, and afterwards at rest and under observation for a considerable time, so as to maintain the heart in a state of quiescence, and not to throw any strain on the diseased structures, thus enabling them to recover as far as possible, and limiting the growth and development of connective-tissue corpuscles. I quite agree with Dr. Fothergill that it is a serious error on the part of any practitioner to aim at making his patients sit up as soon as possible after an attack of acute rheumatism with endocarditis, or to discharge them as cured, in order to exhibit the advantages of some particular line of treatment for this disease.

## II. ACUTE MYO-CARDITIS.

**ÆTIOLOGY.**—Inflammation of the heart-substance is frequently set up in the layers contiguous to an inflamed endocardium or pericardium. Myo-carditis has been stated to have arisen independently in a few instances—due to acute rheumatism, cold, injury, or other causes, either as a diffuse or localized inflammation, the latter having terminated in abscess. Pyæmia and septicæmia frequently lead to myo-carditis, with formation of abscesses.

**ANATOMICAL CHARACTERS.**—Myo-carditis is attended with discoloration and softening of the heart-substance, this being infiltrated with a sero-sanguineous fluid, fibrinous exudation, or sometimes pus, which may collect in abscesses. The disease occasionally leads to the formation of a cardiac aneurism; or to rupture of the walls of the heart. If recovery ensues, depressed scars may be left.

**SYMPTOMS.**—The clinical signs of myo-carditis are very obscure. It tends to render the cardiac action extremely rapid, weak, and irregular, and to cause much præcordial distress, and when these are prominent features in the course of peri- or endocarditis, implication of the heart-substance may be suspected. Restlessness and anxiety are often conspicuous symptoms; with marked dyspnoea, and pallor of the face or cyanosis. The general symptoms are pyrexia of an adynamic type; with signs of blood-poisoning, and of collapse or asthenia. The fatal issue is often preceded by delirium. Sudden death is apt to occur.

**TREATMENT.**—The only hope lies in free stimulation.

## III. ACUTE PARENCHYMATOUS DEGENERATION.

It must suffice merely to call attention to the condition affecting the heart thus named, which demands recognition as liable to occur in the course of certain acute febrile affections, especially typhoid, typhus, diphtheria, and septicæmia. It is believed to be due either to the direct effects of the several morbid poisons, or to prolonged or excessive febrile temperature. By some the change is regarded as of an inflammatory nature, and hence termed “parenchymatous inflammation.” It has also been called “granular degeneration,” and “cloudy swelling.” The whole heart becomes exceedingly soft, flabby, and friable; and of a dirty greyish-red colour. The muscular fibres are seen under the microscope to be indistinct in their striæ, and dull, swollen, granular, or ruptured.

Acetic acid dissolves many of the granules, and shows a few fatty globules, as well as often an increase of pigment-particles.

Clinically, acute parenchymatous degeneration of the heart may be recognized in the course of the diseases above mentioned, by great prostration, with symptoms and physical signs of progressive cardiac weakness, culminating in failure. These phenomena indicate the necessity for energetic stimulation in treatment.

## CHAPTER XXIII.

### CHRONIC DISEASES OF THE HEART.

IN the present chapter it is proposed to give an account of the several chronic organic diseases to which the heart is liable. Their diagnosis, prognosis, and treatment will be considered in a separate chapter.

#### I. AFFECTIONS OF THE VALVES AND ORIFICES.

GENERAL ÆTIOLOGY AND PATHOLOGY.—The various conditions which may give rise to *cardiac murmurs* have been previously pointed out, and the characters of the different murmurs described. At present attention will be directed only to those cases in which there is some definite *organic* mischief, affecting the valves or their appendages, or the orifices of the heart, which interferes with the circulation, either by causing *obstruction*, or by permitting *regurgitation*. In the first place it will be well to give a general summary of the pathological modes of origin of these derangements. They may arise from:—1. *Acute endocarditis*, especially, but not solely, in connection with acute rheumatism. 2. *Chronic endocarditis* or *valvulitis*, which in many cases appears to be merely a process of fibroid degeneration, the structures implicated being very liable to undergo further degenerative changes, in the direction of atheroma and calcification. This is observed as a rule in persons advanced in years, especially in gouty subjects, or in those suffering from chronic renal disease; it may, however, occur in younger individuals who are subject to violent physical strain, in consequence of which great pressure of blood is thrown upon the aortic valves, for example, strikers, colliers, gymnasts, or boat-racers. Chronic changes in the valves may also be associated with syphilis, chronic alcoholism, and other conditions. 3. *Laceration* of a valve or of chordæ tendineæ, due to injury. 4. *Chronic myocarditis*, involving the muscoli papillares, which consequently become contracted and indurated, thus preventing the valves from closing. 5. *Atrophy of the valves*, or, as some suppose, *congenital insufficiency*, rendering them inefficient, or giving rise to so-called “reticulation” or more or less extensive perforation. 6. *Enlargement of the cavities* of the heart, involving the orifices without proportionate increase in the valves, which are therefore rendered incompetent; or altering the normal relations of the valves and their appendages to the orifices. 7. *Congenital malformations*, which are believed to result mainly from endocarditis occurring

during intra-uterine life. 8. *Fibrinous deposits* from the blood. 9. *Tumours* very rarely.

It must be mentioned that more than one orifice may be affected from the same cause; and that disease at one orifice may subsequently set up mischief in another, either by direct extension, by throwing an extra strain upon the valves and thus inducing chronic valvulitis, or by enlarging the corresponding cavity of the heart.

**SPECIAL VALVULAR DISEASES.**—Having given this general outline, the main facts connected with each orifice will now be specially considered in detail, under the following headings:—1. **Ætiology.** 2. **Anatomical characters.** 3. **Clinical phenomena**, including the signs immediately connected with the lesion; its direct effects upon the circulation, and the symptoms resulting therefrom; and its remote effects upon the heart. It may be remarked once for all at the outset, that *local symptoms* are very uncertain and unreliable. Uneasiness or pain over the cardiac region may be complained of, but is absent in the majority of cases; it is most frequent in connection with aortic disease. Palpitation and dyspnoea are common symptoms, and not uncommonly render the patient incapable of much exertion.

#### A. MITRAL REGURGITATION.

**ÆTIOLOGY.**—1. Acute endocarditis is the ordinary cause of this condition, the subsequent chronic changes increasing the primary mischief. 2. Cases occasionally are met with in which no history can be obtained of acute endocarditis, and the affection seems to have been chronic and gradual in its progress from the first, being either due to chronic inflammatory changes or to degeneration. 3. Mitral regurgitation is liable to supervene upon aortic disease, being produced in one or other of the ways already indicated. 4. It may result from mere dilatation of the left cavities, causing enlargement of the orifice, and displacing the musculi papillares. As stated in a former chapter, this last condition is believed by some authorities to be the cause of the murmur met with in chorea and in anæmia.

**ANATOMICAL CHARACTERS**—The chief morbid conditions observed in different cases of mitral regurgitation are more or less contraction and narrowing of the tongues of the valve, with irregularity, thickening, and rigidity, there being in some instances scarcely any appearance of a valve; atheroma or calcification; laceration of one of the valve-tongues; adhesion of one or more of them to the inner surface of the ventricle, or of the tongues to each other; rupture of chordæ tendineæ; shortening, thickening, induration, or adhesion of these structures, the smaller ones having often entirely disappeared; contraction and hardening of the musculi papillares; and fibrinous deposits, sometimes in considerable abundance.

**CLINICAL PHENOMENA.**—**Immediate signs.** These are:—1. A *systolic thrill* at the left apex, present only in a small proportion of cases. 2. *Mitral systolic murmur.* 3. *Intensification of the pulmonary second sound*, which is not uncommonly louder than the aortic.

**Effects upon the Circulation.**—The arterial system will be insufficiently and irregularly supplied with blood; hence the pulse tends to be small, weak, and often unequal in force and fulness, as well as in some cases irregular in rhythm. These characters are shown in the sphygmographic tracing. Two striking phenomena are sometimes observed in



connection with mitral regurgitation, namely, that the patient presents a markedly anæmic appearance; and that, although the heart may be acting violently, and the great arteries in the neck may even appear to throb, scarcely any pulsation is felt in them. Through the "back-working" of mitral regurgitation, the pulmonary circulation becomes necessarily overloaded more or less speedily, the symptoms and ultimate consequences of which have been already described. Emboli may also be carried from clots in the right heart, and give rise to pulmonary infarctions. In time the right side of the heart and the general venous system become involved, leading to venous stagnation and its consequences, often to an extreme degree.

**Effects upon the heart.**—The left auricle first becomes the seat of dilatation with hypertrophy, and afterwards the right ventricle, which is often greatly enlarged, and as a consequence tricuspid regurgitation follows. A moderate degree of hypertrophy with dilatation of the left ventricle is generally observed in course of time, and it may become very considerable. Degeneration of the heart-structure is liable to be set up in time; and the endocardial lining of the left auricle tends to become thickened, opaque, and atheromatous.

### B. MITRAL OBSTRUCTION.

**ÆTIOLOGY.**—Mitral obstructive disease is usually the result of acute endocarditis and its consequences. Sometimes it cannot be traced to this cause, and then has been supposed to be congenital, and to be due either to endocarditis occurring during foetal life, or to malformation.

**ANATOMICAL CHARACTERS.**—Mitral obstruction is often due to the orifice being more or less in a state of constriction or *stenosis*; its margins being also rough, irregular, and thickened. One or other of these conditions predominates. In other cases the tongues of the valve adhere together by their edges, so as to form a diaphragm stretching across the orifice—*diaphragmatic valve*, or more rarely a funnel-shaped opening being thus formed—*button-hole valve*. Abundant vegetations on the valves or about the orifice may cause mitral obstruction in exceptional instances.

**CLINICAL PHENOMENA.**—The phenomena of simple mitral obstruction differ from those accompanying mitral regurgitation in the following particulars:—1. *Thrill* is much more frequently felt, and is *præsystolic*. 2. The *murmur* may be either simply *præsystolic*, or may continue throughout the entire diastole up to the systole. A separate *diastolic* murmur is said to be heard occasionally, with a distinct interval between it and the *præsystolic*. 3. The second sound at the base is often markedly and persistently *reduplicated*. 4. With regard to the *pulse*, in my experience of a considerable number of cases of mitral constriction it continued regular for a long period, but some authorities regard irregularity of the pulse as an important sign of this disease. Dr. George Balfour is very strong on this point, stating that the irregularity is extreme, and he attributes it to defective nutrition of the cardiac trunk and ganglia, leading to irregular action. 5. The *left ventricle* is small, and disposed to become atrophied, rather than enlarged; but it is said that the left auricle is much more liable to become affected, and to pulsate above the fourth rib to the left of the sternum. The effects on the circulation are similar in both cases, but

they are less marked and more slowly produced in simple mitral obstruction.

In not a few instances *mitral regurgitation* and *obstruction* exist together. The interference with the circulation, and the changes in the cavities of the heart, are necessarily more readily induced under these circumstances. A double thrill may be felt. Frequently there are two distinct murmurs, but there may be but one, and it may be purely systolic, but Dr. Balfour states that the murmur is often preceded by a more or less evident thump, and that it ceases at or about the middle of the infra-axillary space. It is in these cases of double mitral disease that the pulse tends to become extremely irregular, according to my observation. Mitral disease is common in young persons.

### C. AORTIC OBSTRUCTION.

**ÆTIOLOGY.**—1. As a rule chronic valvulitis, ending in atheroma and calcification, originates aortic obstruction; the morbid process gradually advancing: hence this affection is very frequent in old people. It is this valve also which is involved in those individuals who undergo severe muscular strain. 2. Cases are not uncommonly observed, however, in which aortic obstruction can be distinctly traced to acute endocarditis associated with rheumatic fever.

**ANATOMICAL CHARACTERS.**—In most cases aortic obstruction depends upon the valves, which project inwards, and become rigid, thickened, irregular, opaque, contracted, atheromatous or calcareous, so that they cannot be pressed back by the blood, but remain constantly in the current of the circulation. Often they are covered with considerable fibrinous masses, and the opening of the artery may thus be almost completely closed. Occasionally constriction at or about the aortic orifice is the pathological condition giving rise to obstruction.

**CLINICAL PHENOMENA.**—**Immediate signs.** These are:—1. *Systolic thrill* at the right base occasionally. 2. *Aortic systolic murmur*. 3. *Feebleness or absence* of the *aortic second sound*, if there is no regurgitation, that over the pulmonary artery being unaffected; or in some cases reduplication of the second sound.

**Effects upon the circulation.**—Should the arteries be imperfectly filled, there will be pallor, with a tendency to symptoms of cerebral anæmia. The pulse is small, regular, and compressible, but is generally modified by hypertrophy or degeneration, and under the influence of the latter it may become intermittent. A sphygmographic tracing shows a difficult and very oblique ascent; a rounded summit; and the secondary waves absent or slight. There is no evidence of obstruction to the pulmonary circulation, unless the mitral orifice becomes involved, so as to permit regurgitation. It is important to notice that particles of fibrin are very liable to be detached from the valves, and carried into the circulation, thus giving rise to signs of embolism, especially in connection with the brain.

**Effects upon the heart.**—The tendency of aortic obstruction is to produce pure hypertrophy of the left ventricle, which compensates for the obstruction, so long as there is no degeneration. In course of time mitral regurgitation is apt to follow, being set up either by extension of disease from the aortic orifice; or by the forcible pressure of the blood upon the mitral valves.

## D. AORTIC REGURGITATION.

ÆTIOLOGY.—1. This disease is also often the result of chronic changes, and is especially frequent in those who undergo prolonged or severe strain. 2. In other cases it follows acute endocarditis. 3. There may be a sudden rupture or laceration of the valve, the result of extreme pressure thrown upon it. 4. Regurgitation may take place through perforations in the valve, due to atrophy or congenital insufficiency. 5. In rare instances the aortic orifice is dilated, and the valves are therefore incompetent to close it properly. 6. Degeneration at the root of the aorta may lead to the imperfect adaptation of the valves, and thus give rise to regurgitation.

ANATOMICAL CHARACTERS.—The ordinary state of the valves is that described under aortic obstruction, and they are often so shrunk, deformed, and rigid, that they permit regurgitation, as well as cause obstruction. Sometimes they adhere to the walls of the vessel; or a tongue is seen lacerated or ruptured, or having a considerable perforation in it; or there may be scarcely any remnant of the valve.

CLINICAL PHENOMENA.—It will be sufficient to point out the important characters which distinguish aortic regurgitation from obstruction.

1. Very rarely is there any *thrill*, but possibly a diastolic thrill may be felt. 2. A well-marked *diastolic murmur* is generally present. 3. The *arterial pulse* is quite characteristic, owing to the blood being forced into the vessels under unusual pressure by the enlarged left ventricle, thus causing their excessive distension; while they afterwards rapidly subside, on account of the regurgitation. This can be observed in all the arteries, and has even been seen in the vessels of the eye by the aid of the ophthalmoscope. They become visible, tortuous, and elongated with each systole of the heart, presenting a vermicular movement; the pulse having a jerky, abrupt, and hard feel, succeeded by a rapid collapse or “fall-back,” and being usually known as Corrigan’s, or the “water-hammer” pulse. The sensation has been well described as resembling “balls of blood shot under the finger.” When the arm is raised vertically, the pulse continues to exhibit the same characters, and these may even be intensified. There is no irregularity so long as the heart-tissue continues healthy. The important characters of the sphygmographic tracing are that the line of descent falls suddenly; and that the aortic wave is more or less indistinct or absolutely wanting. By observing the latter character, the degree of regurgitation may be determined. The line of ascent is usually long and vertical, with a sharp summit, but this may be square or convex if obstruction also exists. The distension-wave is raised, and the notch preceding it is exaggerated, while unusual vibratory-waves are not uncommon. Murmurs are often heard in the arteries. In course of time these vessels are very prone to become dilated and the seat of degenerative changes, owing to the repeated strain exercised upon them. 4. Pallor is in some cases a very conspicuous symptom in aortic regurgitation; or *capillary pulsation* may be observed not uncommonly, when this disease exists along with hypertrophy of the left ventricle. It may be noticed under the nails, on the cheeks, or over the forehead, especially when a portion of the surface is irritated, alternate blushing and pallor being then evident. 5. The principal morbid change developed in connection with the heart consists in hypertrophy with dilatation of



the left ventricle, which tends to become extreme. The hypertrophy is at first usually in excess of what is required for compensation, which accounts for the great distension of the arteries. Degeneration of the enlarged heart is, however, liable to set in in course of time. This was formerly attributed to deficient supply of blood to the cardiac walls, on the assumption that the supply was sent into the coronary arteries by the aortic recoil during the diastole of the ventricle; but this theory has now been completely disproved. The mitral orifice is in danger of becoming involved in cases of aortic regurgitation, as well as in those of obstruction, and the usual clinical signs of this event are likely to follow, should the patient live long enough. As a matter of experience, it is found that aortic obstruction and incompetence often exist together, and then a double sawing murmur is generally heard, while the phenomena characteristic of each disease are more or less modified.

#### E. TRICUSPID REGURGITATION.

**ÆTIOLOGY.**—Practically this condition is either associated with dilatation of the right cavities, consequent upon some obstruction to the circulation in the lungs, especially associated with emphysema and bronchitis; or it follows mitral disease, being then partly due to disease of the valves, owing to the continued extra-pressure exerted by the blood upon them.

**ANATOMICAL CHARACTERS.**—The tricuspid orifice may be simply dilated, the valves being thus rendered incompetent, but being free from disease; or the valves, especially the fixed tongue, with the chordæ tendineæ, are occasionally contracted and deformed. I have now and then observed abundant fibrinous deposit upon the ventricular surface of the tricuspid valves, when there was scarcely any organic mischief affecting their structure.

**CLINICAL PHENOMENA.**—**Immediate signs.** 1. It is stated that a *systolic thrill* has been felt in the epigastrium, but this must be extremely exceptional. 2. The characteristic *systolic murmur* is more frequently absent than present, and requires usually an experienced auscultator to detect it. In exceptional instances, however, it is well-marked.

**Effects upon the circulation.**—The general venous system suffers speedily and seriously in connection with tricuspid regurgitation, and all the symptoms resulting from overloading of the veins are liable to follow, this condition being a prominent cause of cardiac dropsy. The abdominal circulation is affected very early, on account of the deficiency of valves in the veins in this region. In addition, some important *physical signs* are thus originated, namely:—*a.* Distension and varicosity of the cervical veins, especially of the right external jugular; and sometimes of the veins over the chest. *b.* Venous pulsation in the neck, and sometimes in the inferior vena cava and hepatic vein. *c.* Filling of the external jugular vein from below, after it has been emptied by pressure. The pulmonary circulation is relieved, and thus pulmonary symptoms are often diminished when tricuspid regurgitation supervenes in cases of mitral disease.

**Effects upon the heart.**—Tricuspid regurgitation tends to increase hypertrophy of the right ventricle; and to cause enlargement of the corresponding auricle. If considerable, it may diminish the intensity of murmurs on the left side of the heart.

## F. TRICUSPID OBSTRUCTION.

This condition is of very rare occurrence. It might possibly be the consequence of endocarditis occurring during intra-uterine life; and its signs and effects are similar to those of regurgitation, except that the murmur is *præ systolic*.

## G. PULMONARY OBSTRUCTION AND REGURGITATION.

A very few observations will suffice for the affections of the pulmonary orifice. They are of rare occurrence, especially *regurgitation*. In the great majority of cases *pulmonary obstruction* is due to congenital constriction of the orifice, which may be extreme; sometimes the valves are much thickened, atheromatous, or calcareous. This disease originates a *systolic thrill* and *murmur* at the left base. The pulse is not affected, this constituting an important distinction from aortic disease. Signs of right hypertrophy and dilatation, with tricuspid regurgitation, often appear in course of time; followed by overloading of the systemic veins. *Pulmonary regurgitation* would give rise to a diastolic murmur at the left base. I have met with one instance in which marked constriction and regurgitation existed together, accompanied with a loud double murmur.

## II. ENLARGEMENT OF THE HEART.

Enlargement of the heart may be due to:—1. *Hypertrophy* of its muscular walls. 2. *Dilatation* of its cavities. In most instances there is a combination of these conditions, though in very variable degrees, but the varieties met with are sufficiently indicated in the following classification:—1. *Simple hypertrophy*. 2. *Eccentric hypertrophy*, or *hypertrophy with dilatation*, the former being in excess. 3. *Dilatation with hypertrophy*, the dilatation being the more marked. 4. *Dilatation with attenuation of the walls*, or *simple dilatation*. A form has been described as *concentric hypertrophy*, in which the cavities are contracted, but in reality this appearance is merely due to *post-mortem* contraction of the walls of a hypertrophied heart.

It will be convenient to consider hypertrophy and dilatation together, any special facts relating to either of these morbid conditions being indicated as occasion arises.

ÆTIOLOGY AND PATHOLOGY.—The numerous causes of cardiac enlargement may be ranged under certain heads.

1. *Direct obstruction, either in connection with the orifices of the heart or with the vessels, which interferes with the passage of the blood.* Cardiac obstruction is usually seated at the aortic or mitral orifice, very rarely at the pulmonary or tricuspid opening. The *aorta* may be obstructed from extensive atheroma or calcification; aneurism; congenital constriction or coarctation; or external pressure upon the vessel by a neighbouring aneurism or other tumour. In connection with the *general circulation* the chief conditions giving rise to cardiac enlargement are extensive atheroma and calcification of the arteries; changes in the arterioles and capillaries accompanying chronic renal disease; and alterations in the calibre of the small vessels associated with exophthalmic goitre. In the *pulmonary circulation* obstruction may

arise from congenital constriction of, or external pressure upon, the pulmonary artery; chronic pulmonary diseases, especially chronic bronchitis with emphysema, extensive pleuritic adhesions with retracted side, and interstitial pneumonia; or atheroma of the pulmonary vessels.

Obstruction tends more especially to lead to hypertrophy, but if it is brought about suddenly, a primary dilatation ensues; when, however, the obstruction is gradual in its progress, the hypertrophy is often of the most pure type.

2. *Distension of the walls of the heart during diastole, under increased pressure.* This is a most important cause of cardiac enlargement, being chiefly exemplified by the effects of aortic and mitral regurgitation, and to a less degree by those of tricuspid regurgitation. In these conditions there are two currents of blood entering the cavity into which regurgitation takes place, often under excessive pressure. At first dilatation is produced, but in most cases hypertrophy is soon super-added, the relative proportions depending upon various circumstances. The heart may ultimately assume enormous dimensions.

3. *Constrained action of the heart, in consequence of which the contraction of this organ is impeded, and it has to work under physical disadvantages.* Displacement of the heart from any cause, but especially from pleuritic effusion; interference with its action in consequence of a deformed thorax; and pericardial agglutination, are the important causes of enlargement coming under this head, and they tend chiefly to develop hypertrophy.

4. It is probable that mere *excessive cardiac action*, such as is observed in habitual nervous palpitation, or even in connection with dyspepsia, may induce hypertrophy. Some authorities would explain this result by affirming that an obstacle is set up in the arterial circulation, owing to contraction of the muscular coat of the vessels, and that this leads to compensatory hypertrophy.

5. It has been stated that permanent enlargement may follow the dilatation which frequently results from some *temporary loss of resisting power in the walls of the heart*, such as is associated with cardiac softening in low fevers; myo-carditis accompanying peri- or endocarditis; anæmia; or mere nervous debility and want of tone, due to excessive smoking or venery, and various other causes. After recovery a compensatory hypertrophy is said to be set up, and Fothergill is inclined to believe that this may lead to a reduction of the ventricular cavity to its original and normal dimensions.

6. I have deemed it best to notice separately that important class of cases, in which cardiac enlargement is the result of *repeated violent effort, especially with the arms*, such as is carried on in connection with certain laborious occupations (hammermen, colliers, &c.), gymnastic exercises, rowing, or climbing mountains. The enlargement is principally due to the obstruction to the circulation which is caused by the muscles crossing the arteries, the former also, owing to their rigid condition, opposing the passage of blood through their own vessels; and, after a time, to the aortic disease which is originated. The excessive action of the heart which is excited from time to time must not, however, be overlooked as probably aiding in bringing about the morbid condition. Enlargement of the right cavities is very liable to be developed in runners, swimmers, divers, and others who tax their wind unduly from time to time.



7. A *plethoric state of the system*, resulting from over-eating, especially of nitrogenized food, and abuse of alcohol, has been stated to cause hypertrophy of the heart, but this is probably associated with lithiasis.

8. Cases of hypertrophied heart now and then come under observation in which no cause can be traced, and then the hypertrophy is presumed to be *idiopathic* and *primary*.

With regard to *dilatation*, it is necessary to mention further, that this condition is more liable to supervene, and is more marked in those conditions which give rise to great internal pressure on the cardiac walls during diastole; when obstruction arises rapidly; and when the walls of the heart are deficient in resisting power from any cause, as after an acute or long-continued chronic illness, or when they are the seat of congestion, serous infiltration, inflammation, or various degenerative changes, especially fatty and fibroid.

Before proceeding to the consideration of the morbid appearances and clinical phenomena associated with cardiac enlargements, it is needful to remark that these are materially influenced by certain important circumstances, prominent among which may be mentioned:—1. The nature of the enlargement, whether this is due to hypertrophy, dilatation, or to both conditions; and the relative degrees in which they are combined. 2. The part of the heart affected. 3. The state of the cardiac walls. 4. The condition of the valves and orifices. 5. The presence of pericardial adhesions.

ANATOMICAL CHARACTERS.—The important alterations produced in the heart by hypertrophy and dilatation may be indicated as follows:—

1. *Increase in bulk*, this being mainly in proportion to the dilatation. 2. *Increase in weight*, which is due to, and therefore in the ratio of, the hypertrophy. The enlargement and excess in weight vary greatly in degree, the heart being sometimes three or four times heavier than normal, and enormously increased in dimensions. It is then called the *cor bovinum vel taurinum*. 3. *Change in shape*. In general dilated hypertrophy the heart tends to become more or less globular, the apex being rounded or obliterated. If the left cavities are alone involved, and especially merely hypertrophied, the heart is elongated and more conical, the apex of the left ventricle extending downwards some distance beyond the right. When the right side is solely affected, there is a tendency to roundness of outline and increase in breadth, the right ventricle lying forwards, so as to overlap the left, and to form the apex. 4. *Alteration in position and axis*. As a rule the heart is lowered, and its apex is displaced to the left, while the right border becomes more horizontal, the last character being especially observed in enlargement of the right side, which may also extend the heart's limits in an upward direction. 5. *Changes in the thickness of the walls, and in the size and shape of the cavities*. There are generally obvious alterations in these respects, but they vary considerably, according to both the absolute and relative amount of hypertrophy and dilatation. The walls of the left ventricle may measure from  $1\frac{1}{2}$  to 2 inches in thickness; those of the right from 1 to  $1\frac{1}{3}$  inch. The septum is usually involved, and tends to bulge towards that cavity which is least implicated. It must be remembered that there may be considerable hypertrophy with little or no thickening of the walls, because it is accompanied with much dilatation. In simple dilatation the walls of an auricle may become so thin as to consist of scarcely anything but pericardium and endocardium, and to be almost transparent. 6. *Physical characters of the heart*—

*tissue.* In hypertrophy, provided degeneration has not set in, the cardiac walls appear either of normal colour or of an unusually bright-red tint, and, as it were, more healthy and robust than normal, while the tissue feels very firm and resistant. Fatty degeneration may, however, give rise to various tints, as well as to diminution in consistence. The heart generally feels soft and flabby in proportion to the degree of dilatation. 7. *Structural changes.* In the true form of cardiac hypertrophy now under consideration the *muscular tissue* is increased. It has been held that the previously-existing fibres become enlarged and lengthened; but it is far more probable that there is a new formation, the fibres being augmented in number, and arranged more closely together. Fatty degeneration frequently follows hypertrophy, and it has been supposed that the recently-formed fibres are more liable to undergo this change. The coronary vessels become enlarged, and some observers affirm that the nerves and nerve-ganglia are also increased in size; others believe that there is only a hyperplasia of the connective tissue associated with these structures. The valves of the heart may become hypertrophied in the same ratio as the muscular tissue.

According to the cause of the morbid changes, enlargement of the heart may be general; limited to the left, or less commonly to the right side; to one cavity, especially a ventricle; or even to particular portions of a cavity. As a general statement it may be affirmed that the left side of the heart is more liable to hypertrophy; the right to dilatation. The auricles are probably never solely hypertrophied, being always dilated as well.

**SYMPTOMS.**—It is for many and obvious reasons difficult to indicate precisely what symptoms are directly due to hypertrophy and dilatation in any particular case, and on this part of the subject it must suffice to state the main general facts.

1. *Pure hypertrophy*, provided it is strictly compensatory and no more, may be unattended with any symptoms whatever.

2. In many cases, however, the hypertrophy is *excessive*, and hence gives rise to sensations of the increased cardiac action, both in the heart and in the arteries; as well as to the signs of active congestion of the systemic circulation, especially that of the brain, or of the pulmonary circulation, or of both, according as the hypertrophy is left, right, or general; these symptoms being aggravated by whatever tends to excite the heart, such as a little exertion. The undue distension of, and strain upon the arteries resulting from excessive hypertrophy ultimately leads to their degeneration, and it certainly may cause rupture of the cerebral vessels, with consequent apoplexy. It is believed that the pulmonary vessels may undergo degeneration from the same cause, and that they also may finally give way.

3. If the hypertrophy is *insufficient*, or is associated with *dilatation* or *degeneration*, then the symptoms are more marked. In the first condition there is palpitation, with dyspnoea, especially after any exertion, and now and then irregularity or intermittency of the heart's action is observed. Degeneration is indicated by feebleness of the circulation; weak, irregular, and easily disturbed cardiac action; and a tendency to syncope. Dilatation will be considered in a separate paragraph.

4. In proportion to the amount of *dilatation* will the functions of the heart be disturbed, and its motor force be impaired, so that it becomes more and more difficult to carry on the circulation, which is retarded

and rendered languid, and thus the mass of the blood is insufficiently aërated, while the capillaries and veins are overloaded, and the arteries are imperfectly filled. Most uncomfortable sensations are often experienced over the cardiac region, which may amount to intense anginal pains. Palpitation, irregularity, or intermittency are either constantly present, or are liable to be brought on by slight causes, especially by exertion or flatulence. Dyspnœa is also persistent to a greater or less degree, being easily intensified, and often amounting to orthopnœa; while all the consequences of pulmonary congestion are very liable to arise. The symptoms indicative of general venous congestion are present to their fullest extent, when there is much dilatation of the right cavities of the heart. It is important to notice that while in hypertrophy the urine is unaltered, in proportion to the dilatation does it become more scanty and concentrated, and it then usually contains albumin, which may amount to one-eighth or one sixth of its bulk.

PHYSICAL SIGNS.—In the following description an attempt is made to indicate the chief variations in the *physical signs* which may be met with in the different forms of cardiac enlargement.

1. *Bulging* over the cardiac region is often observed, being in proportion to the degree of hypertrophy; to its duration; and to the youth of the patient. Its seat and extent depend upon those of the hypertrophy. The intercostal spaces may be widened, but are not protruded. Dilatation does not cause bulging.

2. The *impulse* is much altered. In hypertrophy it is usually displaced downwards and towards the left, sometimes reaching to the seventh or eighth rib, and three inches or more to the left of the nipple; somewhat increased in area, though well-defined and limited; forcible, in some instances being extremely powerful; slow, impulsive, heaving or pushing in a downward and forward direction; and regular. Dilatation tends to enlarge the impulse transversely, especially towards the right, but does not materially lower it; it becomes extensive, diffused, and ill-defined; liable to change its place with different beats of the heart; more or less feeble, being sometimes seen when not felt, or perceptible neither to touch nor sight; of jerking or slapping quality, or occasionally almost undulatory; unequal in force, and irregular in rhythm or even intermittent; sometimes double, or with a diastolic impulse. It will be readily understood that, according to the degree in which hypertrophy and dilatation are combined, the impulse will partake more of the characters significant of one or other of these conditions. Further, the part of the heart involved will influence it. When the right side is affected, the chief impulse lies behind and to the right of the sternum and ensiform cartilage, or in the epigastrium, and it appears to be superficial; while the true apex-beat may be concealed by the dilated right ventricle. Hypertrophy of the left auricle causes an auricular pulsation above the fourth rib to the left of the sternum. Valvular diseases and fatty degeneration frequently affect the impulse associated with an enlarged heart.

3. *Cardiac dulness*. In all forms of cardiac enlargement the area of dulness necessarily tends to be increased, but this may be concealed by emphysematous lung. It is important to notice in what directions any increase takes place, as well as the shape of the dulness. Hypertrophy generally enlarges it downwards and to the left, and causes it to assume a vertically-elongated form. Dilatation extends it transversely, especially towards the right, rendering it somewhat square or circular, but



does not lower it much. General hypertrophy with dilatation gives rise to the greatest enlargement, both laterally and downwards, the shape being more or less square. It is said that the dulness of hypertrophy is more marked than that of dilatation, and that the sense of resistance is greater, but these characters are by no means reliable. Enlargement of one or other side of the heart will cause the dulness to increase in a corresponding direction; while localized enlargement will give rise to localized dulness.

4. *Cardiac sounds.* In hypertrophy the first sound at the apex becomes obscure, muffled, low-pitched, and somewhat prolonged, the muscular element being in excess. In some cases there can scarcely be said to be a real sound, but rather a sensation conveyed to the head through the stethoscope; and occasionally a sound as of the heart knocking against the chest-wall is heard. At the base the first sound may be much clearer and more valvular. The second sound is often well-accentuated at the base, so as to resemble a first sound. In dilatation the sounds tend to be feeble, but clear, short, sharp, and valvular. The first sound becomes weaker towards the base, but the second may be well-accentuated in this situation. A peculiar sensation is described in simple dilatation, which is communicated through the stethoscope, as of a diffused tumble of the heart against the chest-walls with rolling over, followed by a pause, compared to "the sudden halt of strikers on an anvil" (Richardson); or "to a horse changing its feet while cantering" (Fothergill). Combined hypertrophy and dilatation cause the first sound to become extremely loud, full, prolonged, and accentuated, so as to be heard very extensively; if the valves are also hypertrophied, this sound may have a clanging quality. The sounds will be unusually plain towards the right or left, if either side of the heart is particularly affected; and right hypertrophy may be attended with increased accentuation of the pulmonary second sound. Reduplication of the sounds is common in enlargement of the heart.

5. *Murmurs* occasionally result from enlargement of the cardiac cavities, as already explained. Hypertrophy with dilatation will intensify murmurs due to valvular diseases; and the latter will necessarily modify the sounds above described. Simple dilatation tends to weaken murmurs.

6. An enlarged heart may cause *displacement of neighbouring structures*. The lungs, especially the left, may be pressed upon, deficient resonance and feeble respiration at the base being thus induced. The diaphragm, liver, and stomach may also be depressed.

7. *The pulse.* In hypertrophy involving the left ventricle the larger arteries are generally seen to throb more or less violently, and sometimes the smaller vessels also. The pulse is disposed to be less frequent, slow and prolonged, full, tense, powerful, heaving, incompressible, and regular, having the characters known as constituting the *hammering* pulse. A sphygmographic curve presents a more or less square summit. In proportion to the amount of dilatation the pulse tends to become more feeble, small, compressible, lagging, and irregular or intermittent. When the right side is alone involved the radial pulse is not affected, or only to a less degree, and this may be of assistance in diagnosis. The pulse is often influenced by valvular diseases, changes in the vessels themselves, and other causes upon which the cardiac enlargement may depend, or with which it may be associated.

## III. ATROPHY OF THE HEART.

**ÆTIOLOGY.**—Atrophy of the heart may be met with under the following circumstances:—1. As a *congenital condition*, especially in females. 2. In connection with *general wasting* from old age, starvation, low fevers, phthisis, cancer, and other affections inducing marasmus. 3. From *pressure upon the heart* by pericardial agglutinations or effusion; or excessive accumulation of fat. This cause acts partly by interfering with the supply of blood through the coronary vessels. 4. As the result of *disease or obstruction of the coronary arteries*, the heart being on this account imperfectly nourished; atrophy is then generally accompanied with degenerative changes.

**ANATOMICAL CHARACTERS.**—Diminution in weight is the characteristic feature of cardiac atrophy, and the heart may be reduced in weight to  $3\frac{1}{4}$  ozs. or even less. As a rule the heart is small, and its cavities are contracted, the shape being normal. An *eccentric* form is described, however, in which there is dilatation as well as atrophy. The muscular tissue is usually wanting in tone, and fatty degeneration is not uncommon.

**SYMPTOMS.**—Feebleness of the circulation is the only symptom which can be attributed directly to atrophy. When it follows pressure upon the heart, or interference with its supply of blood, severe symptoms are often present, such as palpitation, dyspnoea, and general venous congestion; but these are not the immediate consequences of the atrophy. The *physical signs* are:—1. A feeble and limited apex-beat, which may be raised. 2. Diminished area of dulness. 3. Weak or sometimes almost extinct sounds. 4. Pulse small, feeble, but regular.

## IV. FATTY DISEASES OF THE HEART.

There are two distinct pathological conditions in connection with the heart to which the term *fatty disease* is applied, each requiring separate consideration.

## 1. Fatty Infiltration.

**ÆTIOLOGY.**—Fatty infiltration is observed:—1. As a part of *general obesity*, especially in elderly persons. 2. In some individuals who suffer from cancer, phthisis, and other *wasting affections*. 3. In connection with *chronic alcoholism*.

**ANATOMICAL CHARACTERS.**—There is an infiltration of the connective-tissue cells around and in the substance of the heart with fat, leading to a kind of *fatty hypertrophy*. This commences under the pericardium, but the fat penetrates inwards between the muscular fibres, causing their degeneration and absorption, so that finally the affected portions of the cardiac walls may be almost or entirely composed of adipose tissue. The ventricles are chiefly affected, especially the right, and there is a particular tendency to the accumulation of fat along the sulci, and about the base and apex. The tissue is necessarily pale, soft, flabby, and lacerable.

**SYMPTOMS.**—Fatty infiltration may generally be suspected when it exists, but in many cases it cannot be made out by positive signs. If considerable in amount, it is liable to be accompanied with uncomfortable sensations in the cardiac region; palpitation on exertion; shortness

of breath ; and a weak and languid circulation, leading to incapacity for any effort, chilliness of the extremities, and a tendency to giddiness or faintness. *Physical examination* only reveals a feeble impulse and sounds ; and a weak compressible pulse. Often, however, there is so much fat over the chest as to render the local signs mentioned quite unreliable.

## 2. Fatty Degeneration or Metamorphosis.

**ÆTIOLOGY.**—The pathological modes of origin of fatty degeneration of the cardiac walls are as follows :—1. In the large majority of cases it results from *mal-nutrition*, owing to some interference with the supply of blood through the *coronary arteries*. This may arise from atheroma or calcification of the vessels themselves ; embolic obstruction ; or external compression, especially by pericardial thickening. A hypertrophied or dilated heart is very liable to degenerate, chiefly in consequence of inadequate blood-supply. 2. Cardiac degeneration is sometimes a manifestation of the *fatty diathesis*, similar changes being observed in the kidneys, lungs, vessels, cells of the cornea, and other structures. These changes may be set up without any evident cause ; or in connection with senile decay, alcoholism, gout, and lowering diseases, such as phthisis or cancer. Most authorities regard them as being the result of some unhealthy condition of the blood ; but it has been suggested that the trophic nerves of the heart are at fault. Some are of opinion that the heart and other structures may become fatty as a consequence of renal disease, which renders the blood impure. 3. More or less fatty degeneration is associated with *fatty infiltration*, and it may follow *myo-carditis*. 4. The heart has been found to have undergone fatty degeneration in cases of *poisoning by phosphorus*, as well as by phosphoric and several other acids. 5. It has been suggested that *disease of the cardiac ganglia and nerves* may lead to fatty degeneration.

**Predisposing causes.**—The most important of these are age, fatty degeneration of the heart being very uncommon in the young, and increasing in frequency after middle life to about the sixty-third year (Watson), after which it gradually becomes less common ; the male sex ; sedentary and indolent habits, especially when combined with over-eating and drinking, this disease being hence believed to be more common among the higher classes, and in those whose occupations lead to the above habits, such as publicans or butlers ; and the presence of gout or Bright's disease. Neither general obesity nor the opposite condition seems to have any influence in the development of the disease.

Allusion may here be made to the condition which has been specially named the "gouty heart." It is really merely hypertrophy followed by fatty degeneration of the cardiac walls, associated with changes in the vessels and in the kidneys, occurring in connection with lithæmia. Chronic changes in the valves may also be set up.

**ANATOMICAL CHARACTERS.**—Fatty metamorphosis may be observed in a heart of normal size, or in one enlarged or atrophied. The ventricles are by far most frequently affected, especially the left, and the change may be seen over a considerable extent, or be limited to certain parts, if due to localized vascular obstruction. It may commence primarily at either surface, or deep in the walls.

Certain alterations in *physical characters* are marked if the degeneration is advanced. The colour is paler than normal and dull, either brownish-red, pale brown, or presenting various "faded-leaf" tints, being some-



times actually yellow. These colours may be seen throughout, or merely in streaks. The consistence is diminished, the tissue tearing and breaking down readily under pressure, and the cardiac walls occasionally resembling mere "wet brown paper." There may be a greasy feel, oil being also yielded on pressure, or to the knife, blotting-paper, or ether.

The *microscopic changes* are characteristic, and may be observed before there are any alterations evident to the naked eye. At first the striæ of the muscular fibres are merely rendered indistinct by the presence of fat-granules and oil-globules, being again brought into view by the action of ether. Gradually they become more and more obscured, until finally they disappear altogether, the fibres being made up entirely of fat granules. Some pathologists are of opinion that fat is formed between as well as within the fibres.

**SYMPTOMS.**—Undoubtedly fatty degeneration may exist without there being any clinical indications whatever of this condition, or only such as are of doubtful significance. Sudden death has occurred from this disease, when there had been no previous suspicion of any cardiac mischief. Cases, however, come under observation not uncommonly in which the diagnosis may be made with tolerable positiveness. As a rule the progress of the disease is very gradual and insidious. Most of the symptoms are attributable to the feeble action of the heart.

Unpleasant sensations are frequently complained of over the cardiac region, and anginal attacks are very liable to arise. Palpitation is often felt during the progress of the degeneration, not, however, directly associated with the diseased fibres, but with those which are unaffected, these being insufficient to carry on the circulation. The principal disturbances of the cardiac action observed in different combinations are infrequency, the beats being reduced to 50, 40, 30, 25, 20, or even fewer in a minute; feebleness; irregularity; and intermittency. Any exertion tends to increase the frequency, and to render the action more irregular.

The appearance of the patient may afford signs of the disease. There is not infrequently a sallow earthy tint, combined with anæmia, or with lividity about the lips and enlarged capillaries on the cheeks, of which appearances I have met with well-marked examples. Fothergill describes the skin as sometimes resembling discoloured parchment, having a greasy feel, and presenting changes in the epidermis. The tissues are generally flabby and wanting in tone. There are frequently evidences of degeneration of the vessels and other structures. Among these the *arcus senilis* has been considered of material diagnostic importance, especially when it is yellow, ill-defined, and passes into a cloudy cornea; but this is very questionable.

The patient feels weak and languid; deficient in vitality; subject to chilliness, and may be incapacitated for any exertion, which brings on shortness of breath, faintness, or actual syncope. Involuntary sighing is sometimes a prominent symptom, and also the Cheyne-Stokes respiration.

Owing to the inadequate supply of blood to the nervous centres important symptoms are liable to arise. The chief of these are habitual depression of spirits; irritability and moroseness; various sensations in the head; disturbances of vision; feebleness of intellect, with failure of memory, and inaptitude for thought; tremulousness and an unsteady gait; a tendency to sudden attacks of giddiness, which make the patient

cling to the nearest object; restless and disturbed sleep, attended with sudden startings, which may be due to a feeling of impending suffocation; and unusual sensations in the limbs. Sudden cerebral anæmia is very liable to occur, inducing syncope; apoplectiform or epileptiform attacks; or a combination of these conditions. These attacks are, however, soon recovered from as a rule, and do not leave any permanent ill-effects behind.

The digestive organs are generally out of order. A sensation of sinking at the epigastrium is often complained of. Sexual inclination and power are frequently notably deficient.

It must be borne in mind that fatty degeneration may set in in connection with a hypertrophied or dilated heart, or with valvular disease, and it will then modify the symptoms as well as the physical signs accompanying these conditions, especially adding to the difficulty in carrying on the circulation.

**PHYSICAL SIGNS.**—The only positive signs of fatty heart are the following:—1. The *impulse* is feeble or absent, but if perceptible it is well-defined. 2. The *sounds* are weak, especially the first, which may be almost inaudible, particularly at the base, where it is weaker than at the apex. The second sound may be fairly accentuated. In extreme cases no sounds can be heard. 3. The *pulse* is very feeble, small, and compressible; often infrequent, there being sometimes but one pulsation to two ventricular contractions; while it tends to be irregular, and may become hurried paroxysmally, so as to be almost uncountable from its frequency and irregularity (Walshe).

**COURSE AND TERMINATIONS.**—Patients suffering from fatty disease of the heart may go on for years, but death is to be feared at any moment if the lesion is advanced. The fatal termination may occur quite suddenly from syncope, usually after some effort; rupture of the heart, either sudden or gradual; cerebral anæmia; or gradually from asthenia, which may be attended with dropsy. This symptom, however, is often absent from first to last, even in extreme cases, and it is a question whether fatty disease alone can give rise to dropsy, although it may assist materially in its development.

## V. PECULIAR DEGENERATIONS AND NEW FORMATIONS IN THE WALLS OF THE HEART.

In addition to the fatty degeneration just described, it is requisite to mention the following:—1. *Softening* of the tissue of the heart in connection with low febrile conditions, especially typhus, typhoid, small-pox, scarlatina, and septicæmia from any cause. A form of simple softening has also been described, chronic in its course. 2. *Brown atrophy* of the heart. 3. *Fibroid infiltration* or *degeneration*, or so-called *cirrhosis*. This is localized, especially in the muscoli papillares, but it may form scar-like patches in the walls. In some cases it results from inflammation; but in others it seems to be a gradual change, from proliferation of an imperfect fibroid tissue; or some believe that there is an actual infiltration of material between the muscular fibres. 4. *Calcification*. 5. *Syphilitic growths*. 6. *Albuminoid degeneration*. 7. *Cancer*, which is extremely rare, being usually medullary and nodular. 8. *Tubercle*, also very rare. 9. *Parasitic formations*, namely, the cysticercous cellulosus, and the echinococcus hominis.

## VI. CARDIAC ANEURISM.

Cardiac aneurism signifies a localized dilatation of the walls of the heart. It may involve the entire thickness, or the endocardium and contiguous muscular strata may be destroyed. The size and form of the aneurism vary much, but there are the two types of general and equable dilatation of a portion of the parietes; and the sacculated variety, the latter opening into the heart by a wide or narrow orifice. More or less stratified fibrin or coagulated blood is usually found in the sac, and it may thus be completely obliterated, and the aneurism cured. The left ventricle is almost invariably affected, and more than one aneurism may be present.

Cardiac aneurism is almost always the consequence of some previous structural change in the ventricular walls, especially fatty or fibroid degeneration; inflammation; softening from any cause; rarely ulceration or rupture of the endocardium; or hæmorrhage into the muscular structure. As a rule it is formed gradually, but may be developed suddenly from violent strain. Fibroid and other degenerative changes are likely to be increased, or to be subsequently set up at the seat of an aneurism of the heart.

SYMPTOMS.—There are no reliable symptoms or signs of aneurism of the heart. Sometimes a localized pulsating prominence is observed, over which a single or double murmur may be heard. Hypertrophy and dilatation are developed in course of time. Death may take place suddenly from rupture of the aneurism.

## VII. RUPTURE OF THE HEART.

ÆTIOLOGY.—This rare lesion may be considered in this chapter, as the rupture is generally the result of some chronic structural change in the cardiac walls, and probably even in traumatic cases they are never quite healthy. The more important morbid conditions which have been observed are fatty disease, especially degeneration; great dilatation; cardiac aneurism; abscess or gangrene; ulcerative or other form of destruction of the endocardium; hæmorrhage into the walls; calcification; and parasitic formations. Rupture of the heart may occur in connection with aortic aneurism or coarctation, but then its walls are probably always diseased as well. It almost invariably results from some exciting cause, being rarely spontaneous; while it is by far most frequent in males and old persons.

ANATOMICAL CHARACTERS.—The size, shape, and other characters of a rupture of the heart vary considerably. On the whole it is much more frequent in the left ventricle, but traumatic rupture is more common on the right side. The direction of the laceration is generally parallel to the chief fibres of the heart.

SYMPTOMS.—These vary according to the mode in which the rupture takes place, and its dimensions. Death may be instantaneous, or very rapid, after sudden insensibility preceded by a shriek. If this does not happen, the important symptoms are sudden extreme pain in the cardiac region; a sense of great oppression and dyspnoea; signs of intense shock and collapse; and indications of grave interference with the cardiac action. Patients occasionally rally, and there may be repeated attacks, supposed to indicate rupture of successive layers of the heart's fibres. It is even stated that recovery may take place.



## CHAPTER XXIV.

## GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT OF CHRONIC CARDIAC AFFECTIONS.

## I. DIAGNOSIS.

IN making a diagnosis with regard to chronic affections of the heart it is necessary to determine:—1. Whether there is any actual organic mischief, or merely functional disturbance, which gives rise to symptoms associated with this organ. 2. The nature, exact seat, and extent of any organic disease present, the main conditions to be borne in mind being:—*a.* Diseases of the valves and orifices. *b.* Alterations in the size or capacity of the heart. *c.* Changes in its walls. *d.* Interference with its supply of blood. *e.* Pericardial effusion or adhesion. It must be remembered that these lesions are often variously combined, and an endeavour should be made to determine the precise state of the structures in every particular just mentioned. 3. The pathological cause of any existing lesion, if this can be discovered.

A separate account of the diagnosis of each disease would involve unnecessary repetition, and it will be sufficient to indicate the data on which this should be founded, these being:—1. The *previous history* of the patient, special inquiry being made with regard to *acute rheumatism* and *violent exertion*. It may also be of some help to note whether there is any family predisposition to cardiac disease. 2. The *age, sex, and general condition*, particularly observing whether there are signs of degeneration. 3. The *symptoms* present, especially noting any disturbance of the circulation, and the phenomena resulting therefrom. 4. The *physical signs* discovered on examination. Physical examination is the only positive and reliable means by which cardiac diseases can be diagnosed, and daily experience enforces the importance of investigating the state of the heart in any case which comes under observation for the first time, and especially when examining for life-insurance. The points to be attended to in this examination are:—(i.) Whether there is any change in the shape or size of the chest over the cardiac region. (ii.) The characters of the impulse in every particular. (iii.) Whether any thrill or pericardial fremitus can be felt. (iv.) The position, form, directions of increase, and extent of the cardiac dulness. (v.) The characters of the heart-sounds, these being compared over different parts of the cardiac region. (vi.) Whether any pericardial or endocardial murmur can be detected, with the characters of such murmur. It is also requisite to examine carefully the arteries and veins, making use of the sphygmograph when necessary in connection with the former, and observing whether these vessels present evidences of degeneration.

It is important to draw attention to the following facts:—1. The heart may be displaced by extrinsic conditions, thus presenting abnormal physical signs when it is not itself actually diseased; while signs of organic mischief may be modified by the state of contiguous structures. 2. Murmurs may belong to the inorganic class; or may be

merely due to roughness of the endocardium, which is unattended with any danger. 3. The bulging and dulness indicative of pericardial effusion or cardiac enlargement may be simulated by excessive temporary distension of the right cavities of the heart; aneurism of the aorta; tumour, abscess, or accumulation of fat in the mediastinum; localized pleuritic effusion; and consolidation or retraction of the anterior edges of the lungs, especially the left. 4. Organic disease, even of a serious nature, is not unfrequently unattended with any symptoms whatever; and there may be no distinctive physical signs, particularly in the early stage of degeneration. 5. Severe cardiac symptoms may be complained of, and there may even be marked objective disturbance of the heart's action, amounting to irregularity or intermittency, in connection with mere functional disorder. Much stress has been laid on certain points in making out whether local cardiac symptoms are due to organic mischief or not, namely, that mere functional disturbance is not increased by effort; is inconstant; and is usually brought on by some obvious exciting cause. My own experience would lead me to avoid placing any implicit reliance on such distinctions, except that grave disorder of the cardiac action following slight exertion may be an useful sign indicative of degeneration.

## II. PROGNOSIS.

Any organic affection of the heart should be regarded as serious, but numerous circumstances influence the prognosis, and every case has to be considered in several aspects before a satisfactory opinion on this matter can be given. It must be premised that great care should be exercised against mistaking mere functional disorder for organic disease and *vice versâ*, which might lead to a wrong prognosis in either direction, and it is therefore highly improper to attempt to found any conclusion on mere subjective symptoms associated with this organ.

The questions which have to be considered in any particular case of heart-disease are mainly these:—1. Whether there is any danger of sudden death? 2. What are the events likely to arise in the progress of the case, and the dangers to be feared? 3. What will be the probable duration? 4. Whether a cure is possible? In the following remarks an endeavour will be made to indicate the chief data upon which a correct conclusion on these questions is founded; and to state the principal facts which experience has established.

1. The prognosis must necessarily be guided by the **nature, seat, and extent** of the disease or diseases present, the knowledge of these points being of course founded on a satisfactory physical examination. Instances are met with occasionally in which a murmur arises from mere roughness of the endocardium; in such cases there is not much danger, except that the mischief might spread to the orifices and valves or their appendages. Any organic affection in connection with either orifice, inducing obstruction or regurgitation, is decidedly serious, but the dangers are of a very different character at the different orifices, and depend further upon the cause of the lesion, and the exact conditions present. In estimating the probable evils, moreover, the effects of the various lesions upon the circulation must be borne in mind, as well as the secondary changes which they are likely to originate in the heart. With regard to *sudden death*, *aortic regurgitation* is the only form of valvular

disease in which this event may be anticipated with any probability, but it is said to have occurred in exceptional cases of *mitral regurgitation*. *Obstructive disease* in connection with the left orifices is mainly injurious by its "back-working," and by its consequent effects on the heart, lungs, and circulation. *Aortic obstruction* often lasts a long time without producing any particular mischief; and cases of *mitral constriction* also frequently go on for a considerable period. *Mitral disease* is more immediately dangerous on account of its effects on the lungs. *Tricuspid regurgitation* is one of the most serious affections of the orifices, on account of the distressing symptoms by which it is certain to be followed in course of time, and often very speedily, through overloading of the venous circulation; but the progress of the case is frequently slow and tedious, the patient leading a miserable existence. *Pulmonary constriction* or *regurgitation* produces the same effects, but less rapidly. It will readily be understood that extensive or double disease at an orifice increases the gravity of the prognosis. As a rule also it is worse when two or more orifices are involved; but secondary implication of an opening sometimes gives temporary relief, as in the case of tricuspid regurgitation following mitral disease, which diminishes the severity of the pulmonary symptoms.

With respect to the question whether valvular disease is ever curable, I certainly have met with cases in which marked mitral constrictive murmur has disappeared entirely in young persons; and, though complete restoration to the normal condition is probably not possible, it is not unlikely that inflammatory deposits leading to both aortic and mitral obstruction may be partly absorbed or otherwise removed in course of time.

*Hypertrophy* of the heart in the majority of cases is decidedly a preservative or compensatory lesion, and cannot under such circumstances be looked upon as of evil import. It is dangerous only when excessive, as it may then lead to rupture of vessels, especially if these are diseased, a condition which it tends itself to produce through constant over-distension: when on the right side it may be further injurious through keeping up a persistent state of active congestion of the lungs. Some authorities are of opinion that hypertrophy may subside if the cause which has induced it can be removed.

*Dilatation* is a highly dangerous condition, and in proportion to its degree and to its excess over hypertrophy does the prognosis become worse. Sudden death may occur in connection with a weak, flabby, greatly dilated heart; while dilatation always materially augments the difficulties experienced in carrying on the circulation, thus contributing to the development of dropsy and other serious symptoms.

*Degeneration* of the heart's walls, especially fatty disease, is another very grave lesion. It is when this change sets in that the prognosis becomes so much worse in cases of compensatory hypertrophy. Extensive fatty degeneration is one of the most frequent causes of sudden death from cardiac disease.

*Pericardial agglutination* adds much to the evils of other cardiac diseases, and also tends to originate changes in the heart itself. I have observed some cases of pneumonia in which this condition seemed to have considerable influence in bringing about a fatal termination.

In many cases the cardiac affections just alluded to are variously combined, and the prognosis has then to be determined from a careful consideration of the exact lesions present.



2. The existing **symptoms** will influence the prognosis considerably. Severe anginal attacks; great irregularity or intermittency of the cardiac action; or a tendency to syncope or to apoplectiform or epileptiform seizures, increase the danger of a case very materially. When the general venous circulation becomes much interfered with, and dropsy sets in, the duration is not likely to be very prolonged; it is impossible, however, to make any definite statement on this matter, as patients often linger on for a considerable time, and may even improve remarkably under appropriate treatment. It is important to notice further that acute pulmonary complications may arise, and cause very serious symptoms, at the same time materially increasing the dropsy, so that the case appears to be approaching a speedy termination; but, on the subsidence of these complications, great improvement may take place, the patient again rallying for a time, and sometimes even feeling better than before.

3. The **cause** of any cardiac organic disease may influence the prognosis, as well as the possibility of removing such cause. For instance, improvement in valvular disease can be hoped for only when it results from acute inflammation; if induced by chronic and degenerative changes, matters always tend to become worse. After a sudden injury to a valve, the lesion does not tend to increase, but usually remains stationary, whereas after valvulitis progressive changes are likely to take place, which derange more and more the functions of the structures connected with the affected orifice. As has been previously stated, some authorities regard a certain degree of hypertrophy, or even of dilatation as capable of being completely cured, if the cause can be removed.

4. The **state of other organs and structures**, especially of the lungs, kidneys, and arteries, will considerably modify the prognosis in any given case of heart disease, and hence their condition ought to be carefully investigated. If the vessels are much diseased, the muscular tissue of the heart is very likely to undergo speedy degeneration.

5. Among **general matters** affecting the prognosis are the age of the patient; the family history, as indicating a tendency to death from heart disease at any particular time; and the social position and habits of the patient. It is only in young persons that curative changes can be at all expected. Those who are so circumstanced that they are able to live quietly, without either the anxiety or the labour arising from having to provide day by day for themselves and their families, and who can procure a suitable diet, have a much better chance of length of life than those not so fortunately situated. Laborious occupations are especially hurtful. Continuance in injurious habits, such as intemperance or debauchery, will necessarily render the prognosis more unfavourable. The prognosis of cardiac disease with reference to marriage, parturition, and suckling is of much importance, and those who are interested in the subject will find valuable information in the work by Dr. Angus Macdonald on "Heart Disease during Pregnancy."

### III. TREATMENT.

Very seldom can any hope be entertained of curing a chronic cardiac affection, but undoubtedly much may be done in the way of prolonging life; averting further mischief in the heart; warding off unpleasant or

dangerous symptoms; and relieving such symptoms when they arise. After any acute affection involving the heart, the patient should be kept strictly under observation until this organ has been restored to as normal a condition as possible; while any chronic case ought to be kept constantly under medical supervision, though this does not imply that medicines must be persistently given. Different forms of heart-disease require particular modifications in their management; but it must suffice here to point out the main principles which apply to all cases more or less; and, as occasion requires, to call attention to any special treatment which needs to be commented upon in connection with particular affections.

1. **General management** is always of essential importance. A patient suffering from heart-disease should, if possible, give up any laborious employment, especially if this has evidently originated and is increasing the mischief. At the same time warning should be given against all forms of severe exercise, particularly those which involve sudden effort; and it is well to give special instructions against running or walking hurriedly, or straining at stool. In some instances complete rest should be enforced for a time, which often produces a marked improvement in the state of the heart. Many cases, however, are benefited by more or less exercise, or at all events by being in the open air during some portion of the day, and carriage driving is often useful. Many patients are able to go about their usual avocations without any harm resulting, provided these are of a satisfactory character. The question of the amount of exercise to be permitted must be determined by the actual conditions present, and the effects which follow it; it may be stated generally, that in proportion to the degree of dilatation or degeneration present is the capacity for effort diminished. These lesions, if extensive, as well as aortic regurgitation, imperatively forbid any great exertion. It is very important further to avoid all causes of mental disturbance. Anxiety and mental strain or excitement in connection with pecuniary matters, business, politics, or excessive study, as well as everything which is likely to rouse strong emotion, must be carefully shunned, and a proper amount of sleep should be habitually obtained. Warm clothing is requisite, but there must be no pressure or constriction about the chest or neck. Cold or tepid sponging of the skin is often useful if it is well borne. All injurious habits which depress the nervous energy of the heart must be prohibited, such as abuse of alcohol, tobacco, or tea, late hours, or venereal excesses; and close inquiry may be necessary with regard to various matters in order to detect such mischievous habits. Change of air to a moderately warm and rather bracing climate frequently proves beneficial.

2. It is most needful to attend to the **diet** in every particular, and to the state of the **digestive organs**. When there is degeneration of the heart a very nutritious diet is indicated, which should contain abundant protein elements, if these can be digested, but anything indigestible must be avoided. Milk and cream are exceedingly useful articles in many cases. With regard to alcoholic stimulants, no rule can be laid down, but a moderate amount is generally beneficial, and there are not unfrequently symptomatic indications calling for considerable quantities. The bowels should be kept acting regularly. Remedies for improving the tone of the stomach and relieving dyspeptic symptoms are frequently very serviceable, and this applies

especially to flatulence, which mechanically interferes with the heart's action.

3. If there is any **constitutional diathesis**, such as gout or syphilis, treatment directed against such a condition is often beneficial. One of the most essential matters in many cases of cardiac disease is to look to the state of the **blood**, and should there be any indication of anæmia, to give some preparation of iron. Indeed, independently of this condition, this drug is frequently of considerable value, especially in the form of tincture of steel. Other *tonics* are useful in many cases, such as quinine and mineral acids, strychnine or tincture of nux vomica, particularly if the heart is in a state of degeneration, or is wanting in tone.

4. Excellent observations have been carried on for several years, with the object of determining the effects of **medicinal agents** upon the heart. Digitalis has long been regarded as one of the most important drugs which affect this organ, and it demands special notice. Experiments have shown, as regards its physiological effects, that it influences not only the heart, but also the arteries. These effects will differ according to the dose of the drug, and Dr. Mitchell Bruce divides them into four stages. It must suffice to state here that in ordinary medicinal doses digitalis causes the heart to beat less frequently; the period of diastole is lengthened, so that the ventricles are well-filled; the ventricular contractions are more powerful and complete, so that the ventricles are thoroughly emptied; and the blood is driven in larger quantities and with greater force into the arteries, arterial tension being increased and maintained. "The condition is that of a perfect circulation, which *empties the veins and fills the arteries*" (Bruce).

There are considerable differences of opinion as to the cases in which digitalis is indicated, and as to its most efficient mode of administration. The following remarks may serve to convey the main practical facts bearing upon the therapeutic uses of this drug, partly founded on the statements of others, partly on personal clinical observation.

(i.) In all cases in which digitalis is given, its effects should be carefully watched, especially as regards the cardiac action; the state of the pulse; the urine; and any dropsy which may be present. When the action of the heart is rapid, irregular, ineffective, or embarrassed, the pulse being at the same time weak, the good results of the use of digitalis are seen in that it calms the heart, and makes this organ act regularly and more vigorously, often relieving unpleasant local sensations; while the pulse is simultaneously improved, becoming less frequent, stronger, fuller, and more regular. Intermittency has been considered by some as contra-indicating digitalis, but though more than usual caution is required under such circumstances, it may be given in many cases with excellent results, and Fothergill is of opinion that intermittent action sometimes indicates a necessity for increasing the dose. If it appears to induce irregularity or intermittency, with much feebleness of pulse, digitalis should be discontinued. The urine is often much increased in quantity by its use, but only if dropsy is present (Ringer). Should it become diminished, this is considered an indication for stopping the drug. Its diuretic action is presumed to be due to the force of the heart being increased, and through this the arterial tension in the kidneys, by which the flow of water out of the renal vessels is promoted; it has, however, also a direct action upon these vessels themselves. The influence of digitalis on cardiac dropsy is often most marked, but not



invariably. As signs which suggest the discontinuance of digitalis may be mentioned exaggeration of unpleasant sensations about the heart, if evidently due to the drug; tendency to faintness; noises in the head; and persistent vomiting. It has been supposed to have a cumulative action, and may thus give rise to sudden symptoms of poisoning. Its ultimate effect as a poison is to arrest the heart in diastole, and death occurs by general circulatory failure.

(ii.) Digitalis is given chiefly in the form of the tincture or infusion, some practitioners preferring one, some the other. If the remedy is required to act rapidly upon the heart, and especially to diminish dropsy, the freshly-made infusion is certainly preferable; but the tincture is very useful for continuous administration. The powder of the leaves is also recommended when it is required to keep up the action for some time, and if it cannot be taken internally, external applications of poultices of the leaves or fomentations of the infusion may act beneficially, especially in promoting the flow of urine and diminishing dropsy. Digitaline has also been used, either internally or by subcutaneous injection. It is generally advisable to begin with a small dose (3ss-3i of infusion, or  $\text{m}\text{v}-\text{x}$  of tincture three or four times daily), and gradually increase the quantity, as well as the frequency of administration, according as circumstances indicate. Digitalis is advantageously combined with other medicines, especially iron, various *tonics* and *diuretics*. It may be necessary to continue the medicine for a long period, even for years; but it has appeared to me that it is in many instances preferable to intermit its administration from time to time. In the less advanced cases it often brings about such good results that it can be left off for considerable periods, but it should be resumed as soon as any signs of disturbed cardiac action return. In very advanced cases attended with general dropsy the drug may lose its power, and the dose has often to be increased considerably in order to produce any effect, which is a bad omen.

(iii.) The cases in which digitalis is indicated or the reverse must now be noticed. Simple hypertrophy of the left ventricle only requires the drug when excessive, and when the heart is acting tumultuously; or when the hypertrophy is insufficiently compensatory. The dose should be very small, and the effects closely watched, as symptoms of poisoning may speedily arise in these cases. In proportion to the degree in which dilatation becomes evident, and the heart's action is consequently inefficient, does the remedy generally become more valuable, much larger doses being required and being well borne. Mitral disease and changes in the heart resulting therefrom are greatly benefited as a rule, the pulmonary and other symptoms associated with these conditions being also effectually mitigated. It is especially when there is great irregularity that digitalis proves so serviceable, and Ringer believes that the drug causes the muscoli papillares to act more regularly, thus checking regurgitation which depends upon their disturbed action. Many object to the use of digitalis when the aortic orifice is involved. I quite agree with those, however, who do not look upon this as a contra-indication, provided the state of the ventricle is such as to require it, having frequently seen marked benefit follow its administration, but these cases must be closely watched. The conditions due to enlargement of the right heart with tricuspid regurgitation, when this lesion exists alone, as the result of pulmonary disease, are not improved by digitalis unless there is irregularity in the cardiac action,

and the drug may even do harm; when these morbid changes follow mitral disease, however, much good may be effected by its use.

Fatty degeneration is also looked upon by many as contra-indicating the use of digitalis; but, with due precautions, I am convinced that it may be given with undoubted benefit when this condition is present, should it be called for; and it then probably acts by aiding the contraction of those fibres which still remain healthy. Extensive atheroma has likewise been regarded as forbidding the employment of this drug, and it certainly ought to be used with particular care when the arteries are much affected. Bronchitic attacks associated with heart-disease may be often much relieved by the use of digitalis, should there be palpitation, irregularity, or other signs of cardiac embarrassment and want of power. Its value in functional palpitation has already been alluded to.

Numerous other remedies besides digitalis have more or less powerful effects upon the heart, and their number is being constantly added to, as the result of the investigations now carried on. Here it must suffice to mention the most important, and reference must be made to special works on Therapeutics and on Diseases of the Heart,\* for a full account of their action and uses. The chief of these drugs include aconite, or aconitia, belladonna or atropine, morphia, casca bark, strychnine, hydrocyanic acid, veratria, calabar bean or physostigmine, caffeine, scopolarium, squill, convallaria and its active principles, amyl nitrite, pilocarpine, muscarine, and paraldehyde. Some of these drugs require to be given cautiously, as they are powerful agents. On the whole I have been disappointed in convallaria, but in one case it proved of signal service in improving the action of the heart, increasing the urine, and relieving dropsy.

5. Important questions suggest themselves, as to whether any means are known, capable of restoring the heart to its normal condition, when in a state of disease; and whether it is desirable to use such means? As regards valvular lesions, it is useless to attempt to influence these by any therapeutic measures. With respect to the diminution in size of a hypertrophied heart, this is certainly not what ought to be aimed at, and it is more than doubtful whether such enlargement can be affected in the least by any known remedies; such measures towards this end as repeated local bleedings, low dieting, severe purgation, and large doses of iodide of potassium, are decidedly to be condemned, and the great object should rather be to maintain the nutrition of the heart as much as possible, and to prevent it from becoming dilated or undergoing degeneration. There are no direct means of influencing dilatation, except by improving the tone and vigour of the heart by administering good food, *tonics*, and digitalis. It is quite possible that the nutrition of a fatty heart may be improved in some instances by nourishing diet, *tonics*, and cod-liver oil.

6. Various **symptoms** are liable to arise in the course of a case of heart-disease, demanding measures for their relief. Those more immediately connected with the organ itself are pain and other unusual sensations; palpitation; angina pectoris; and faintness or syncope. Abnormal sensations are often much relieved by wearing a belladonna plaster, and many patients are never comfortable except when they have one applied. Sometimes belladonna liniment is very useful. The treatment of the other symptoms has already been pointed out. With

\* An important treatise on "Diseases of the Heart and Thoracic Aorta," by Dr. Byrom Bramwell, has been recently published.

regard to palpitation accompanied with dyspnoea, this is in some instances much quieted by the subcutaneous injection of a very small quantity (gr.  $\frac{1}{12}$  to  $\frac{1}{6}$ ) of morphia, which may in some cases be usefully combined with atropine or digitaline. Aconite in minute doses may also prove of much benefit. Pulmonary symptoms must be treated by the usual remedies, but they are often greatly relieved by digitalis. Needless cough should decidedly be subdued, though it is frequently necessary to promote expectoration. Cardiac dyspnoea may in many cases be relieved by digitalis, or it may require various *sedatives* and *antispasmodics*. Any obvious cause giving rise to this symptom, such as flatulence, should at once be got rid of, and it is generally diminished by enabling the patient to sit up in bed, thus removing any pressure on the diaphragm from below. In some cases the patient cannot remain in bed, and must be allowed to sit propped up in an arm-chair, which may be fitted with a rest upon which he can lean forward. Hæmoptysis occurring in heart-disease should not be rashly stopped, provided there is not sufficient loss of blood to injure the patient, as it may afford considerable relief.

*Local remedies* are often serviceable in the treatment of heart and lung symptoms, such as dry-cupping, hot or turpentine fomentations, and sinapisms. Some recommend irritation along the course of the vagus nerve, by means of sinapisms or gentle galvanism. In cases where the symptoms are severe, and where there is evidence that the right cavities of the heart are greatly over-distended, removal of blood may prove decidedly serviceable for the time, either by venesection, local cupping, or application of leeches; but it must be remembered that this measure tends to induce anæmia, and to impair the nutrition of the heart, and thus may ultimately do more harm than good, so that all the conditions present should be carefully considered in every instance before proceeding to its adoption.

*Dropsy* is a symptom which sooner or later supervenes in a large proportion of cases of heart-disease. Rest and position are of great service in treating this form of dropsy. Those *diuretics* are most beneficial which act upon the heart, and thus increase the arterial tension in the kidneys, especially digitalis, convallaria, and caffein. Dr. Leech agrees with Dr. Breckenridge that caffein should be given alone when the blood-pressure is fairly normal, but combined with digitalis when the pressure is low. Asparagin has been used as a diuretic in cardiac dropsy with advantage, and also casca bark. Well-diluted gin, hollands, and whisky often prove useful as diuretics. Vapour, hot air, or even Turkish baths are beneficial when they can be borne, and with due precautions they may be persevered in for some time. I have frequently found much benefit from the employment of local baths, by wrapping up the legs in warm fomentations along their whole extent, and covering them with macintosh. It has also been recommended to excite the skin into activity by surrounding the patient with hot-water bottles while in bed. Purgation is often attended with beneficial results, but this mode of treatment requires care, on account of the depression which may thus be induced. Frequently it is not desirable to check diarrhoea in cases of cardiac dropsy, as it helps to unload the vessels; and it may be allowed to continue, provided the patient is not evidently lowered from its excessive amount. *Tonics* and *nutrients* are of much benefit in some cases. With respect to operations for the removal of dropsy associated with heart-disease, if anasarca is con-



siderable in amount, and does not soon yield to proper treatment, acupuncture or the use of Southey's trochars should decidedly be resorted to, in my opinion, for I have often observed great relief follow the adoption of these measures, and in extreme cases remarkable benefit may result from free scarification or incision. Of course due regard must be paid to position and cleanliness. A time comes in many cases of cardiac disease when dropsy cannot be relieved, and when medicines administered for this purpose do more harm than good.

Much difficulty is frequently experienced with regard to procuring sleep in advanced cardiac cases. Opiates, hydrate of chloral, bromide of potassium, and other remedies of this class are frequently dangerous, as they tend to induce a condition in which the voluntary efforts necessary for carrying on respiration are suspended, and death might speedily ensue. Still in many such cases they must be given, or it may be justifiable to try small subcutaneous injections of morphia, stimulants being given freely at the same time. When the patient becomes semi-comatose from carbonic acid poisoning, the bladder must be regularly emptied.

7. It is necessary to attend to the state of the other principal organs of the body, when the heart is affected, and, as far as possible, to prevent them from becoming involved, especially the lungs, kidneys, and liver. Every source of cold should be particularly avoided, and the slightest pulmonary complaint must be attended to and treated without any delay. An occasional dose of some medicine which acts upon the liver may be useful.

---

## CHAPTER XXV.

### MALFORMATIONS OF THE HEART AND GREAT VESSELS—CYANOSIS—BLUE DISEASE.

THE term *cyanosis* merely indicates a peculiar appearance presented by a patient, which is especially observed in connection with malformations of the heart and great vessels, but is often associated to a greater or less degree with other affections which obstruct the circulation, and interfere with the due aëration of the blood.

ÆTIOLOGY.—The *pathological causes* of cardiac malformations are in the great majority of cases either :—(a) *Arrested development*; or (b) *Endocarditis* or *myo-carditis* occurring during intra-uterine existence, which is by far more common on the right side, especially in connection with the pulmonary orifice. Possibly some forms of malformation may in very exceptional instances be acquired after birth, in consequence of the rupture of a septum.

ANATOMICAL CHARACTERS.—The following are the chief morbid conditions associated with the heart and great vessels, which come under the class of *congenital malformations*, excluding some which are merely of anatomical interest, and do not give rise to any obvious ill-effects :—

I. **Cardiac.** 1. Patent foramen ovale, or even complete absence of the auricular septum. 2. Perforation or incomplete development of the ventricular septum. 3. Owing to the above-mentioned conditions being carried to an extreme degree, there may be but one auricle or ventricle; or sometimes an auricle and ventricle are thrown into one;

or there may even be scarcely any separation between either of the cavities. 4. Extreme smallness of the right ventricle, either from the septum lying too much in this direction, or from cicatricial thickening and stricture. This condition is in very rare instances observed on the left side. 5. Constrictive disease of the tricuspid orifice, or contraction of the valves, leading to obstruction or regurgitation. The same conditions are said to be met with, but only very exceptionally, in connection with the mitral orifice.

**II. Vascular.** 1. Constriction or incomplete development of the pulmonary artery. In an interesting case which came under my notice only the right branch of the pulmonary artery existed, which was quite pervious, but the valves were extensively diseased and calcareous, the left lung being completely collapsed and disorganized. 2. Constriction or coarctation of the aorta. 3. Transposition of the arteries, the aorta coming from the right ventricle, and the pulmonary artery from the left. 4. Both vessels may spring either entirely or partially from the same ventricle, owing to displacement or imperfection of the septum. 5. Occasionally there is but one arterial trunk, which comes from a single ventricle, and then divides into two. 6. The ductus arteriosus is often pervious.

Some of the conditions just enumerated are met with together, being in fact the necessary consequences of each other. Thus the most common malformation observed is a combination of constriction of the pulmonary orifice with an open foramen ovale and a pervious ductus arteriosus, through which the blood passes from the aorta into the pulmonary artery, some of it also reaching the lungs through enlarged bronchial arteries. If the aorta is closed, the foramen ovale and ductus arteriosus also remain open, the blood being conveyed by the latter from the pulmonary artery into the aorta.

**SYMPTOMS.**—It will be noticed on studying the changes above mentioned that they may disturb the circulation in one or more of three ways, namely :—1. By allowing a free intermixture of venous and arterial blood. 2. By obstructing the circulation, especially interfering with its passage into the lungs, the venous system being hence overloaded; or with its return from these organs. 3. By rendering the systemic circulation entirely venous, the pulmonary entirely arterial, in consequence of transposition of the arteries. Some of the malformations are quite incompatible with life for any length of time. In other cases patients may live for many years, even up to twenty or more, and the symptoms may not declare themselves for a considerable time after birth. These symptoms are simply such as are associated with deficient blood-oxygenation and general venous stagnation, which have already been fully considered, and which are presented in some forms of malformation in their most intense degree. The colour of the surface may be blue, leaden, purple, livid, or of a clarety hue, often mottled, and it is very marked in the lips, ears, fingers, and toes. It is intensified by any act which tends to increase the difficulty in carrying on the circulation, such as crying or coughing. The cause of this discoloration has been much discussed, but it is probably the combined result of intermixture of venous and arterial blood; venous stasis; and imperfect arterialization. Fits of palpitation are common, being often attended with extreme irregularity, and a disposition to syncope or coma. Dyspnœa, cough, and other lung symptoms are also of frequent occurrence, these organs being liable to various morbid changes.

The *physical signs* will vary according to the morbid condition present. If either of the orifices or valves is affected, there will be a corresponding murmur, and a pulmonary murmur is most common in these cases. It is questionable whether a patent foramen ovale can give rise to a murmur. In course of time signs of hypertrophy and dilatation or of degeneration are very likely to be observed.

The *duration* of cases of congenital cyanosis is very variable, and patients sometimes linger on for a long while, apparently becoming accustomed to their semi-asphyxiated state. Death never takes place suddenly, but usually very gradually, being hastened, however, by pulmonary complaints, nervous disorders, and other causes.

TREATMENT.—All that can be done is to attend carefully to hygienic measures, including moderate exercise, baths followed by friction, and the wearing of warm clothing with flannel next the skin; to give plenty of good food, especially of a hydro-carbonaceous kind, including a small quantity of some alcoholic stimulant; and to treat any condition calling for special attention. Iron and other *tonics*, with cod-liver oil, are often beneficial. A dry warm climate is desirable, and every source of cold must be avoided.

## CHAPTER XXVI.

### DISEASES OF THE ARTERIES.

1. **Acute arteritis** is said to be observed in connection with the aorta—*aortitis*, in the course of blood-diseases; and to be characterized anatomically by injection of the vasa vasorum, thickening and softening of the coats of the artery, cloudiness and loss of polish of the inner surface, which also becomes rough from fibrinous deposit. Some observers, however, deny that there is any such disease, and affirm that the red coloration is merely due to staining by the colouring matter of the blood. Dr. Moxon has described inflammation of the first part of the aorta due to the impact of a hard, freely-moving vegetation on one of the segments of the aortic valve. This writer has also called attention to a condition which he terms *inflammatory mollities*, and which he thinks depends on a peculiar general state, where there is softening and swelling of the arterial coats in circumscribed spots, which may bulge out and form aneurisms. Acute arterial inflammation may follow the lodgment of an embolus.

SYMPTOMS.—The symptoms which have been described in connection with aortitis are pain over the vessel, sometimes extreme, accompanied with much tenderness or superficial hyperæsthesia; a sense of heat and throbbing; severe constitutional disturbance and restlessness; sometimes a tendency to syncope; and dread of death. The *physical signs* are objective pulsation; and occasionally a thrill and murmur synchronous with the cardiac systole. In the smaller arteries inflammation may lead to complete plugging and obstruction.

2. **Chronic arteritis.**—**Atheroma.**—**Endarteritis deformans.**—**Periarteritis.**—Chronic arteritis is rather an indefinite term. It has been described as a distinct disease, causing thickening of the coats, and



contraction of the affected artery. Usually it is regarded as originating the condition known as *atheroma*, this being preceded by a parenchymatous inflammation of the inner coat—*endarteritis deformans*. The term *periarteritis* has been applied by Charcot and Bouchard to a morbid process affecting the cerebral vessels, commencing in the perivascular sheath surrounding them, but ultimately involving all the coats, and giving rise to miliary aneurisms, which these authorities consider are the common cause of rupture of the cerebral arteries. The external coat of an artery is also sometimes involved by extension of inflammation from neighbouring structures, which leads to thickening and induration. It will only be necessary to discuss further the atheromatous process, this being the most important form of chronic arteritis.

**ÆTIOLOGY.**—The chief causes of chronic arteritis and consequent atheroma are:—1. *Local injury* from distension of, and strain upon an artery, the disease being thus frequently induced by hypertrophy of the heart, overwork, &c. 2. *Constitutional affections*, namely, gout, rheumatism, syphilis. 3. *Abuse of alcohol*. 4. *Senile degeneration*.

**ANATOMICAL CHARACTERS.**—In the first instance the deeper layers of the internal arterial coat become infiltrated with new cells, softened, relaxed, and thickened. The cells are probably mainly derived from proliferation. As the result of this process thickened patches or more extensive tracts are observed over the inner surface of the artery, and two kinds of patches are described, supposed by some to be merely stages of the same process, namely, those which are soft, jelly-like, moist, and pale-reddish; and firmer, semi-cartilaginous or horny, raised patches, translucent, but more opaque in the deeper layers, and compared in appearance to boiled white of egg (Niemeyer). The superficial portion of the coat is unaffected, and can be stripped off. More or less rapidly fatty degeneration sets in, this change beginning in the soft form chiefly in the superficial layers, in the cartilaginous variety in the deeper layers. In some cases degeneration takes place very speedily, owing to the abundance of cells, and a yellowish, soft, pultaceous substance is formed, like a greasy paste, giving rise to a sort of pseudo-abscess or *atheromatous pustule*, which may ultimately burst into the artery; at first merely a small hole is formed in the inner coat, through which the soft contents escape, being then carried away by the blood, but finally an *atheromatous ulcer* is formed, varying in size and depth, sometimes extending down to or even involving the middle coat. The softened material consists of broken-down fibres, granular cells, abundant fat-granules, and crystals of cholesterin. In other cases, where the process is more chronic, the substance is firmer, becoming caseous; and in others more chronic still, partial organization takes place, leading to fibroid thickening, but this is always accompanied with some degree of degeneration. Ultimately calcification is very liable to happen; or, as some suppose, actual ossification, hard depressed plates being thus originated, or the smaller arteries being involved in their entire circumference, so that they are converted into rigid tubes. The calcareous plates are at first covered by the superficial portion of the lining membrane, but this is liable to give way, leaving a rough surface exposed, upon which fibrin is then very apt to be deposited.

The vessels affected, as well as the extent over which the changes are observed, vary widely, and different stages of the process are usually

seen in the same case. The changes are most marked in those parts of the vessels which are subject to the greatest strain, especially in the ascending and transverse portions of the arch of the aorta, and around the openings of arteries which come off laterally, such as the intercostals. As a rule atheroma is more advanced in the aorta than in the arteries generally.

3. **Fatty degeneration.**—Primary fatty degeneration of arteries is an entirely distinct process from that constituting atheroma. It begins generally in the superficial part of the inner coat, but may extend into the middle coat, or may even implicate this originally. The epithelial and connective-tissue cells of the inner coat are directly changed, becoming more or less filled with fat granules, but in the middle coat the muscular fibres undergo degeneration. This degeneration is usually characterized by small, scattered, irregular, opaque, yellowish-white patches, which are quite superficial, only very slightly projecting, and easily removed, leaving normal tissue underneath. As the deeper layers become involved, the patches appear more opaque and irregular, and are less easily stripped off. In course of time complete destruction and softening may take place, nothing but fat-granules remaining, which are carried away by the blood-current, leaving irregular superficial erosions. Finally calcification may be set up. The capillaries are also liable to become the seat of this fatty degeneration.

4. **Calcification.**—As already stated, calcification frequently follows other forms of degeneration, but it may also take place primarily in the coats of the arteries.

5. **Changes in Bright's disease.**—In certain forms of Bright's disease the small arterioles become contracted and thickened. According to one view the thickening is due to hypertrophy, especially of the muscular coat; according to another it results from a hyalin-fibroid change. The capillaries are also similarly affected, the condition being generally known as *arterio-capillary fibrosis*.

6. **Atrophy.**—Occasionally a large artery, especially the aorta, undergoes simple atrophy, the walls becoming gradually thinned.

7. **Alterations in calibre.**—An artery may be the seat of *dilatation* in its entire circumference; or, on the other hand, of *contraction* or *coarctation*, which may end in its complete closure.

8. **Albuminoid degeneration.**—This change commences in the small arteries of certain organs, especially the spleen and kidneys. It does not affect the larger arteries.

9. **Syphilitic disease.**—It is difficult to determine positively whether syphilis produces any specific changes in arteries. Such changes have been described by Heubner, Davidson, Hughlings Jackson, Wilks, and others; but their existence is denied by other observers. It is stated that the cerebral arteries present gummatous infiltration in some cases, giving rise to nodose swelling, with great thickening of the coats, and narrowing of their calibre, which may lead to thrombosis and cerebral softening.

10. **Aneurism.**—This is one of the most important morbid conditions affecting arteries. Aneurisms, however, come more especially under the care of the surgeon, and therefore for a full consideration of the subject reference must be made to surgical treatises. Small miliary aneurisms are important in medical practice, and these have already been alluded to as being met with in the cerebral and pulmonary arteries. With regard to larger aneurisms, it will only be practicable

in this work to point out the main facts relating to those which are located in the thorax or abdomen, more particularly when the disease is connected with the aorta.

**SYMPTOMS AND EFFECTS.**—The consequences of the chief chronic changes in the arteries just described are very similar, and they are of considerable importance, often giving rise to prominent symptoms connected with various organs, and leading to serious lesions of structure. Here it need only be indicated in a general way what these effects are, and they may be thus stated:—1. The elasticity of the arteries is more or less diminished, until finally it is completely lost; their resistance is increased; and they are ultimately converted into rigid tubes, at the same time their calibre being diminished. Hence an obstacle to the circulation arises, which leads to hypertrophy of the left ventricle, this, however, tending to be more or less speedily followed by cardiac degeneration. The circulation in the different organs is impeded, and among the most frequent symptoms resulting therefrom are those indicating disturbance of the cerebral circulation, especially giddiness and disorders of the special senses. Owing to the impairment of circulation and consequently of nutrition, structures are very liable to undergo degeneration, and to become inflamed from slight causes. 2. When the vessels are roughened on their inner surface, fibrin is often deposited from the blood, which may ultimately cause their complete obstruction. As a consequence softening or death of a part may ensue, which is well exemplified by chronic softening of the brain, and by dry gangrene of the lower extremities. 3. A limited portion of an artery, especially after the formation of an atheromatous ulcer, is very prone to yield gradually, an aneurism being thus originated. 4. The affected vessels become brittle, particularly when calcified, and thus they are more easily ruptured, giving rise to cerebral apoplexy most commonly. 5. Fragments of the degenerate structures or of fibrinous deposits may be detached, carried away by the blood-current, and lodged in some smaller vessels as emboli. 6. *Physical examination* of the arteries reveals that they are visible, tortuous, and locomotive; and that they feel more or less hard, full, incompressible, rigid or cord-like. A sphygmographic tracing is characterized by the large dimensions of its curves; the approximation of the secondary waves to the summit; and the great size of the first secondary wave as compared with the aortic, the latter being much diminished. Murmurs may be present over diseased arteries.

When the arch of the aorta is extensively diseased, especially calcified, a jerking impulse may be observed to the right of or above the sternum, and occasionally a thrill; while a rough systolic murmur is sometimes heard along the course of the vessel, or a cardiac basic murmur may be intensified in this direction. The artery is often somewhat dilated at the same time, and this will increase the signs just mentioned.

**DIAGNOSIS.**—Degeneration of arteries should always be looked for in persons at all advanced in years, and it should be kept in mind as a probable cause of many symptoms of which they complain; the condition may, however, be met with in persons who are comparatively or actually young. Examination of the vessels is the only satisfactory means of diagnosis, and if the arteries generally are affected, probably the aorta will be in the same condition. Some attach considerable importance to the sphygmographic tracing as revealing an early stage of degeneration.



**PROGNOSIS.**—This merely involves a knowledge of the dangers which accompany degeneration, so that they may be guarded against. Many persons live to a good old age whose vessels are much diseased, but at any moment there is a liability to dangerous lesions. The earlier the degeneration comes on, the more serious is the prognosis.

**TREATMENT.**—All that can be done is to avoid everything which is likely to throw a strain upon the vessels; and to maintain the nutritive activity of the system as much as possible by means of good diet, *tonics*, and cod-liver oil, the last being often decidedly useful. Any constitutional diathesis must be attended to; and all injurious habits checked.

### THORACIC ANEURISM.

The aorta is by far the most common seat of aneurism within the chest, but the innominate, the commencement of the left carotid or subclavian, or the pulmonary artery may be involved.

**ÆTIOLOGY.**—Aneurism of the aorta almost always results primarily from some morbid change in the walls of the artery, especially from chronic endarteritis and the atheromatous changes connected therewith, but also sometimes from mere fatty degeneration or simple atrophy. Its *determining cause* is generally some more or less violent exertion, which throws a sudden or frequent strain upon the weak portion of the vessel, and this may even lead to rupture of part of its coats. It may be developed in this manner either suddenly, or more or less gradually.

Aneurism is much more common among males, especially those whose occupation entails violent efforts; and about the middle period of life. It is comparatively extremely frequent in the army, and this has been attributed to the combined effects of great exertion; tight clothing, which compresses the neck and chest, and obstructs the circulation; and heavy accoutrements. The diseases which predispose to changes in the vessels, such as syphilis, gout, and rheumatism, may be considered as *predisposing causes* of aneurism, especially syphilis. It has been stated to be occasionally hereditary, but this is probably only true as regards the degeneration of the vessels.

**ANATOMICAL CHARACTERS.**—The following varieties of aortic aneurism are met with:—1. There may be a *general dilatation*, involving the whole circumference, and either cylindrical, fusiform, or, very rarely, globular in shape. 2. *Sacculated aneurism* is the most important variety, in which there is a lateral bulging or sacculation of a portion of the circumference of the artery, the coats being either entire—*simple* or *true aneurism*; or more or less of the inner and middle coats being destroyed—*compound* or *false aneurism*. Sometimes all the coats give way, and the aneurism is bounded only by surrounding structures—*diffuse aneurism*. 3. In extremely exceptional cases a *dissecting* aneurism is observed, the blood finding its way between the coats of the vessel. The ascending portion of the arch is most frequently affected, especially on its convex side, where the aorta is most exposed to strain; an aneurism may exist, however, on any part of this vessel, even between the pillars of the diaphragm. Great varieties are presented as to size, exact shape, contents, and other characters.

**SYMPTOMS.**—The symptoms of aortic aneurism are far from uniform, being chiefly due to pressure on surrounding structures (*see* page 481),

and therefore influenced by its situation, size, form, rapidity of formation, and direction of growth; while they are also liable to alter during its progress. The symptoms are by no means in proportion to the external physical evidences of aneurism; indeed, the reverse is often true, because the more an aneurism tends in an inward direction, the more severe are the symptoms likely to be, and they may be extremely aggravated when it is impossible to detect any sign by physical examination. In some cases there are no symptoms or physical signs from first to last. Abnormal *local sensations* are usually present, such as pain, varying in characters and intensity, heat, fulness and weight, or throbbing; while tenderness or cutaneous hyperæsthesia is common. If the aneurism passes backwards, the pain may be deep and gnawing or grinding, owing to destruction of the vertebræ. Among the most frequent *pressure-symptoms* are those indicating interference with the main air-tube, which in many cases first attract attention. The *constitution* often suffers markedly, even when there are no particular local symptoms or signs, and I have sometimes observed a very striking appearance of illness, combined with anæmia or a sallow cachectic look, and an anxious, distressed, or irritable expression, but without any particular emaciation, which has led me to suspect internal aneurism when there was no evident cause to account for these phenomena. The posture assumed by patients suffering from aortic aneurism depends upon its situation and other circumstances; in certain cases they cannot lie down, but keep the head high, and some patients have a tendency towards a prone position, so as to take off pressure from the structures behind; bending the head forward and then throwing it back suddenly, has been regarded as a movement suspicious of aneurism. The digestive organs frequently suffer. Head-symptoms are also common, with disturbed sleep. The urine is not altered. Aneurism may give rise to embolism in some distant organ, especially in the brain.

**PHYSICAL SIGNS.**—The following include the physical signs which are to be looked for as indicative of aneurism, but not uncommonly they are very obscure:—1. *Local bulging* may be detected, its site depending upon the part of the aorta involved. If the arch is affected in its ascending or transverse portion, the prominence will be in front, opposite or to the right or left of the upper part of the sternum, the exact situation differing much in different cases. Aneurism of the remainder of the arch, or of the descending aorta, may give rise to bulging posteriorly, generally to the left of the spine, occasionally to the right, and it is sometimes very extensive. In shape the swelling tends to be conical, and it involves the ribs and spaces equally. 2 *Pulsation* over any swelling, or even without any enlargement, is an important sign, this being usually synchronous with the ventricular systole, but sometimes double, or it may be more marked during the diastole. The systolic pulsation is usually expansile, heaving, and throbbing. Sometimes it is distinctly undulatory. In exceptional instances a thrill is felt. It is important to observe that the stethoscope may aid in discovering slight pulsation, when it cannot be detected by the fingers. The cardiograph has been employed to record the pulsations of an aneurism. 3. *Dulness* corresponding to any bulging may be elicited, though frequently extending beyond this to a variable degree, and across the middle line, or being observed when there is no actual prominence; it is of a dull, dead, putty-like character; and is accompanied with increased resistance. When the aneurism presses firmly against the sternum, the dulness may

extend upwards and downwards along this bone beyond the actual site of the tumour. 4. *Auscultation* gives extremely variable results. There may be nothing whatever heard, or only indefinite sounds. The important auscultatory sign of aneurism, however, is the presence of a rough *murmur*, usually systolic, occasionally double, or very exceptionally only diastolic. 5. There may be signs of hypertrophy of the left ventricle, but in most cases in which the *heart* is affected this organ is merely displaced downwards and to the left. If the aneurism lies behind, the heart may be so pushed forward that the chief impulse is observed at the base. It must be remembered that actual cardiac disease may be present along with aneurism. 6. Examination of the *larynx* and *lungs* might reveal functional disorder of, or organic mischief in the former; displacement or compression of, or interference with the entrance of air into the latter; or bronchial catarrh on one or both sides. 7. The *radial pulse* often affords important signs, especially to the sphygmograph. The chief characters are that the pulse is delayed on one side; or that it differs in fulness and force on the two sides, being sometimes completely absent on one side. The sphygmograph reveals even a slight difference in the two pulses, but this is very marked in some cases. The *dicrotism* is often influenced also, and when the descending aorta is involved, this may be much increased, especially on the right side. An aneurism is capable of influencing the pulse, not only by its own direct effect upon the circulation, but also by obstructing the main arteries, in consequence of pressure, closure of their orifices by a clot, or torsion.

MODES OF TERMINATION.—Death is the ordinary termination of aortic aneurism, and it may be immediately due to:—1. Gradual asthenia. 2. Effects of pressure. 3. Rupture of the aneurism and consequent hæmorrhage, which may take place into the pericardium, heart, neighbouring great vessels, pleura, mediastinum, trachea or either bronchus, lungs, œsophagus, or spinal canal; or externally. 4. Independent affections, either acute or chronic.

DIAGNOSIS.—It would be easy to write to almost any extent on the difficulties which might and do arise in the diagnosis of thoracic aneurism, but it must here suffice to offer a few general observations on the subject. It is not only necessary to determine the presence of an aneurism, but also its seat, variety, size, and other characters as accurately as possible. In some cases the signs are so evident that there is but little difficulty in making out all that is required; but the following classes of difficulties are principally met with, namely:—1. There may only be symptoms indicating more or less pressure within the thorax; or sometimes merely obscure and ill-defined sensations, with constitutional disturbance, but no external signs. 2. An aneurism may give rise to the physical signs of a tumour, but without any pulsation or murmur. 3. Other pulsating prominences are occasionally observed besides aneurisms, the pulsation being usually transmitted from the heart or aorta.

The chief morbid conditions which aortic aneurism is liable to simulate, or *vice versa*, are a solid mediastinal tumour or abscess, the latter occasionally presenting pulsation; pulsating empyæma; phthisical consolidation at the left apex, with subclavian or pulmonary murmur; swelling over the sternum from chronic periostitis or abscess; a tumour or suppuration in other parts of the chest-walls; pericardial effusion; innominate aneurism; or cardiac disease. Among very rare conditions may be mentioned coarctation of the aorta; varicose aneurism; and aneurism of the pulmonary artery.



The points to be taken into account in making a diagnosis are as follows:—1. The age and sex of the patient; previous history, especially with regard to occupation and former diseases; family history; and that of the origin and progress of the complaint. 2. The presence or absence and exact nature of pressure-signs. 3. The other symptoms observed, particularly noting whether there is general dropsy or albuminuria. 4. The exact situation of any prominence. 5. The precise site, extent, rhythm, and characters of any pulsation, especially noting whether it is heaving and expansile, double, or attended with thrill; and if it is distinct from the cardiac pulsation. 6. The seat and extent of dulness, particularly observing whether it is in the course of the aorta, or crosses the middle line; and if it corresponds most to any pulsation which may be evident. 7. The presence and characters of murmurs, but care must be taken not to mistake these for conducted cardiac murmurs. 8. The characters of the pulse, especially as revealed by the sphygmograph; and also the effects of pressure upon the great vessels in the neck.

The distinctions between aneurism and a *solid tumour* will be hereafter considered. The chief difficulties arising in the diagnosis of aneurism from *cardiac diseases* are, that aneurism may be simulated by enlargement of this organ accompanying valvular disease, especially if the aorta is atheromatous; or that an aneurism with very thin walls and fluid contents, pushing the heart downwards and to the left, may be mistaken for mere cardiac enlargement. The principal circumstances in favour of cardiac disease are there being but one centre of impulse; the physical signs corresponding to the region of the heart, or being most marked here; the absence of pressure-symptoms; and the presence of general dropsy or albuminuria.

As regards the *form* of an aneurism, the signs in favour of general fusiform dilatation are given by Walshe as more diffuse pulsation, both above and below the clavicle; well-marked thrill; rough, prolonged, rasping, whizzing, or whirring murmur, which is systolic, audible along the arch, or louder there than over the aortic orifice; and absence or slight degree of pressure-signs.

The *part of the vessel* affected must be determined by the locality of the physical signs, and the exact pressure-phenomena observed; comparison of the radial pulses, especially as revealed by the sphygmograph, may afford some aid.

In distinguishing *innominate aneurism* from *aortic* the following considerations have weight:—The physical signs correspond in situation to the innominate artery; the prominence appears early, and it may displace the clavicle; it is said that dysphagia and dyspnoea from the pressure of an innominate aneurism are rare, but I have known both these symptoms extremely severe; there are often signs of pressure on the nerves of the right brachial plexus, and on the right bronchus; the right radial pulse is always modified; and pressure on the carotid and subclavian arteries on the same side diminishes the pulsation.

TREATMENT.—The first object in the treatment of an aneurism should be to endeavour to bring about its cure by promoting gradual coagulation within the sac, but this can only be aimed at in the sacculated variety of aortic aneurism. Failing this, it is necessary to protect the aneurism; to retard its development as much as possible; and to treat the symptoms and complications which so frequently arise.

1.—If it is intended in any case to attempt to cure a thoracic aneurism, it is absolutely essential to keep the patient *at rest in the recumbent posture* for a considerable time, and to avoid every source of physical or mental disturbance. Formerly it was the custom to have recourse to starvation and repeated venesection; but at the present day this has been with good reason modified into a *careful regulation of diet*, a definite quantity of solids and liquids being administered at stated intervals, according to Mr. Tufnell's method. The exact amounts allowed must depend upon each individual case, but everything should be strictly weighed or measured, the object being to support life with as little food and drink as possible, without inducing nervous irritability. Excess of fluid must be particularly avoided, and all stimulants are to be prohibited. In some instances it may be advisable to remove a little blood from time to time, but it is very important to avoid inducing an anæmic condition.

The objects of this attention to rest and diet are to calm the circulation as much as possible, and to render the condition of the blood more favourable for coagulation, and undoubtedly some cases do improve considerably under this treatment alone. *Medicinal agents*, however, may be employed with benefit at the same time, namely, those which subdue and regulate the heart's action, such as digitalis, aconite, or belladonna; and those which promote coagulation, principally gallic or tannic acid, tincture of steel, acetate of lead, and iodide of potassium. The use of iodide of potassium has been particularly advocated by Dr. William Roberts of Manchester, and Dr. George Balfour of Edinburgh, when given in large doses, even as much as from 15 to 30 grains thrice daily, and continued for a long period. I have found this drug of decided value in some cases. Subcutaneous injection of ergotine has also been recommended.

2. It must suffice merely to mention certain operative procedures which have been resorted to with the view of curing aortic aneurism. These are:—*a.* Injection of perchloride of iron, ergotine, or other coagulating agents into the sac. *b.* Manipulation of the sac externally. *c.* Galvano-puncture. *d.* Introduction of a quantity of fine iron wire through a canula, or of horsehair, or carbolized catgut. *e.* Ligature of the right carotid and subclavian arteries.

3. It would occupy too much space even to mention the various *symptoms and complications* which may require attention in the progress of a case of aneurism, and only a few practical points can be alluded to here. It is always well to keep the aneurism covered with cotton-wool, and should it be particularly prominent, some kind of protecting shield might be worn. For relieving pain and procuring sleep the chief internal remedies are opium, morphia, hyoscyamus, lactucarium, hydrate of chloral, bromide of potassium, and conium in full doses. Subcutaneous injection of morphia is also most valuable. External applications are frequently useful, such as belladonna or opium plaster; belladonna or aconite liniment; cold poultices of linseed-meal and vinegar, conium, digitalis, or oak-bark (Walshe); ice, ether-spray, or chloroform cautiously applied; counter-irritation by flying blisters or iodine, which sometimes gives marked relief. Galvano-puncture may greatly alleviate pain. If there are severe laryngeal symptoms, evidently due to pressure on the recurrent nerve, it is decidedly justifiable to perform tracheotomy, and let the patient wear a tube in the trachea permanently. It has been suggested that in certain cases the sterno-clavicular ligaments might be divided with advantage, in order to allow displacement of the clavicle forwards, and thus take off pressure from behind.

## CHAPTER XXVII.

## MEDIASTINAL TUMOURS.

AORTIC aneurism is the most frequent form of mediastinal enlargement, and therefore it has appeared to me most convenient to introduce in this place what has further to be said on the subject of mediastinal tumours. The other chief varieties met with include cancer (either encephaloid or scirrhus-encephaloid), originating in the œsophagus, lymphatic glands, root of the lung, or thymus gland; enlarged masses of absorbent glands in tuberculosis, or in lymphadenoma; fibro-cellular, fibrous, or fibro-fatty tumours; inflammatory exudation and abscess; very rarely masses of steatoma or hair.

**SYMPTOMS.**—The symptoms of mediastinal tumour are mainly those indicative of pressure, and they accordingly present the usual variations. “Currant-jelly” expectoration is said to be common in cancer. There may be constitutional symptoms of this diathesis. The *physical signs* of a solid tumour are also widely different, but the following list will suggest those which are to be sought for:—1. *Local bulging*, especially in front, of variable extent, often irregular, not pulsating. 2. Deficiency or absence of *respiratory movements* over the seat of the growth, or in some instances over the whole of one side, from pressure on a bronchus. 3. Altered *percussion-sound*, often over a considerable area, it being either dull and toneless; hard, wooden, and high-pitched; or occasionally tubular or amphoric; there being also marked *resistance*. 4. *Respiratory-sounds* weak or absent, blowing, or tubular, according to the size of the growth, and its relation to the main air-tubes. 5. *Vocal fremitus* usually absent; and *vocal resonance* either deficient, bronchophonic, or pectoriloquous. 6. *Dry* and *moist râles* in the bronchi, either general, unilateral, or local, which are not infrequent. 7. *Displacement* of the heart and other structures; increased conduction of the heart-sounds; and occasionally a murmur, resulting from pressure on a great vessel.

**DIAGNOSIS.**—Mediastinal tumour has in the first place to be distinguished from other morbid conditions within the chest, especially chronic pneumonia; chronic pleuritic effusion; pericardial effusion; and enlargement of the heart. Careful consideration of the history of the case, as well as of its symptoms, physical signs, and progress, will rarely leave much doubt as to the diagnosis thus far. It is much more difficult, however, to determine the *nature* of any mediastinal enlargement. In the diagnosis between *aneurism* and a *solid tumour*, when this is at all doubtful, the following considerations have weight, which have been chiefly compiled from the observations of Dr. Walshe:—1. The facts of the patient being a female and under 25 years of age point to a solid tumour; the family history may be suggestive of cancer; or the occupation may be in favour of aneurism. 2. As regards *symptoms*, dysphagia and severe pain, especially posteriorly, are more common in aneurism; œdema of the arm and chest, frequent hæmoptysis, and currant-jelly expectoration, are more characteristic of tumour. Occasionally cancer-elements may be discharged in the sputa. 3. The



*physical signs* are of much value. The limitation of such signs to the region of the aorta; the presence of any thrill; a double impulse; and gradual approach of any pulsation to the surface, are suggestive of aneurism. Great superficial extent of dulness; absence of any heaving character in the pulsation, should this sign be present; and the want of accordance between it and the maximum dulness, are in favour of a solid tumour. 4. Careful examination may reveal cancer in other parts; or there may be constitutional indications of its presence.

With regard to the distinction between *different solid enlargements*, all that can be stated is that cancer is the most common; there may be signs of the cancerous cachexia or of cancer in other parts; while abundant hæmoptysis is by far most frequent in this form of tumour, or cancer-cells may be expectorated. It also tends to grow outwards, and has a rapid progress. Lymphadenomatous growths must, however, be borne in mind, as they are likely to be mistaken for cancer.

TREATMENT.—All that can be done in a case of mediastinal tumour is to treat it on general principles, and to relieve symptoms as they arise.

---

## CHAPTER XXVIII.

### ON CERTAIN ABNORMAL CONDITIONS OF THE BLOOD.

THE changes which take place in the blood in different diseases are now attracting considerable attention, and it is becoming more the custom to submit this fluid to clinical examination. The pathology of the blood is such an extensive subject that it is impossible to enter into any systematic consideration of it in this work. All that can be done here is to point out the more common methods of clinical investigation; and to give a summary of the more obvious changes to which the blood is liable.

CLINICAL INVESTIGATION.—Formerly the chief information which the blood was supposed to afford was by the mode in which it coagulated after removal in quantity from the body, especially by venesection, the so-called “buffy coat” being a familiar illustration of what was regarded as an important abnormal condition. At present, however, it is only exceptionally that blood is thus examined; but it must be remembered that the mere appearance of blood in bulk, and the way in which it coagulates, may give useful information. It may be requisite to submit this fluid to *chemical analysis*, or to examine it with the *spectroscope*; but only those specially skilled are competent to conduct such investigations. The methods of examination to which the blood can be ordinarily subjected are as follows:—1. *Simple microscopic examination*. In order to examine recent blood under the microscope all that is necessary usually is to cleanse carefully the end of the finger, and prick it with a needle. No pressure must be used to squeeze out the blood, but the drop which escapes spontaneously must be received on a clean microscopic slide, the cover-glass applied, and the examination carried out immediately. 2. *Examination by special apparatus*. Two kinds of

special instruments are used in the clinical investigation of the blood, namely, the *hæmacytometer*; and the *hæmoglobinometer*. Different forms of these instruments are employed, but in this country those invented by Dr. Gowers are chiefly used. The *hæmacytometer* is for the purpose of counting the number of corpuscles contained in a given volume of blood, both red and white, and thus determining the corpuscular richness of the blood, and the relative proportion of red and white corpuscles. In employing it a definite dilution of a certain quantity of blood is made, and the number of blood corpuscles in a certain volume of this dilution is counted by the aid of the microscope. Dr. Gowers uses a glass-slide, in which there is a cell, having lines engraved at the bottom, so as to enclose squares within which the corpuscles may be counted. The *hæmoglobinometer* is intended to estimate the amount of hæmoglobin in the blood. This object is effected either by diluting the blood to a given point, and comparing the colour with standard solutions of carmine or picro-carmine, or with coloured discs; or, according to Dr. Gowers's method, by progressively diluting the blood until it reaches the tint of a standard, the colour of which corresponds to a dilution of 1 part of healthy blood in 100 of water. The standard consists of glycerine jelly coloured to the required tint. The degree of dilution necessary to make the blood correspond to the standard tint represents the amount of hæmoglobin.\*

CHANGES IN THE BLOOD.—The principal morbid alterations to which the blood is liable may be summed up as follows:—

I. **In its absolute quantity.** This may be:—1. Excessive (*plethora*, *polycæmia*, or *hypercæmia*). 2. Deficient (*hypcæmia*, *oligæmia*, or *anæmia*).

II. **In colour and obvious physical characters.** The blood may be unusually pale and watery; dark from pigment; thick and tarry; or the serum has sometimes a milky or chylous appearance, due to the presence of fat. Occasionally the blood presents puriform streaks. Lake blood is a peculiar condition in which this fluid becomes transparent. Its colour is altered after poisoning by various substances.

III. **In the number and characters of the corpuscles.** 1. The red corpuscles may be:—(a) deficient (*oligocythæmia*); (b) in excess (*polycythæmia*); (c) deficient in hæmoglobin (*oligochromæmia*); or (d) altered in shape and size, in consistence, or in their tendency to and mode of mutual adhesion. 2. The white corpuscles are often too numerous, but this is particularly seen in the condition named *leucocythæmia*. A lesser degree of increase is termed *leucocytosis*. On the other hand, they may be deficient.

IV. **In the normal chemical constituents.** 1. Fibrin may be:—(a) increased (*hyperinosis*); (b) diminished (*hypinosis*); or (c) altered in its tendency to coagulate. 2. Albumen is frequently deficient (*hypalbuminosis*); sometimes above the normal (*hyperalbuminosis*). Hæmoglobin may be deficient (*aglobulism*). 3. Water may be excessive (*hydræmia*); or deficient. 4. A diminution in alkaline or earthy salts, especially those of potash and lime, is considered important in some diseases; occasionally they are increased. 5. Fatty elements, particularly cholesterin, may be present in unusual quantity. Chylous blood is due to the presence of a large quantity of fat in the serum, a cream-like layer forming on the surface, consisting of granules and oil globules. Fat particles may also be separate. 6. Carbonic acid is in excess in some conditions. V. **Abnormal chemical substances**

\* For further details see articles "*Hæmacytometer*" and "*Hæmoglobinometer*" in *Quain's Dictionary of Medicine*.

are often found in the blood; or at all events such as are usually present in so small a quantity as to be detected only with difficulty, and to be practically harmless, for example, lactic, uric, hippuric, formic, and other organic acids; leucine and tyrosine; urea and its products; sugar; bile-elements; and certain metals. VI. **Abnormal microscopic particles** are also not uncommonly observed, such as abundant pigment-granules (*melanæmia*); pus-corpuscles; and vegetable or animal parasites, the most important illustrations being bacteria, the spirillum of relapsing fever, the bacillus anthracis, and the filaria sanguinis hominis.

## ANÆMIA—SPANÆMIA—CHLOROSIS.

The term *anæmia* is used very vaguely, and, in addition to its strictly literal sense, it is applied in practice to three classes of cases, namely:—(a) where the blood as a whole is deficient in quantity; (b) where it presents certain abnormal qualities; and (c) where the arteries are inadequately filled. These conditions, however, are usually more or less combined. The chief alterations in quality include deficiency of red corpuscles, or of hæmoglobin in these corpuscles; often deficiency of albumen; with excess of water and salts, the serum having a low specific gravity—*spanæmia* or *serous polyæmia*. Fibrinogenous elements are proportionately high, and the blood has a tendency to coagulate in the veins. The deficiency of red corpuscles may be due to direct loss of blood; imperfect formation; or excessive destruction. In one form of anæmia they are more numerous, but ill-shaped, and there is much granular matter in the blood. *Chlorosis* or *green sickness* merely signifies that there is a peculiar greenish or yellowish-green tint, such as is frequently observed in anæmic girls, associated with menstrual derangements, the colour being supposed to depend upon some chemical change in the blood-pigments.

**ÆTIOLOGY.**—Very many causes may give rise to anæmia, but the chief include considerable loss of blood at one time, or repeated small losses; unfavourable hygienic conditions, especially constant sedentary or laborious employment in a confined atmosphere, with deficiency of sunlight; improper or insufficient food, particularly a want of animal diet; impaired power of digestion; excessive drain upon the system, such as from over-lactation, diarrhœa, or chronic suppuration; prolonged exposure to malarial influence, with or without attacks of ague; various chronic diseases which interfere with nutrition, for example, phthisis, cancer, renal disease, leucocythæmia, gastric ulcer; acute febrile diseases; excessive venery or masturbation; depressing mental influences; chronic poisoning by lead, mercury, and other metals. In many instances several of these causes have combined to induce anæmia. An anæmic aspect may be a striking feature in persons suffering from disease of the mitral or aortic orifice, or of the aorta itself, such as coarctation or aneurism, this appearance being mainly the result of an insufficient supply of blood to the arteries.

Females, especially those from 15 to 25 years of age, are most frequently the subjects of anæmia or chlorosis. This has been supposed to be mainly the result of the great demands made upon the developmental powers about the period of puberty, but commonly it will be found in the history of these cases that there are some other obvious causes, which at all events have aided materially in giving rise to the



anæmic condition, the most important being long-continued habitual constipation, which necessarily leads to derangement of the digestive organs and consequent imperfect nutrition; and abstinence from animal food, this being the consequence of the loss of appetite, or rather disgust for food, which is commonly associated with the unhealthy state of the alimentary canal. Some authorities lay much stress upon uterine displacement as a cause of chlorosis. Other influences are also frequently at work in these cases, such as deficient exercise; close confinement; over-work, particularly with certain sewing-machines; and mental depression. Virchow has affirmed that in chlorotic subjects the aorta and its branches are frequently congenitally small and attenuated; and that irregularities in the origin of the arteries are common. To these conditions he attributes a causative relation in such cases.

A special form of anæmia is described under the terms *progressive, idiopathic, pernicious, or malignant*. It cannot be referred to any definite cause; and although it occurs in both males and females, has been most frequently met with in middle-aged pregnant women. This form has been regarded as only an advanced type of ordinary anæmia; but there seems to be an unusual destruction of corpuscles as a characteristic feature. Its pathology, however, is at present by no means evident. It cannot be said to be associated with any constant and definite organic lesion, although it has been attributed to atrophy of the stomach and other conditions.

**SYMPTOMS.**—The appearance of persons suffering from anæmia is, as a rule, sufficiently characteristic. They are pale, often waxy-looking, and have usually a clear transparent skin; or, as already mentioned, a greenish or yellowish-green hue is observed in chlorotic girls. The veins are frequently, however, very evident, and may have a peculiar pinkish tint. The mucous membranes present the most marked signs of anæmia, especially the conjunctiva of the lower eyelid, and the membrane covering the lips, gums, and tongue, these being more or less pale and bloodless. The nails also show the anæmic appearance well. The sclerotics appear clear and bluish or white. The general condition will vary according to the cause of the anæmia; in chlorosis the patient is often apparently well-nourished, though the tissues are usually flabby and wanting in tone. Edema about the ankles, and puffiness of the eyelids in the morning, are common symptoms, and there may even be considerable anasarca of the legs after standing for some time. The influence of anæmia in promoting dropsy from other causes has been previously alluded to, and is often considerable.

The subjective sensations complained of are also of a very uniform character in chlorotic girls. These are debility, languor, and incapacity for exertion; general chilliness and coldness of the extremities; shortness of breath and palpitation after any effort, especially after ascending a height or going upstairs, or even when at rest; a tendency to syncope from time to time; headache, dizziness, and noises in the ears; neuralgic and hysterical pains in different parts of the body, but especially in the left side, this being frequently accompanied with tenderness, and being liable to come and go. It has appeared to me that this pain in the left side may possibly be connected with the spleen in some cases. Anæmic females are often low-spirited or irritable, and subject to hysterical attacks.

The digestive organs are usually at fault in cases of chlorosis. Appetite is impaired or depraved, and there may be an absolute disgust for

food, especially for meat. Gastralgia and atonic dyspepsia are common complaints. The bowels are obstinately constipated as a rule. Hæmatemesis and melæna occur in some cases. Menstruation is almost always deranged, being absent, infrequent, irregular, scanty, unhealthy, painful, or sometimes menorrhagic. Leucorrhœa is also a frequent symptom.

Certain abnormal *physical signs* are observed in marked anæmia, which have been already described, and therefore need only be enumerated here, namely, *cardiac murmurs*, especially at the base of the heart to the left of the sternum; a *blowing murmur* in the arteries, particularly the subclavian, and sometimes a *thrill*; and a *venous hum*, which is in some cases even heard over the cranium, and may also be accompanied with a *thrill*. The heart's action is very liable to be disturbed, becoming easily excited and hurried, and in severe cases even irregular. The pulse is small, feeble, and compressible; or sometimes scarcely perceptible.

The urine frequently presents important changes, being pale and watery, excessive in quantity, of low specific gravity, deficient in acidity, with a marked decrease in the amount of pigment and creatinin.

It has been asserted that anæmia may ultimately lead to organic diseases, such as phthisis or gastric ulcer. Acute affections occurring in anæmic individuals are apt to assume a low type, and to be followed by protracted convalescence.

*Pernicious anæmia* is indicated at first by the ordinary signs of anæmia, which do not yield to treatment, but progressively increase, becoming at last excessive. As the disease advances the patient becomes very weak and emaciated; gastric disturbances may be prominent; irregular febrile paroxysms occur; while anasarca, serous effusions, and hæmorrhages beneath the skin, from mucous surfaces, into the retina, and into internal organs often supervene. Sometimes there is slight jaundice. The red corpuscles are paler than usual, while some of them may be ill-shapen, deep-coloured, atrophied, and nucleated; granular masses of protoplasm may also be present. The termination of pernicious anæmia is almost always fatal, within from six to twelve months. Death occurs either from asthenia, loss of blood, or hæmorrhage into the brain.

**TREATMENT.**—The first thing to be done in all cases of anæmia is to find out its causes, and remove these if possible. Attention to *hygienic conditions* is most essential, especially in the case of chlorotic girls. Fresh air, good light, out-door exercise, avoidance of late hours and of hot and crowded rooms, change of air and scene to some dry and bracing climate, especially to the sea-side, cheerful society, and the removal of all disturbing mental influences, are most efficient aids in treatment. Baths, particularly sea-bathing, or douches followed by friction, are very beneficial if followed by good reaction.

In the next place it is most important to look to the *diet*, and to the state of the *digestive organs*. Nutritious food must be given at stated intervals, and it is often requisite to lay down strict rules on this matter, particularly with regard to meat, to which many of these patients have a strong objection. It ought to be taken underdone, and if it causes pain it may be pounded. Nutritious soups are also useful, and beer or wine is generally indicated. The state of the bowels demands particular notice, and the patient should be fully impressed with the necessity of having a sufficient daily evacuation. Aloes is one of the best forms of aperient in these cases, given at night in the form of aloes

and myrrh pill; or as the extract with extract of belladonna and nux vomica. Remedies which act upon the stomach are frequently very beneficial also; carbonate of bismuth with hydrocyanic acid being particularly valuable for relieving the unpleasant and painful sensations connected with this organ, when taken shortly before meals.

Iron in some form is the great *medicinal remedy* in anæmia. The compound iron mixture is eminently efficacious in chlorosis, and among other excellent preparations may be mentioned the compound iron pill, the saccharated carbonate, the ammonio-citrate, tartarated iron, and ferrum redactum. Tincture of steel is invaluable in many cases, especially when the anæmia is associated with excessive discharges. The solution of the perchloride or pernitrate, the sulphate, the tincture of acetate, and the magnetic oxide are also very useful preparations; while in anæmic children steel-wine and the syrup of phosphate of iron produce excellent results. Dialyzed iron is now much in use. Chalybeate waters are beneficial in some instances. Iron may in suitable cases be variously combined with infusion of quassia or calumba, quinine or strychnine, arsenic, manganese, pepsine, and other remedies. The citrate of quinine and iron, and Easton's syrup are very valuable preparations. It is frequently desirable to change the form of the preparation from time to time; or even to stop the administration of iron temporarily, should it appear to disagree.

The pain in the side often requires attention in chlorosis, and is usually much relieved by making the patient wear a belladonna plaster. Other symptoms must be treated as they arise.

In *pernicious* anæmia remedies have little or no effect. Transfusion of blood has been performed in several instances, but without success. This measure might also be required for anæmia from great loss of blood.

#### PYÆMIA—SEPTICÆMIA.

This subject belongs principally to surgery, and in this work it is merely intended to indicate the chief practical facts with regard to cases of pyæmia or septicæmia which may come under notice in medical practice.

ÆTIOLOGY.—Excluding obvious injuries and operations, the exciting causes of pyæmia or septicæmia may be arranged as follows:—1. Disease of bones, either acute or chronic, leading to suppuration; it may thus arise from disease of the temporal bone. 2. Affections of the heart or vessels originating septic materials which contaminate the blood, for example, endocarditis; softening of clots, especially in the veins; phlebitis. 3. Abscesses or gangrene in any part, either external to or within organs. 4. Ulceration of mucous surfaces, such as the gall-bladder or its duct, or the intestines. 5. Inflammation of a low type and attended with suppuration, implicating the pelvis of the kidney, the bladder, or the urinary passages. 6. Diseases characterized by external inflammation of an unhealthy character, leading to the formation of pus, especially certain varieties of erysipelas, variola, vaccinia in connection with revaccination, malignant pustule, glanders, carbuncles or boils; under this class may also be mentioned dissection and *post-mortem* wounds. 7. Low fevers occasionally, such as typhus, there being no evident local source of blood-poisoning. 8. Idiopathic pyæmia has been described, but it must be borne in mind that pyæmia may follow a very



slight injury in unhealthy subjects, and that there are many internal causes which might escape detection.

There is much controversy with regard to the immediate pathological cause of pyæmia and septicæmia; but this subject has already been sufficiently discussed under INFLAMMATION. It must suffice to say here that some infective poisonous material enters the blood, the chief views as to its nature being that it is merely a chemical fluid; that it consists of pus-elements; or that it is of the nature of living organisms, such as bacteria. Most probably there is no essential difference between pyæmia and septicæmia.

ANATOMICAL CHARACTERS.—It is highly probable that pyæmia may cause death without originating any characteristic *post-mortem* appearances. The morbid changes which it tends to produce may be summed up thus:—1. Intense congestion throughout the various organs and tissues of the body. 2. Hæmorrhages, in the form of petechiæ or vibices, in connection with the skin, mucous, and serous membranes; hæmorrhage into serous cavities; extravasations into muscles and among deep tissues; and apoplectic clots in the substance of organs, which are prone to undergo rapid destructive changes. 3. Acute inflammation in the solid organs, of a low type. 4. Formation of abscesses in these organs, often in considerable numbers, of good size and containing unhealthy pus; resulting either from hæmorrhagic clots, inflammation, or sloughing. 5. Gangrene of portions of organs. 6. Low serous inflammations, with a tendency to purulent effusion, which may be confined within adhesions; and to the production of unhealthy lymph. 7. Inflammation of mucous surfaces, leading to suppuration, ulceration, or sometimes to submucous abscesses or gangrene. 8. Severe inflammation of joints, with a great tendency to rapid formation of pus, and to destruction and disorganization of tissues, both within and around the joints, several of them being usually involved. 9. Inflammation and formation of abscesses in various parts of the body, namely, in the substance of muscles; in the cellular tissue, either superficial or deep; or sometimes in the skin itself, giving rise to pustules.

SYMPTOMS.—In many instances pyæmia reveals itself very insidiously, but its characteristic symptoms in an acute case are mainly these:—Rigors set in suddenly, being severe and prolonged in character, and repeated at irregular intervals. The temperature often rises rapidly to a high point, being usually very elevated throughout, but subject to marked and extremely irregular changes. Profuse sweating follows the rigors, in the intervals the skin being hot, dry, and harsh. There is a marked expression of illness, and a tendency to early prostration, combined with restlessness or heaviness. The skin soon assumes a sallow and yellowish aspect, and frequently considerable jaundice becomes evident; signs of congestion and petechiæ may often be seen, and sometimes sudamina, or a vesicular or pustular eruption. The digestive organs are usually much disturbed from the outset, there being anorexia, great thirst, nausea, and vomiting, frequently with a glazed or furred and irritable tongue, and in some cases fœtid diarrhœa. The pulse is frequent, feeble, and liable to rapid variations. Respiration is also hurried, and the breath has a peculiar sweetish odour in some cases. Albuminuria is not uncommon.

In a short time the symptoms and signs of the *local lesions* usually appear in various parts, these necessarily differing according to the structures which are affected. The joints are involved with considerable

frequency, becoming very painful and swollen. The further tendency is towards rapid and extreme prostration and adynamia, with low nervous symptoms; the face becoming pale and pinched; the heart's action exceedingly rapid, weak, irregular, and intermittent, as evidenced by the impulse, sounds, and pulse; the tongue brown and dry, with sordes on the teeth and gums; delirium, coma, or rarely convulsions setting in at last, with involuntary discharge of fæces and urine.

In some instances pyæmia is evidenced chiefly by its general symptoms; in others it runs a somewhat chronic course, and may then terminate in recovery. Some authorities regard certain diseases, which will hereafter be considered, as originating in a local pyæmia.

DIAGNOSIS.—It is important to distinguish pyæmia from various fevers and acute inflammatory affections which the disease may simulate; and to bear in mind its possible occurrence in connection with the morbid conditions mentioned under its ætiology. In some cases its course of temperature causes the complaint to resemble, and to be mistaken for ague.

TREATMENT.—The only chance of recovery in septicæmic and pyæmic conditions lies in the free and regular administration of nutritious food, *stimulants* and *tonics*, especially mineral acids, bark, quinine, and tincture of steel. *Antiseptics* have also been strongly recommended, and may be given along with the remedies just indicated. *Antipyretic* remedies may be needed, and one recently introduced, antipyrin, seems to be beneficial. The administration of boracic acid (gr. 5-15) with sulphuric ether has been specially advocated in the treatment of septic diseases. Local lesions must be attended to as they arise.

## CHAPTER XXIX.

### THROMBOSIS AND EMBOLISM.

By *thrombosis* is meant a local coagulation of blood during life, either within the heart or a vessel, a clot being thus formed, named a *thrombus*.

*Embolism* signifies the partial or entire plugging of a blood-vessel by a solid fragment or *embolus* conveyed from some distant part by means of the circulation, which has become impacted in the vessel.

#### I. THROMBOSIS.

ÆTIOLOGY.—The causes which tend to the formation of a thrombus may be thus stated:—1. Anything which impedes or retards the *blood-current*, for example, valvular and other organic diseases of the heart, pressure upon its cavities, or mere feeble cardiac action, such as is observed in fevers, or in various chronic wasting affections; diseases of the lungs impeding the pulmonary circulation; obstruction of a vessel as the result of constriction, pressure, or internal plugging, especially by an embolus; pressure upon the capillaries of a part; solution of continuity of a vessel; and dilatation of vessels, particularly in connection

with aneurisms, varicose veins, and distended venous plexuses. A generally feeble state of the circulation, and gravitation of the blood into dependent parts, may also contribute to the formation of a thrombus. 2. Conditions which give rise to an abnormal state of the *inner surface of the heart*, or of the *coats of the vessels*, for example, acute inflammation; fissuring of the surface; atheroma or calcification; projection of cancerous and other new formations into the interior of vessels; and the changes in their walls associated with surrounding gangrene or inflammation. 3. Certain conditions of *the blood*, namely, hyperinosis or increased tendency to coagulation of fibrin, such as may be observed in various acute inflammatory affections, and in pregnancy; probably pyæmia and allied states; and anæmia. Increased heat of the blood, either local or general, has been looked upon by Richardson and others as a probable cause of thrombosis. In many instances more than one of the above-mentioned conditions has contributed to the clotting process.

In further considering this subject it will be convenient to treat separately thrombosis of the *heart*, *pulmonary artery*, *systemic veins*, and *systemic arteries*.

**A. Cardiac Thrombosis.—Intra-cardiac Blood-concretions.**—Coagula may form in the heart after death; immediately before this event; or at some previous period more or less remote. These are distinguished from each other by their colour; consistence; mode of arrangement with regard to, and degree of adhesion with the cardiac walls; whether they are laminated or not; and whether they have undergone changes, either in the direction of organization or softening. It is desirable to make a few special remarks respecting the coagulation occurring shortly before death, as this is probably often a very dangerous event, and one which aids materially in bringing about a fatal result. It is observed in connection with organic diseases of the heart which obstruct the circulation, or which roughen the endocardial surface, but is most important in certain acute diseases, being then due to a condition of the blood favourable to coagulation, combined with a gradual loss of power in the cardiac contractions, in consequence of which the blood is not properly expelled out of the cavities of the heart, but is partially whipped up and its fibrin deposited. Obstruction in the lungs frequently contributes to the clotting process. Among the most important diseases in which this has been observed are croup, diphtheria, endocarditis, pneumonia, peritonitis, the puerperal condition, erysipelas, rheumatic fever, and pyæmia and its allies. Cardiac thrombosis is much more common, as well as more dangerous, in the right cavities than the left, but may occur on both sides of the heart. Usually the clots are decolorized, pale or yellowish, but not uniform throughout; firm and fibrinous; often laminated and fibrillated or granular; entangled among the muscular bands and tendinous cords; somewhat adherent to the surface, but separable without injuring the endocardium. Occasionally they soften in the centre. They may extend for a variable distance into the pulmonary artery or aorta, these portions frequently presenting marks of the valves, but they can be readily removed.

**SYMPTOMS AND SIGNS.**—The effects of cardiac thrombosis will vary with the rapidity of its production, its seat, and its extent. The dangers arising from this condition are that it causes obstruction to the circulation, and interference with the heart's action; that large portions may become detached, and lodged either in one of the main orifices or in an



arterial trunk; or that smaller particles may be separated, and conveyed to the smaller vessels as emboli. Probably also the products of the softening of a clot may poison the blood generally. Sudden extensive clotting is characterized by great disturbance of the cardiac action, which becomes irregular and very hurried, the pulse being extremely weak and small; a tendency to syncope; urgent dyspnoea; intense restlessness and anxiety; followed by signs of obstruction, either in the pulmonary or venous circulation or both, according to the situation of the clot. In less rapid cases the obstructive symptoms are chiefly observed, combined with more or less cardiac distress. Plugging of a cardiac orifice or great vessel by a clot may cause instantaneous death. The *physical signs* are tumultuous action of the heart, or great irregularity in the rhythm and force of the impulse; increased cardiac dulness, especially towards the right; obscurity and irregularity of the sounds, particularly the first; and alteration in murmurs, or the production of a new murmur, especially a pulmonary systolic bruit.

**TREATMENT.**—The measures required in this condition are absolute rest in the recumbent posture; the administration of *stimulants*, especially if there is a syncopal tendency, with as much liquid nourishment as the patient can take comfortably; the application of heat to the extremities; and free dry-cupping over the chest. Formerly alkaline bicarbonates were recommended to be freely given, along with carbonate of ammonia. Dr. Richardson has advocated the use of liquor ammoniæ (℥ x in iced water every hour) with iodide of potassium (gr. iii to gr. v every alternate hour), which he has found highly successful. In some instances digitalis might perhaps be of use, in order to excite stronger contraction of the heart; or gentle galvanism might be tried. All lowering measures are injurious; and opiates must be avoided.

**B. Thrombosis in the Pulmonary Artery and its Branches.**—Much discussion has been carried on relative to pulmonary thrombosis, especially as it occurs in women after parturition. Occasionally such persons die suddenly, and after death extensive clots are found in the pulmonary artery and its divisions, which some authorities believe to have been the cause of death, and to have formed there primarily and independently; others think that they are the result of embolism, fragments having become detached from clots in the veins or heart, and lodged in the pulmonary vessels as centres for coagulation; while still others regard death as being due to syncope, and the clot to be merely of *post-mortem* formation. The probability is that in most instances at all events embolism has something to do with the phenomena observed; and it is quite possible that a large mass may be sometimes carried into the pulmonary artery, so as to obstruct this vessel more or less completely.

Clots may be found only in the main pulmonary trunk and its larger divisions; in the smaller branches; or more or less throughout. According to the extent involved, and to the rapidity of coagulation, will the clinical phenomena vary. In some instances, as already stated, sudden death is believed to occur, preceded by a cry, this event following some effort after parturition. When only the smaller branches are affected there are no symptoms. If the clotting is more extensive, the symptoms are more or less dyspnoea and sense of want of air, with a feeling of oppression across the chest; evidences of cardiac embarrassment; faintness or actual syncope; much general distress and anxiety; followed by signs of overloading of the right heart, and general venous congestion.

These symptoms may temporarily subside, and then recur. It is highly probable that coagulation in the pulmonary vessels often adds to the danger of various diseases of the lungs and certain general diseases, and that it prolongs the duration of the former.

TREATMENT must be similar to that indicated for cardiac thrombosis.

### C. Thrombosis in the Systemic Veins.—Phlegmasia Dolens.—

The formation of clots in the systemic veins is by no means an uncommon occurrence, resulting from pressure, obstruction, feeble circulation, altered blood, and other causes; but it is most important in connection with the affection named *phlegmasia dolens*, in which as a rule the lower extremity is involved, the external iliac or femoral vein becoming obstructed on one or both sides, or sometimes the common iliac; occasionally the arm is affected. The formation of clots in the venous sinuses of the dura mater is also a very serious matter, which I have observed as the result of injury or disease affecting the cranial bones.

Phlegmasia dolens is most frequently associated with the puerperal state, coming on at a variable period after delivery, but it may also occur as a sequela of acute febrile diseases, especially typhus or typhoid fever, pleurisy, and pneumonia; and in the advanced stages of various chronic diseases, particularly phthisis, and malignant uterine disease. Different views are held as to the pathology of this affection. Some regard inflammation of the veins—*phlebitis*—as the primary lesion after parturition, this having extended from the uterine veins; others consider that the plugging is the first event, thrombosis resulting from unhealthy blood or embolism, the emboli frequently coming from thrombi in the pulmonary vessels, and that the inflammation is secondary. Certainly in the cases which have fallen under my observation, where phlegmasia has arisen independently of parturition, coagulation has appeared to be the primary morbid condition, and it has sometimes been brought about by an effort, especially when this was accompanied with temporary obstruction of the venous circulation in some part.

ANATOMICAL CHARACTERS.—A thrombus in a vein varies in its characters, according to its age and mode of formation. If a vessel is suddenly plugged, the clot is at first uniform throughout, soft, and red; but if this is gradually formed, it presents a stratified appearance, and the strata may consist of alternate layers of fibrin and white corpuscles. The thrombus increases in extent after its first formation, the degree of extension depending chiefly on the force of the circulation, and on the size and situation of the collateral branches. The thrombus undergoes the usual changes in colour and consistence; becomes adherent to the vessel, in which it frequently excites inflammation; and organization often follows, so that ultimately a fibrous cord alone remains, the vein being obliterated—*adhesive phlebitis*. Occasionally calcification takes place, a “phlebolith” being produced. In some instances the clot undergoes partial or complete softening or liquefaction, beginning in the centre, and a puriform fluid may result, consisting either of granules and molecules derived from the fibrin, with broken-down corpuscles; or, as some believe, of actual pus, derived from proliferation of white corpuscles. This is probably the pathology of so-called *suppurative phlebitis*. In this way the clot may be completely removed, or *canalization* takes place; in some instances substances are formed which contaminate and poison the blood, and which are of an infective character, supposed to be due to the presence of micrococci. Rarely the thrombus becomes

converted into a stinking, yellow-red, and highly irritating fluid, due to the entrance of the bacterium termo from a foul or gangrenous surface.

In *phlegmasia dolens* the smaller veins and lymphatics also become speedily involved, and more or less inflammation is in many cases set up in the skin and subcutaneous tissue, or even in the deeper structures.

**SYMPTOMS AND EFFECTS.**—The symptoms which may be associated with venous thrombosis are those due to:—1. Local irritation by the clot. 2. Obstruction of the vein, and consequent interference with the circulation. 3. Detachment of embolic fragments. 4. Constitutional disturbance, which is especially liable to result from the formation of septic matters and contamination of the blood. In *phlegmasia dolens* pain and tenderness along the veins and lymphatics of the thigh are usually complained of to a variable degree. One case which came under my notice, in which the disease followed typhus fever, commenced with a sudden intense pain at the moment of coagulation, which afterwards became most excruciating, being deeply situated in the course of the femoral vein, and of an aching or somewhat neuralgic character. In phthisis, also, phlegmasia is often the cause of much distress. The veins are in time felt to be thickened and firm, or cord-like, and the lymphatics are visible as superficial red lines. Soon there may be signs of venous congestion, but very speedily this is followed by a deadly whiteness of the limb—*white leg*—which swells from below upwards, chiefly on account of œdema, sometimes attaining an enormous size, and becoming tense or elastic, with a most uncomfortable subjective feeling of tightness. In course of time the superficial veins become enlarged and varicose, if the obstruction is not removed, and the leg often remains swollen for many months, or even permanently, the tissues being thickened and indurated. Rigors may be experienced at the outset, followed by pyrexia and much prostration.

**TREATMENT.**—In *phlegmasia dolens* the most efficient treatment is to support the patient by nourishing food and *stimulants*; to keep the leg perfectly at rest in a horizontal posture, or even a little raised; to use hot opiate or belladonna fomentations assiduously; and to give *sedatives* for the purpose of relieving pain, if required. Subsequently *tonics*, especially iron and quinine, good diet, and change of air are most beneficial; with douching, friction, and shampooing of the limb, this being either carefully bandaged, or an elastic stocking being worn. Great improvement may be effected, even after a long interval.

**D. Thrombosis in the arteries.**—This is almost always associated either with a diseased condition of the walls of a vessel; or with embolism. Its symptoms are merely those indicative of local obstruction of the artery involved. In medical practice arterial thrombosis is most important in connection with the brain, and it will be more particularly considered in relation to this organ.

## II. EMBOLISM.

It is only intended to give here a brief account of the principal general facts relating to embolism, this morbid condition being separately considered in connection with the organs in which it occurs.

**ORIGIN OF, AND ANATOMICAL CHANGES RESULTING FROM EMBOLI.**—The following are the principal sources of emboli:—1. Most commonly a thrombus or clot, either in a systemic vein; in the heart; in an artery,



especially in connection with aneurism; or, rarely, in the pulmonary vessels. A thrombus may give rise to an embolus, either in consequence of a portion of it becoming detached, or as the result of softening of the clot. 2. Vegetations about the valves and orifices of the heart, particularly if associated with obstructive disease, especially mitral stenosis. 3. Atheroma and calcification of the cardiac valves or of arteries, portions of the morbid materials becoming detached. 4. New growths, such as cancer, communicating with the interior of vessels. 5. Particles resulting from gangrene of organs. 6. Parasites which have gained access into the vessels. 7. Pigment granules. 8. Fat particles from bone-marrow.

According to the size and place of origin of the embolus will the seat of its arrest vary. It may be sufficiently large to plug an artery of considerable size, or is only stopped in the capillaries, or it may be small enough to pass through even the finest of these vessels. When originating in the venous portion of the circulation, emboli rarely pass beyond the pulmonary capillaries, but become impacted in them as a rule; those coming from the pulmonary vessels, the left side of the heart, or the arteries, lodge either in smaller arteries or in the capillaries, and especially in the vessels of the brain, spleen, and kidneys; those from the portal tributaries are generally arrested in the capillaries of the liver. An embolus is chiefly carried in the direction of the main stream, and it is somewhat influenced by gravitation, especially if of large size. The seat of impaction is usually at a bifurcation, or where the calibre of a vessel rapidly diminishes, owing to large branches being given off. The closure may be complete or only partial at first, and this depends not only upon the size and shape of the embolus, but also upon its nature and consistence; thus a soft fragment of a recent thrombus will adapt itself to the channel of the vessel, and thus completely close it, while a firm irregular fragment of calcareous matter may only partially obstruct it. Subsequently, however, *secondary thrombi* always form, both in front of and behind the embolus, so that ultimately the vessel is entirely blocked up to a variable extent. *Secondary emboli* are sometimes separated from the primary one or from the resulting thrombus, and pass on into smaller vessels.

Embolism causes local irritation of the vessel in which the embolus lodges, and if the circulation is not permanently disturbed this will be the only result, the embolus and its secondary thrombi being usually either absorbed or organized. Under certain conditions, however, namely, in organs where there is but one supplying artery, or only insignificant ones besides, or where the branches of the main vessels communicate only by capillaries (*terminal arteries* of Cohnheim), so that no collateral circulation can be established, other important changes follow, a *hæmorrhagic infarct* being formed. Accordingly to the experimental observations of Cohnheim, the arterioles supplied by the obstructed artery at first empty themselves, but as the result of venous pressure the blood regurgitates to fill the arterioles and capillaries beyond the obstruction, the arteries around the area dilating, and the capillaries becoming full of blood. The vascular walls also become impaired in their vitality, so that first fluid, and then white and red corpuscles pass through them, the escape taking place through the capillaries and veins, and there being no actual rupture of their walls. A *hæmorrhagic infarct* thus produced presents at first a dark central area of black-red colour, and likened to damson-cheese, due to stagnant

venous blood and red corpuscles cramming the tissues, surrounded by a zone of arterial redness. On section the infarct is generally seen to be wedge-shaped, with the base directed towards the surface of the organ, and slightly raised above it. Litten's experiments are opposed to Cohnheim's view of venous reflex causing infarction, and he attributes it to the continued supply of blood by small arteries, which become dilated. He also thinks that the diapedesis of the red corpuscles is merely due to stretching of capillaries and small veins by mechanical congestion. Litten states that in the case of truly terminal arteries no infarct occurs in the great majority of cases after obstruction.

In course of time an infarct undergoes certain changes, either becoming decolorized, more consistent, and organized; or a process of softening and molecular disintegration taking place, beginning in the centre of the infarct, and extending more or less to the circumference, the *debris* of the involved tissue being evident in the softened mass. Ultimately it may be absorbed; or remain as a caseous encapsuled mass; or become calcified. The nature of the changes after embolism will depend upon the degree to which the circulation is obstructed, and the difficulty or impossibility of establishing the collateral circulation; the tissue affected; the size of the infarct; and the nature of the embolus. If this has septic properties, as is the case when it comes from a gangrenous part, it sets up rapid and violent inflammation, ending in speedy disorganization, with the production of a puriform material, constituting an *embolic abscess*, which is surrounded by hyperæmia.

The effects of an embolus, as regards the part supplied by the vessel which is blocked up, are similar to those of obstruction from any other cause, namely, anæmia, atrophy, softening, fatty degeneration, or actual gangrene. In the arteries of the limbs embolism may lead to the formation of an aneurism, and it is now generally believed that cerebral aneurisms in young people are often due to this cause. The formation of the aneurism is supposed to be chiefly due to inflammatory softening of the walls of the artery, resulting from the irritation of the embolus.

The most important seats of embolism are the vessels of the lungs, brain, spleen, kidneys, and heart. Petechial spots on the skin, or on mucous and serous membranes, are sometimes due to this cause. A very interesting case came under my notice in which sudden embolism occurred in connection with the main vessels of the fore-arm.

**SYMPTOMS.**—The clinical phenomena of embolism necessarily vary greatly according to the vessels affected; the rapidity and degree of obstruction; the characters of the embolus; and other circumstances. It need only be stated here in a general way that the early symptoms, if any, are those significant of sudden or gradual obstruction of the vessels supplying some particular organ or part; followed by those indicating the local effects of the embolus, and in some cases by evidences of septicæmia. Embolism affecting individual organs is considered in the chapters devoted to the discussion of their several diseases. Fatty embolism is important in some injuries and diseases of bones, particles from the marrow gaining access into the circulation; and this form of embolism has also been regarded by some observers as a cause of death in diabetes.

**TREATMENT.**—There is no special treatment applicable to embolism. Attention must be directed to the organ involved, the ill-effects resulting from the morbid process being obviated by rest, and by the adoption of any other measures which may be indicated in each particular case.

## CHAPTER XXX.

## DISEASES OF THE ABDOMEN.

## PHYSICAL EXAMINATION OF THE ABDOMEN.

BEFORE proceeding to the consideration of the diseases of the abdomen and its contained organs, it is necessary to point out the various methods of *physical examination* which may be employed in their investigation; and to indicate the information which each of them is capable of affording. In order to make this examination satisfactorily, the abdomen should be properly exposed, and the patient placed in an appropriate position. The best posture ordinarily is on the back, with the head and shoulders considerably raised, and the knees and thighs bent, so that the abdominal muscles may be relaxed; however, it is requisite in many cases to make the patient assume other positions, such as lying on either side or on the face, or kneeling supported on the hands. The patient should be desired to breathe deeply, or the attention may be occupied with conversation, so that a state of contraction and tension of the abdominal muscles may be avoided, which is very apt to be produced. The examination should be carried out carefully and thoroughly, and not uncommonly it has to be made on more than one occasion, before a satisfactory conclusion can be arrived at.

The following outline indicates the different modes of examination which might be required in the investigation of any particular case, though it does not often happen that they are all called for in the same individual. Some of them are similar to those employed in the exploration of chest-affections, though their relative value is very different; others are peculiar to the examination of the abdomen.

**I. Inspection.**—By this method is ascertained:—1. The state of the superficial parts, namely, the integuments, superficial veins, and umbilicus. 2. The general shape and size of the abdomen; as well as any local alteration in these respects. 3. The characters of the abdominal respiratory movements. 4. The presence of any visible pulsation. 5. Certain movements, such as those due to flatus; to fluid, when the position of the patient is altered; or to the presence of a foetus.

**II. Application of the hands, Palpation, or Manipulation.**—If properly carried out, this becomes one of the most valuable methods of examination applicable to diseases of the abdomen; but it requires considerable practice, and manipulation has to be employed in different ways in different cases. *Palpation* reveals:—1. The condition of the abdominal walls, as regards amount of fat, œdema, and the state of the muscles. 2. The shape and size of the abdomen more accurately than inspection. 3. The sensations conveyed as regards mobility of the abdomen as a whole, degree of resistance, consistence, fluctuation, regularity and smoothness or the reverse, over the surface generally, and over its different parts. 4. The existence of any enlarged organ or tumour, as well as its position and characters. 5. The extent of the respiratory movements, and their influence upon any tumour which may be present. 6. The situation and characters of any pulsation. 7. The presence of friction-fremitus developed during the act of breathing.



8. Any movements set up within the abdomen, such as gurgling from accumulation of gas in the intestines, or foetal movements.

**III. Mensuration or Measurement.**—Merely an ordinary single and double measuring tape of sufficient length are required for measurement of the abdomen, and this affords exact information as to its size; and as to the diaphragmatic respiratory movements. It is particularly valuable as showing the progress of many cases, with the effects of treatment. The measurements ordinarily required are:—1. *Circular* in different parts, but especially a little above and below the umbilicus. 2. *Semicircular*, so as to compare the two sides. 3. *Local*, namely, from the umbilicus to the ensiform cartilage; to the pubes; and to the anterior superior spine of the ilium on each side.

**IV. Percussion.**—*Mediate* percussion must usually be practised in connection with the abdomen. A modification of this method is employed in the production of what is termed *hydatid-fremitus* or *vibration*, which is elicited by applying three fingers of the left hand firmly over certain cystic tumours, and striking the middle finger suddenly with the point of the right middle finger. Another method consists in applying the fingers of one hand over one side of the abdomen, and tapping or filliping the opposite side with those of the other hand, which is the usual plan adopted for producing *fluctuation*.

The objects for which percussion of the abdomen is practised are:—1. To bring out certain sounds. 2. To realize certain sensations communicated to the fingers, especially the *degree of resistance*; *hydatid-fremitus*; and *fluctuation*. The sounds elicited may be grouped under the terms *dulness* and *tympanitic sound*, these necessarily varying in their exact characters. In the normal state they are both met with over different parts of the abdomen, according to the organ which corresponds to the point percussed, and by this mode of examination the exact position and limits of most of the organs can be marked out. The deviations from health, as regards sound, which may be observed, are:—(i.) Excess in the intensity, clearness, or extent of the tympanitic sound. (ii.) Dulness, either too extensive, or in unusual positions. The last is the deviation which requires most attention, and when any abnormal dulness is detected, it is necessary to make out carefully:—*a.* Its site, exact limits, and shape. *b.* Whether it differs according as superficial or deep percussion is made. *c.* If it is influenced by posture; the act of breathing; pressure; or manipulation: while in some doubtful cases it may be requisite to observe the effects upon the percussion-sound of taking food or drink; of the act of vomiting; the use of an enema; or the removal of urine by a catheter. With respect to *resistance*, by noticing its degree an accumulation of fluid may be distinguished from a solid mass, and the actual density of the latter can to a great extent be realized; the sensations on percussion are likewise useful in separating flatulent distension of the abdomen from enlargement due to the presence of fluid.

*Hydatid-fremitus* is a peculiar vibrating or trembling sensation, produced in the manner already described, and formerly supposed to be characteristic of hydatid tumours, but it can be brought out in connection with any large cyst which has thin and tight walls, and which contains a fluid of watery consistence, and may even be simulated by localized collections of fluid in the peritoneum.

*Fluctuation* indicates the presence of fluid within the cavity of the abdomen, and it is needful to observe the degree of facility of its pro-

duction, and its distinctness; the part of the abdomen over which it is felt; and whether it is influenced by change of posture. Information is thus obtained as to the quantity of fluid, its freedom or limitation by cysts or adhesions, and its consistence. It must be borne in mind that a relaxed or very fat condition of the abdominal walls may give rise to a sensation simulating fluctuation.

**V. Auscultation.**—This is not nearly so useful a mode of examination in connection with the abdomen as in the case of the chest, and it need scarcely be mentioned that the intervention of a stethoscope is always advisable when practising it. Auscultation generally only gives negative information, except in cases of pregnancy, but sometimes by its aid certain positive signs are detected, namely:—1. Friction-sound or grating, heard during the act of breathing, and due to exudation on the peritoneum, or to roughness of the surface of certain organs. 2. Murmurs in connection with aneurism, with regard to which it is requisite to notice their site, intensity, extent of conduction, synchronism, pitch, and other characters; as well as any effects produced upon them by pressure, or by change of position. 3. A murmur over the aorta, or over one of the common iliac arteries, due to pressure, such as that of a tumour. 4. Unusual conduction of the heart-sounds over the abdomen. 5. Sounds due to the movement of flatus in the intestines; or to the falling of food or liquid into the stomach, when swallowed. 6. Fluctuation or splashing-sound, elicited by shaking the patient, and indicating the presence of both air and fluid. 7. Murmurs and sounds heard in connection with the pregnant uterus.

In the majority of cases the methods of examination thus far considered are sufficient for establishing a diagnosis in abdominal diseases; but there are others which might be called for in doubtful cases, and from which much aid may be derived. At present it must suffice to enumerate them, as illustrations of their usefulness will be given hereafter.

**VI. Examination directed to the Alimentary Canal.**—This generally includes:—1. The passage of a probang into the stomach, the end being felt through the abdominal walls, which helps in making out dilatation of this organ. 2. Examination of materials discharged from the stomach, and it has even been recommended in certain cases to make use of the stomach-pump, and thus obtain some of the food at different stages of digestion. 3. The use of purgatives and enemata, in order to clear out the bowels, which should never be neglected in doubtful cases, the effects being noted; or the injection of a quantity of water or gas *per anum*. 4. Examination of the anus and rectum, by inspection, the speculum being employed if required; by the aid of the finger or hand; or by the bougie. 5. Examination of the stools.

In order to test the efficiency of the pyloric orifice, it has been recommended to administer first a solution of carbonate of soda, and afterwards one of tartaric acid, and to determine by percussion whether the carbonic acid gas thus generated passes readily through the pylorus, or remains in the stomach.

**VII. Examination per Vaginem**, as described in obstetric works.

**VIII. Examination of the Bladder and Urine.**—The use of the catheter must always be thought of, but especially when there is any reason to suspect an accumulation of urine in the bladder. It may also be requisite to examine this organ with the sound. In all cases it is absolutely necessary to *examine the urine* carefully.

**IX. Exploratory puncture with a small Trochar, or with the Aspirateur.**—This is done for the purpose of determining the presence and nature of fluid within the abdomen.

**X. Administration of Chloroform.**—This may be required in order to aid in carrying out other modes of examination; while it at once determines the nature of the so-called *phantom-tumour* of the abdomen.

Physical examination of the abdomen is chiefly useful in the investigation of the following abnormal conditions:—1. General enlargements. 2. General retraction or depression. 3. Local enlargements, or so-called *tumours*. 4. Diminution in the size of organs; as well as certain changes in their physical characters, either with or without alteration in dimensions, for instance, cirrhosis of the liver. 5. Pulsation, associated or not with any change in shape or size. 6. Interference with the abdominal respiratory movements from various causes.

*General and local abdominal enlargements* are of very common occurrence, and therefore, although this will involve some repetition, it may perhaps be of service to indicate specially the course of investigation to be pursued, and the points to be observed, in conducting physical examination with the view of arriving at a diagnosis as to the cause of any such enlargement.

**I. General enlargements.**—1. Examine as to the state of the abdominal walls, by inspection and palpation, paying particular attention to the characters of the umbilicus. 2. Ascertain the degree of enlargement, and its exact form, by inspection, palpation, and measurement. 3. Note the extent of the abdominal respiratory movements, by the same methods; and observe whether they give rise to any sensation of fremitus. 4. Manipulate thoroughly over every part of the abdomen, in order to determine the sensations conveyed as to smoothness and regularity, amount of resistance, consistence, gurgling, &c. 5. Observe specially if there is any feeling of fluctuation, as well as its seat, extent, and facility of production. 6. Percuss carefully, noting the sounds elicited over the abdomen in different parts, and the sensations conveyed to the fingers during the act. Sometimes it is requisite to examine for hydatid-fremitus. 7. Apply the stethoscope, chiefly to ascertain whether any kind of friction-sound can be heard during the act of breathing; if there are any of the sounds usually observed in connection with a pregnant uterus; or if a pressure-murmur is audible over either iliac artery. 8. Having examined thus far as the patient is lying in the ordinary position, it is then necessary to observe the effects of various changes of posture, especially as regards the shape of the abdomen; the percussion-sounds; and any fluctuation which may be present. 9. If after this a satisfactory diagnosis cannot be made, it will be requisite to have recourse to the other methods of examination mentioned, particularly examination by the rectum or vagina; and the employment of the aspirateur or exploratory trochar. Of course the urine should always be thoroughly tested.

**II. Local enlargements.**—Manipulation is by far the most important mode of examination which can be employed in the investigation of localized abdominal tumours, and therefore it is specially necessary to educate the sense of touch for these cases. 1. Any local change in the skin, limited œdema, or enlargement of veins must be noted. 2. A cursory examination is desirable, in order to determine if there is but one tumour or more; and, in the latter case, whether they are separate or connected. The further remarks will apply to each enlargement,



should there be more than one. 3. Ascertain the precise situation of the tumour, paying particular attention to the following points:—*a.* Whether it extends into the pelvis. *b.* If it is median, or occupies one or other side, and to what extent. *c.* If it can be traced within the margin of the thorax; and if it enlarges the lower part of this cavity, or alters the intercostal spaces in any way. *d.* Whether it corresponds to, or can be made out to be part of either of the organs. *e.* Its depth, noting whether it lies in the abdominal walls; within the cavity of the abdomen near the surface; or deep down near the spine. 4. Mark out the dimensions and shape as nearly as possible, observing the form of the margin; if this is well or ill-defined; and if the outline corresponds to that of any abdominal organ. 5. Feel carefully over the surface and margins, in order to determine whether they are smooth, granular, nodular, or lobular, noticing the characters of any prominences which may exist; and at the same time try to realize the consistence, which may be more or less hard and firm, elastic, doughy, fluctuating, &c. It is important to observe whether the sensations are uniform or not over the enlargement; and if any change is produced by pressure and manipulation, or if any gurgling or grating is thus elicited. 6. Ascertain whether the tumour is movable or fixed, as well as the degree of mobility, both by manipulation, and under the influence of the respiratory movements. 7. Should there be any pulsation or thrill, the exact seat and characters of each must be noted. 8. Percussion is, of course, most valuable in bringing out sounds and tactile sensations, and in many cases the precise limits and characters of an enlargement can only be made out in this way. It is important to notice whether the results of percussion are uniform over its entire surface or not. 9. Auscultation is sometimes useful, especially in order to investigate murmurs, which may be associated with the tumour itself, or be the consequence of its pressure upon an artery. 10. The posture must be altered, as in the case of general enlargements, and the effects observed, as regards the site of the tumour; its percussion-sound; or any changes affecting fluctuation, pulsation, or murmur, should either of these signs be present. 11. Without again recapitulating the more unusual methods of examination, it may be stated that either or all of them may be called for in doubtful cases, and under any circumstances it is most desirable that the bowels should be thoroughly emptied by the use of purgatives and enemata, as collections of fæces may cause much obscurity, and not uncommonly simulate tumours of a very serious nature.

---

## CHAPTER XXXI.

## DISEASES OF THE PERITONEUM.

## 1. ACUTE PERITONITIS.

**ÆTIOLOGY.**—Peritonitis may be divided into certain varieties according to its mode of causation, namely :—1. **Traumatic.** This form may arise from mere external injury to the abdomen; penetrating wounds; or rupture by violence of internal organs. It must be remarked, however, that the danger of peritonitis from direct injury has been much exaggerated, as the membrane is often considerably injured in operations without any particular harm resulting. 2. **Perforative.** The special causes of this important variety of peritonitis will be separately considered. 3. **Irritative.** In not a few instances peritonitis arises from some local irritation, being then either limited, or spreading throughout the sac. Thus it may be associated with diseases of organs; hernia or intestinal obstruction; ulceration of the bowels; morbid deposits, especially acute miliary tubercle; inflammation and abscess of the lacteal glands; or it may be the result of extension of inflammation through the diaphragm from the pleura or pericardium. The source of local irritation may be very obscure, requiring to be carefully sought after. 4. **Secondary,** from blood-poisoning. Peritonitis from this cause is especially important in connection with the puerperal state and Bright's disease. It may also be met with in the course of small-pox, typhoid fever, pyæmia, erysipelas, glanders, gout, rheumatic fever, and other diseases. In some of these cases it has been supposed to be metastatic. 5. **Idiopathic.** Under this head are included those cases of peritonitis which cannot be traced to either class of causes above mentioned, but which are attributed to cold, over-eating or drinking, and other injurious influences. Many doubt the existence of this variety, but exceptional cases come under observation which certainly seem fairly to belong to this class. 6 **Puerperal peritonitis,** which can most probably be conveyed by contagion.

*Predisposing causes.* Children are only very rarely attacked with acute general peritonitis, and in such subjects it is usually associated with some exanthematous fever. Certain forms of blood-poisoning, but especially that connected with renal disease, are very favourable to the occurrence of peritonitis from any slight irritation. Puerperal peritonitis may assume an epidemic form.

**ANATOMICAL CHARACTERS.**—The special features in the morbid anatomy of peritonitis which seem worthy of notice, as distinguishing it from other serous inflammations, are as follows :—The vascularization is often very intense, and is most marked where the coils of intestines touch. The subserous tissue and the muscular coat of the alimentary canal are usually much sodden, infiltrated, and softened. The lymph, though not uncommonly tolerably firm, matting together the coils of intestines, yet appears to be more frequently of a soft, non-organizable nature than in other serous inflammations, and flakes of it are always detached in

abundance, or even gelatinous-looking masses of considerable size may be observed. In some instances it presents a greasy appearance. The effusion is as a rule comparatively small in quantity, and the explanation of this may be that the intestines are generally so distended with gas that they prevent much accumulation of fluid. This fluid is always more or less flaky and turbid, and not uncommonly presents an almost purulent appearance, occasionally being actually purulent, especially in puerperal and other low forms of peritonitis. Sometimes there is an admixture of blood. Much foetid gas is frequently present in the peritoneal sac, and in certain cases foreign materials are found there. Gangrene is occasionally observed. The morbid products of peritonitis are in some instances of the most virulently septic character, and the introduction of the minutest quantity into the system is highly dangerous; hence special care should always be taken in conducting a *post-mortem* examination in cases of this disease.

According to the extent of the inflammation, peritonitis is named *general* or *local*, the former term merely indicating that it is extensive, for the entire surface is but rarely involved. The local varieties are named according to the part affected, such as *parietal*, *hepatic*, *omental*, or *nephritic*.

Should recovery take place, thickenings, agglutinations, or bands of adhesion form, which subsequently may prove highly injurious.

**SYMPTOMS.**—Peritonitis presents considerable variations in its clinical history, but it will be convenient first to describe a typical case, and then to point out the principal forms which call for special notice.

The *onset* or *invasion* of the disease is usually definite and marked, being attended with distinct, and often severe and repeated rigors. These are soon followed, sometimes accompanied, or rarely even preceded by local symptoms referable to the abdomen and its contents; with evidences of constitutional disturbance. Some of the main symptoms are due to irritation of organs excited by the inflamed peritoneum; or to paralysis of the muscular coat of the hollow viscera, especially of the stomach and bowels. The symptoms may therefore be considered as *local* and *general*.

*Local.*—Pain over the abdomen is in most cases a prominent symptom in peritonitis. It usually begins locally, especially below, but may ultimately extend over the entire surface, though it is frequently more marked in some particular region. Its intensity and characters vary much, but it is generally very severe, sometimes agonizing; and hot, burning, shooting, or darting in character. Any disturbance of the parts increases the pain considerably, such as that induced by change of posture, a deep breath, coughing, vomiting, defæcation, or even the movement of flatus in the intestines. There is extreme tenderness, especially on making deep and limited pressure, but in some cases even the weight of the bed-clothes cannot be borne. The alimentary canal is gravely affected, as evidenced by a very small, irritable, red tongue, slightly furred, and tending to dryness; complete loss of appetite, with great thirst; nausea, and vomiting of everything swallowed; and usually absolute constipation.

*General.*—The appearance of the patient is often highly characteristic. The face, pale or flushed, presents an expression of evident suffering and grave constitutional disorder, combined with anxiety, the features being drawn and pinched. There is much prostration, with general uneasiness and restlessness, but though the arms may be thrown about,



the patient keeps the body perfectly still, on account of the pain which movement causes, and instinctively assumes a characteristic posture, with the view of relaxing the abdominal muscles, namely, lying on the back, with the head and shoulders raised, the thighs bent, and the knees drawn up and flexed.

Pyrexia is usually, but not always, present to a very marked degree; but there is no regularity in the temperature. The pulse is increased in frequency, ranging from 100 to 150 or more, small, sharp, often hard, and wiry or thready; in bad cases it tends to become extremely frequent, feeble, and irregular. Its characters are clearly revealed by the sphygmograph. The blood is, in many cases, highly hyperinotic. Respiration is hurried, but shallow; while persistent hiccup causes much distress in some cases. The urine is markedly febrile, and not uncommonly contains albumen. It may be almost suppressed or retained. Micturition is sometimes very frequent. As a rule no particular cerebral symptoms are observed, except headache and sleeplessness; in bad cases, however, low muttering delirium may set in, or in puerperal peritonitis the delirium is sometimes of a wild type.

**Physical examination** of the abdomen reveals some important signs in cases of acute peritonitis, namely:—1. *Tympanitic distension*, often extreme. 2. Absolute cessation of all *abdominal respiratory movements*. 3. Occasionally *friction-fremitus* or *sound*, if the patient can be made to breathe deeply, especially over the liver, due to the presence of lymph. 4. Usually signs of a certain amount of fluid, namely, *dullness* in dependent parts; with in some instances a sense of *fluctuation*, movable with change of posture.

**COURSE AND TERMINATIONS.**—Peritonitis is a very fatal disease, death being usually preceded by extreme prostration and collapse, as indicated by the aspect of the features; cold, clammy sweats; coldness of the extremities; and an extremely rapid, feeble, and irregular pulse. The pain in the abdomen often ceases, sometimes suddenly, and the tympanites may disappear. Sometimes large quantities of a dark fluid containing altered blood are expelled from the stomach and bowels, without any effort. Low nervous symptoms usually set in, but the intellect may remain clear almost to the last. Occasionally death seems to result from asphyxia or coma. Should recovery take place, there is a gradual subsidence of the symptoms, and among the chief signs of improvement are a favourable change in the expression; increase in the force and fulness of the pulse, with a more satisfactory sphygmographic tracing; relief of constipation; and increase in the quantity of urine. Peritonitis is said to terminate occasionally by *crisis*, with critical discharges, but this must be an extremely rare event.

**VARIETIES.**—The special forms of peritonitis requiring notice are as follows:—1. **Perforative**, which will be separately considered. 2. **Latent**. Cases are sometimes observed in which there is extensive peritonitis without any symptoms at all; or none of any definite character. This may be due to the mental condition of the patient, but not always. A remarkable illustration of this variety came under my care at University Hospital, in which there were absolutely no local signs of peritonitis, and yet this was the only lesion found after death. 3. **Adynamic**. Here there is a rapid tendency towards the typhoid condition, with a dry and brown tongue, sordes on the teeth, and low nervous symptoms. 4. **Erysipelatous**. Puerperal peritonitis is the best illustration of this form, in which the inflammation is very extensive, intense, and

rapid in its progress, the products being remarkably non-plastic, consisting chiefly of a purulent-looking fluid, often very abundant. The symptoms are proportionately severe, and of a low type. This variety is also observed after low fevers sometimes; and in cases of pyæmia.

**5. Local.** When peritonitis is limited to, or more marked over some particular organ, the pain may be localized, and symptoms connected with this special organ become prominent. When the parietal peritoneum or great omentum is involved, there is extreme superficial pain, with marked tenderness. **6.** The symptoms of peritonitis may be modified by **complications**. Of these the most important is *muco-enteritis*, which may give rise to diarrhœa instead of constipation.

**DIAGNOSIS.**—It is important to bear in mind the possibility of peritonitis being *latent*, should there be any condition present likely to originate this disease. The principal affections from which it has to be distinguished are cramp or colic; muscular rheumatism of the abdominal walls; enteritis; enteralgia and other neuralgic painful affections within the abdomen; the passage of a gall-stone; and certain cases of hysteria, attended with tympanites and other local symptoms closely simulating peritonitis. The diagnosis is founded on:—**1.** The *history* of the case, as to the *exciting cause* and *mode of onset*. **2.** The *aspect* of the patient, which usually suggests grave constitutional disturbance in peritonitis, while there is no evidence of hysteria. **3.** The *posture* of the patient, and state of absolute rest as regards the body. **4.** The *local symptoms*, especially the severity and characters of the pain; marked tenderness, which is not merely superficial, but also deep; urgent vomiting; and obstinate constipation. **5.** The *physical signs*, as evidencing much tympanites, with a certain amount of fluid, and possibly the presence of lymph. **6.** The existence of more or less *pyrexia*, often accompanied with special characters of the pulse, tongue, and urine.

**PROGNOSIS.**—Peritonitis is always a highly dangerous affection, but its gravity differs materially according to its cause. *Perforative* peritonitis is extremely fatal; and next in order of danger come the *puerperal* form, and those cases which are associated with pyæmia and other forms of blood-poisoning. *Traumatic* and *local* varieties are much less serious. Among the unfavourable symptoms may be mentioned typhoid and low nervous phenomena; and great dyspnœa. The condition of the pulse, especially as revealed by the sphygmograph, will assist in determining the prognosis. The duration of fatal cases may vary from twenty-four or forty-eight hours to three or four weeks, but they do not often extend beyond a week.

**TREATMENT.**—No exact rules for the treatment of peritonitis can be laid down, as the management of this disease has to be materially modified in different cases, and it will be only practicable to indicate the general principles which are to be followed, and the main remedies for carrying them out. It must be premised that any cause which is setting up or intensifying peritonitis must be at once removed, if possible, and should be carefully sought for in doubtful cases, as, for instance, an intestinal hernia. The main principles of treatment are:—**1.** To procure rest for the affected parts. **2.** To subdue the inflammation, and promote the removal of the inflammatory products. **3.** To sustain the strength of the patient. **4.** To treat various symptoms as they arise.

The *removal of blood* by venesection, or by the application of a large number of leeches over the abdomen, is a measure which is very commonly adopted in the treatment of peritonitis, and certainly it seems to

be more serviceable in this than in other serous inflammations; but at the same time there are very many cases for which bleeding is by no means suitable, so that all the circumstances of each individual case must be carefully considered before having recourse to this treatment. It is when extensive peritonitis occurs in a healthy, strong, and plethoric subject that withdrawal of blood is indicated, and then only in the early stages of its progress. When the disease is associated with blood-poisoning, or with a low condition of the system; if the patient is weak, either constitutionally or from any pre-existing illness; or if the inflammatory process is far advanced, it is decidedly injurious to take away any blood. The balance of evidence is opposed to bleeding in puerperal peritonitis. The application of leeches is much preferable to venesection, the number employed varying from 10 to 20, 30, or even more in appropriate cases.

Mercurialization, by means of calomel administered with opium, is another very common mode of treatment adopted in cases of peritonitis, but it appears to me to be as useless, or even injurious, in this as in other serous inflammations. Opium is a remedy of the utmost importance. It not only relieves symptoms, especially pain and vomiting, but also prevents the peristaltic action of the bowels, and thus contributes greatly to the maintenance of rest. It is best given in the form of pill—gr.  $\frac{1}{2}$ —ij, repeated every two, three, or four hours, according to circumstances. If there is renal disease, opium can only be employed very cautiously. Morphia, either administered as a pill—gr.  $\frac{1}{4}$ — $\frac{1}{2}$ , or by subcutaneous injection, is also highly valuable in some cases. When the stomach is extremely irritable, tincture of opium may be introduced by enema. Other *anodynes* may be given when opium is inadmissible. Quinine in full doses has been recommended, and in low forms of peritonitis it might probably be administered with much advantage along with opium. Aconite and digitalis have also been employed in some forms of peritonitis.

The *diet* requires the most careful attention in acute peritonitis. Only liquids should be given, cool or even cold, and they must be administered in small and definite quantities, at stated intervals. Frequently abundant nutriment is required, especially in the form of milk and well-made beef-tea. In many instances also *alcoholic stimulants* are needed, and in low forms of peritonitis these are the chief remedies on which reliance can be placed. The sucking of ice is highly to be commended; or small quantities of iced drinks might be allowed. In many cases it is requisite to have recourse to nutrient enemata, especially when the stomach is very irritable.

*Local applications* over the abdomen are of decided value in peritonitis. The most serviceable are hot linseed-meal poultices, not too heavy, sprinkled over with laudanum, and changed frequently; and warm anodyne or turpentine fomentations, for applying which spongio-piline is useful. Sinapisms may also prove beneficial. Some authorities recommend the employment of cold compresses, frequently changed. In tubercular peritonitis Dr. McAll Anderson uses constant applications of flannels dipped in iced water. In the more advanced stages a blister might be beneficial in some cases. It is often desirable to employ some apparatus for the purpose of keeping off the weight of the bed-clothes from the abdomen.

The chief *symptoms* requiring attention in acute peritonitis are pain; vomiting; tympanites; constipation or diarrhoea; urgent dyspnoea;



and those indicative of adynamia. The remedies already considered will assist in relieving most of these symptoms. Sickness should be combated by means of small quantities of an effervescent mixture with hydrocyanic acid and morphia; soda-water and milk; the sucking of lumps of ice; or creosote in drop doses. Tympanites is best relieved by enemata of turpentine; or by the passage of a long tube *per rectum*. Puncture of the colon with a minute trochar or the aspirateur may be had recourse to in extreme cases, if other measures fail. With regard to constipation, in some instances it is desirable to endeavour to clear out the bowels at the outset by a full dose of calomel, and afterwards to employ enemata; but when there is perforation, on no account must the bowels be disturbed. Diarrhœa is best treated by enemata of starch and tincture of opium (m̄ xv-xxx). Dyspnœa is usually relieved by removing the tympanitic condition. Adynamic symptoms call for ammonia and bark, ether, or turpentine internally; along with abundance of alcoholic stimulants, and nutritious liquid food. In extreme cases ether may be subcutaneously injected.

## II. CHRONIC PERITONITIS

ÆTIOLOGY.—Chronic peritonitis is observed:—1. As a sequel of one or more acute attacks. 2. After repeated paracentesis for ascites. 3. In connection with chronic diseases of certain abdominal organs, such as cirrhosis or cancer of the liver, or chronic ulcer of the stomach or intestines. 4. Associated with some diathesis, especially when this leads to a morbid formation in the peritoneum, such as cancer or tubercle; chronic peritonitis may also arise in connection with Bright's disease, and possibly from rheumatism.

ANATOMICAL CHARACTERS.—These necessarily vary greatly in different cases, but in a general way they may be described as thickening of the peritoneum, sometimes carried to an extreme degree; adhesions, in the form of bands, or of extensive matting together of the organs; accumulation of more or less fluid, which ranges from mere serum to actual pus, or contains an admixture of blood, and is often confined in loculi limited by the adhesions. In some cases large masses of organized lymph are seen; and much pigment is often present. Caseous degeneration may have taken place in parts; or cancer or tubercle may be evident.

SYMPTOMS.—In some cases there are no clinical evidences of chronic peritonitis, or only such as are very obscure; in others merely *physical signs* are observed. When present the symptoms include various subjective sensations in the abdomen; disturbance of the alimentary canal; sometimes evidences of pressure; with generally more or less constitutional disorder. Abdominal uneasiness or more or less actual pain may be experienced, which, however, is never severe, liable to come and go, often colicky, and increased by shaking the body. Sometimes there is a sense of local soreness or heat. Tenderness is common, being frequently more marked in particular spots. The digestive organs are generally disturbed, but it is often difficult to say how far this is due to the peritonitis or to other causes. This affection does, however, tend to give rise to constipation, and the bands of adhesion may lead to absolute intestinal obstruction. In chronic tubercular peritonitis diarrhœa is common, owing to intestinal ulceration. Occasionally jaundice, ascites, or anasarca of the legs are observed, as the result of pressure on the

common bile-duct, or on certain veins. More or less emaciation; a dry and harsh skin; occasional pyrexia, tending towards a hectic type; and other general symptoms often indicate constitutional disturbance, but it is probable that these are in most cases chiefly due to the condition with which the peritonitis is associated.

**Physical examination** often yields important information in cases of chronic peritonitis. 1. The abdomen is liable to be *enlarged*, and this may have first attracted the patient's attention. The enlargement is never very great; and it is usually regular in shape, though not always quite symmetrical. 2. The *sensations* on palpation are seldom uniform over the entire surface. Fluctuation may be detected in parts, but only indistinctly; while it is often very limited, or is felt in unusual situations, owing to the fluid being enclosed in locular spaces. In other regions there may be a more firm and solid sensation, or even distinct growths may be felt sometimes. The abdomen may be curiously movable as a whole. 3. *Dullness* is frequently very extensive, owing to the arrangement of the fluid, and it may lie chiefly in front. In some instances tympanitic and dull sounds are heard over contiguous and irregular spots. There may be a sense of much *resistance* on percussion. 4. *Friction-fremitus* and *sound* can sometimes be detected. 5 *Change of posture* frequently produces little or no effect, on account of the fluid being loculated.

**TREATMENT.**—The main indication in most cases is to treat the *constitutional state* with which chronic peritonitis is associated, by means of cod-liver oil, *tonics*, mild ferruginous preparations, light nutritious diet, a suitable climate, and proper hygienic conditions. Iodide of potassium or iodide of iron may be tried internally, with the view of removing the inflammatory products; as well as *local* counter-irritation over the abdomen, especially by means of iodine liniment or ointment. This region should be covered with cotton-wool and well bandaged. I have known considerable benefit follow in simple cases from systematic pressure, obtained by carefully bandaging the abdomen. Pain and constipation must be relieved by the usual means, but caution must be exercised in giving opium, and also in administering strong purgatives. Hot air or vapour baths may be useful if the peritoneum contains much fluid. It may become necessary to have recourse to paracentesis.

### III. MORBID GROWTHS IN THE PERITONEUM.

The most important morbid formations met with in the peritoneum are *tubercle* and *cancer*. *Hydatids* are occasionally found; and very rarely tumours of other kinds. The folds of the peritoneum, especially the omentum, frequently enclose a great quantity of fat.

*Tubercle* occurs in the peritoneum, either over limited patches corresponding to intestinal ulcers; as part of acute miliary tuberculosis; or extensively, secondary to tubercle in other parts.

*Cancer* is met with in the form of scirrhus, encephaloid, or colloid, the omentum being a comparatively frequent seat of the last-mentioned variety. Usually the peritoneum is involved secondarily, by extension from one of the abdominal organs, but in rare instances it is affected primarily and solely.

These morbid growths tend to originate *ascites*, or *acute* or *chronic peritonitis*, and it is to these conditions that their local symptoms are

mainly due. Sometimes fluid collects with extreme rapidity in cancer. There will also probably be more or less general symptoms. *Colloid in the omentum* yields the following physical signs:—1. The enlargement of the abdomen may be very great, but is wanting in uniformity; the umbilicus appears stretched, but not everted. 2. Firm irregular masses can generally be felt, and even if fluid is present, fluctuation is very indistinct. 3. Dulness is usually elicited over the front of the abdomen. 4. Change of posture produces no effect, unless there is much fluid present. 5. The aspirateur or exploratory trochar may bring away a slimy gelatinous fluid, and this is occasionally discharged by vomiting, or *per rectum*.

#### IV. ABDOMINAL PERFORATIONS AND RUPTURES.

Apart from the effects of traumatic injury, *perforations* and *ruptures* are liable to take place in connection with the abdominal contents, and in order to avoid repetition, it will be convenient to indicate the chief facts pertaining to this subject in the present chapter, as the peritoneum so commonly suffers in these cases.

**ÆTIOLOGY AND PATHOLOGY.**—The principal structures which are liable to give way, and the pathological conditions which cause these lesions, may be thus summarized:—1. Perforation of the stomach or intestines from within, in connection with ulceration or the resulting cicatrices; gangrene; cancer; the action of corrosive poisons, especially on the stomach; or mechanical irritation and destruction, particularly by foreign bodies introduced from without, but sometimes merely by hardened fæces, worms, or gall-stones. It must be mentioned that extensive *post-mortem* softening and destruction of the coats of the stomach may, under certain circumstances, result from the action of the gastric juice. 2. Rupture of an abscess, hydatid-cyst, or soft cancer in the liver. 3. Perforation of the gall-bladder, either by gall-stones which have caused ulceration; or from cancer. 4. Rupture of the spleen, from extreme enlargement and softening; or abscess. 5. Various ruptures in connection with the uterus and ovaries. 6. Bursting of any accumulation in the pelvis of the kidney; of an abscess or cyst in this organ; or of the bladder from over-distension. 7. Bursting of an abscess unconnected with any organ; or of a soft morbid accumulation in the absorbent glands. 8. Rupture of an aneurism. 9. Perforation of a hollow viscus from without, owing to the destruction of its coats by some solid tumour. 10. Bursting of a peritoneal accumulation. 11. Very rarely perforation of the diaphragm, with escape of some fluid collection from the chest into the abdominal cavity.

These lesions usually occur without any immediate *exciting cause*, but certain of them may be brought on by some mechanical disturbance, such as vomiting, coughing, or laughing; straining at stool; or, in the case of ulceration of the alimentary canal, by indulging in excess of, or in irritating articles of food, or in such articles as cause flatulent distension.

**ANATOMICAL CHARACTERS.**—The perforation or rupture may take place into different parts, and the pathological consequences will vary accordingly. 1. Most frequently the opening communicates with the peritoneum, into which foreign matters are poured more or less freely, exciting *perforative peritonitis*, severe and rapid in proportion to the



quantity and irritant nature of the materials thus introduced into the sac. 2. Sometimes the opening takes place into the subperitoneal cellular tissue, local inflammation, ending in the formation of abscesses, being set up in this structure. 3. Not uncommonly one hollow organ forms an adhesion with another, and when perforation occurs, a communication is established between the two viscera; or it may unite with a solid organ, and when perforation is completed, this organ may make up for the deficiency, and thus prevent serious symptoms. 4. Union may be set up with the abdominal walls, so that ultimately the opening is formed on the external surface.

**SYMPTOMS.**—From the facts just stated it will be evident that the symptoms indicating perforation must differ considerably, and there may be none at all, or death may take place almost instantaneously, as from rupture of an aneurism. As a rule there have been previous signs of some morbid condition in connection with which the lesion occurs. Presuming the perforation to be sudden and of any extent, and that the communication takes place into the peritoneal cavity, this event is usually indicated by a sudden intense pain at the seat of rupture, often of a burning character, which spreads rapidly over the abdomen, being sometimes attended with a feeling as if something were pouring out; while at the same time there are the ordinary signs of more or less collapse or shock, and death may rapidly ensue from this cause, or from hæmorrhage. Should the patient rally, peritonitis will be speedily set up, the peculiar features of which are that the local symptoms precede any rigors; that the pain starts as a rule from a certain spot; that the course of the disease is usually very rapid; and that the termination is almost always fatal. If the perforation takes place into the cellular tissue, there will be signs of local inflammation, followed by abscess; with general pyrexia. The attacks of sudden pain and collapse may be repeated, this probably indicating extension of the perforation, or the formation of fresh communications.

**DIAGNOSIS.**—If any morbid condition is known to exist which might lead to abdominal perforation, the sudden occurrence of the local and general symptoms indicated above would justify the diagnosis of this untoward event, and the subsequent course would probably soon clear up any doubt. Should there have been no previous evidence or knowledge of such a condition, however, there is often much obscurity, but perforation must always be borne in mind when urgent abdominal symptoms set in, accompanied with signs of collapse or shock.

**PROGNOSIS.**—This is always exceedingly grave, but the termination is not invariably fatal. Much will depend upon the condition of the patient; the cause of the perforation; the structure into which it takes place; its extent; and other circumstances.

**TREATMENT.**—In any case of abdominal perforation the patient must be kept *absolutely at rest*, and this applies still more emphatically to the organ which is the seat of the lesion. In the case of the stomach or bowels, there should be complete abstinence from food by the mouth, and only small nutrient enemata administered, or nutrient suppositories might be used. Opium is the great remedy for the purpose of counteracting shock, relieving pain, and checking peristaltic action. It should be given in full doses, at short intervals. Collapse must also be treated by free administration of *stimulants*, which, if the alimentary canal is affected, must be given by enemata; application of heat to the extremities; and the use of sinapisms. Hot fomentations may be ap-

plied over the abdomen. Should peritonitis or other form of inflammation be set up, appropriate treatment must be adopted. After perforation of the stomach and intestines it is extremely important to avoid giving anything by the mouth for some time; and to refrain from any attempt to act upon the bowels by means of aperients.

#### V. ASCITES.—DROPSY OF THE PERITONEUM.

**ÆTIOLOGY.**—Ascites is merely a localized dropsy of the peritoneum, and the chief causes from which it may result are :—1. Pressure upon the branches of the portal vein within the liver, especially from cirrhosis and other forms of chronic contraction of the liver; or infiltrated cancer. 2. Pressure upon the portal trunk in the fissure outside the liver. It is from this cause that ascites is most frequently associated with many diseases of the liver, such as cancer, albuminoid disease, hydatids, or abscess; either projections from the liver pressing on the vein, or the glands in the fissure being simultaneously affected. Inflammatory thickening from peri-hepatitis; any tumour in the vicinity; or an aneurism may also cause pressure on the portal trunk. 3. Internal obstruction of the portal vein by a thrombus. 4. Pressure upon the inferior vena cava, after it receives the hepatic trunk. 5. Cardiac or pulmonary diseases obstructing the venous circulation, these in time originating organic changes in the liver. 6. Renal disease. 7. Chronic peritonitis; or morbid deposits in the peritoneum, the latter being supposed to act by inducing active congestion, but probably chiefly originating dropsy by causing pressure upon the small vessels, or even upon veins of some size. 8. Exposure to cold; suppression of discharges or of chronic skin diseases; and other causes which may lead to internal active congestion. The reality of this class of causes is questionable, but cases of ascites have been attributed to them.

**ANATOMICAL CHARACTERS.**—The quantity of dropsical fluid which may collect in the peritoneum varies extremely, but it not unfrequently amounts to several gallons. It distends and macerates the tissues in proportion to its amount. In characters it is usually in the main watery in consistence; clear and transparent; colourless or faintly yellow; alkaline in reaction, or very rarely neutral or acid. It may be yellow, turbid, dirty-looking, stained with bile or blood, gelatinous, or mixed with soft fibrinous masses. The composition of ascitic fluid is far from being uniform, but generally it contains much albumen; occasionally it yields fibrin, urea, or cholesterin. Ascites tends to displace or compress the abdominal structures, as well as those within the chest. The heart may be considerably raised.

**SYMPTOMS.**—The only symptoms directly due to ascites are those dependent upon the mechanical effects of the fluid. There is more or less discomfort and sense of fulness, in proportion to its quantity; or aching may be felt in the loins. Digestive disturbances are common, flatulence and constipation being often prominent symptoms, and sometimes vomiting takes place. Owing to the interference with the diaphragm, dyspnoea is likely to be complained of, and may be urgent, being much increased often by flatulence, and by the recumbent posture. The heart's action may also be disturbed, as evidenced by palpitation, irregularity, or sometimes a tendency to syncope. Anasarca of the legs is liable to follow ascites, resulting from pressure exerted by the fluid

on the *inferior vena cava*, which also leads to enlargement of the veins of the abdominal wall. When ascites is caused by pressure on the *vena cava*, of course anasarca of the legs is observed simultaneously with, or even before the peritoneal dropsy. Albuminuria may be induced by pressure on the renal veins, the urine being also concentrated and deficient in quantity. The skin is often dry and harsh.

**PHYSICAL SIGNS.**—These require careful consideration, and in the majority of cases they are sufficiently characteristic, but necessarily depend upon the quantity of fluid present. 1. The skin usually appears stretched to a variable degree, smooth, and shining, feeling thin; the superficial veins are often enlarged; and the umbilicus is stretched, everted or pouched out, and becomes finally obliterated. 2. The abdomen is more or less enlarged, in some cases enormously; quite symmetrical; and of a rounded form, though it tends to bulge in the flanks or in the hypogastric and iliac regions, according to the position of the patient. The greatest circumference is about the level of the umbilicus, which is the highest point of the abdomen; the thorax appears small and depressed, and its lower margin may be everted, or the ensiform cartilage is sometimes bent sharply up. Usually a history can be obtained that the enlargement commenced below; and that it increased steadily, though slowly in most cases. 3. Abdominal respiratory movements are frequently either deficient or absent; and breathing is generally hurried and shallow. 4. The surface of the abdomen feels quite regular and uniform; and fluctuation is generally readily elicited from side to side, or in other directions. 5. Dulness is observed first towards the lumbar regions, if the patient lies in the supine position; then in the lower part of the abdomen; and it extends by degrees towards the front and upwards, until finally it may be observed all over the abdomen. The umbilical region retains the tympanitic sound longest, and it is often excessive for a time in this part. When the patient sits up, the prominence in front between the recti becomes tympanitic. 6. Auscultation affords negative signs. 7. Change of posture gives important signs, namely, the fluid can occasionally be seen moving as the position is altered; the form of the abdomen is modified, bulging being observed in the most dependent part; while the seat of dulness and fluctuation is changed. 8. Examination *per rectum* reveals the sensation of the resistance of fluid. 9. Examination *per vaginam* indicates that the vagina is short, and the uterus pushed down or flexed; occasionally a pouch projects through the vulva. 10. Any fluid which is removed by tapping usually consists of mere serum, containing generally a considerable amount of albumin. 11. The heart may be displaced upwards and to the left, occasionally a basic murmur being thus originated.

**DIAGNOSIS.**—There are two chief points to be attended to in the diagnosis of ascites, namely:—1. To determine whether fluid is present; and to distinguish enlargement due to this cause from that dependent upon other morbid conditions. 2. To make out the pathological cause of the dropsy. The chief *general abdominal enlargements* which may simulate ascites are those associated with great obesity, with much fat in the omentum; a flabby relaxed state of the abdominal walls, with flatulence; considerable subcutaneous oedema; peritonitis, especially chronic; infiltration of colloid cancer in the omentum; a greatly dilated stomach; an ovarian tumour; distension of the uterus with fluid, or a pregnant uterus; an extremely distended bladder; a large hydatid tumour in



connection with the liver or any other structure; an enormous cyst in the kidney; and a phantom tumour.

It is by *physical examination* that ascites is mainly distinguished from the conditions just enumerated, but it is important to observe that its ordinary signs may be modified or obscured by the co-existence of certain of these conditions; by the association of the dropsy with a tumour, morbid deposit, or enlarged organ; by the fluid being either very small in quantity, or on the other hand extremely abundant; by the mesentery being so short as not to allow the intestines to come forward; or by the existence of adhesions limiting the fluid. When ascites is associated with any solid enlargement, the latter may frequently be recognized by making sudden firm pressure with the fingers, by which the fluid is pushed aside and the firm mass reached; or in doubtful cases the fluid can be removed, and satisfactory examination then carried out.

Important aid in the diagnosis may also be derived from:—1. A careful *general history* of the case; and the conditions of the patient with respect to *age* and *general appearance*. 2. The *history of the enlargement*, as to whether it has been more or less acute or chronic in its progress, and whether it has fluctuated or steadily progressed; as well as its seat of origin, and directions of increase. 3. The accompanying *symptoms*; and the condition of the *main organs*, which should all be thoroughly examined. 4. The *results of treatment*, not forgetting the use of the aspirateur or trochar; of the catheter; and of means for clearing out the alimentary canal.

The characters of most of the enlargements mentioned above are described in other parts of this work, to which descriptions reference must be made for individual diagnosis. It is necessary, however, to point out specially the characters distinguishing *cystic tumour of the ovary* from *ascites*. 1. *Physical signs of ovarian tumour*. (i.) The umbilicus is often thinned and flattened out, but not everted or pouched out. (ii.) The enlargement is not so globular in shape; projects anteriorly; does not bulge in dependent parts; and is frequently not quite symmetrical, this being accurately determined by semicircular measurements, or by comparing the distance from the umbilicus to the anterior superior iliac spine on each side. The greatest circumference is said to be about an inch below the umbilicus, in the recumbent posture; and the measurement from the ensiform cartilage to the umbilicus is generally shortened. (iii.) As a rule fluctuation is indistinct; the enlargement feels more or less firm and resistant, or even nodulated; while the sensations are not uniform over the entire surface. Frequently on deep pressure greater resistance or tension is felt on one side than the other. (iv.) Percussion reveals dulness, chiefly in front of the abdomen, even in the umbilical region, while the flanks are tympanitic, and the dulness often extends more towards one side than the other. The prominence between the recti in the sitting posture is dull. There is usually a sense of considerable resistance on percussion. (v.) Auscultation may detect a pressure-murmur over one iliac artery. (vi.) Change of posture does not produce the alterations observed in ascites. (vii.) Examination *per rectum* detects a firm resistance. (viii.) The vagina is long and narrow above, the uterus being raised. (ix.) An exploratory trochar may bring away a thick, glutinous, or coloured fluid, which sometimes contains cholesterin; and after this has been removed solid portions of the tumour may be felt more readily.

2. There is no history of any cause, or evidence of any organic disease likely to originate ascites. 3. Frequently the patient has observed that the enlargement commenced below, and from one side. 4. Symptoms which often accompany ascites are absent; while anasarca of the legs is commonly an early symptom of ovarian tumour, owing to pressure on the veins, which may be entirely or chiefly confined to one side.

With regard to the diagnosis of the *cause* of ascites, this can generally be made out by a satisfactory investigation as regards the history, symptoms, and physical signs, directed to the liver, heart, and kidneys. The distinctive characters referable to the ascites itself have already been pointed out in the chapter on DROPSY. Obscure causes can only be determined by exclusion; and by a thorough consideration of all the circumstances bearing upon the individual case.

TREATMENT.—The measures adopted in the treatment of dropsy generally are applicable for cases of ascites, but the medicinal remedies which affect it most powerfully are *purgatives*, though these often fail, and Dr. Leech is satisfied from his experience that very active purgation is rarely beneficial in hepatic dropsy. The balsam and resin of copaiba have been found decidedly efficacious in some instances, acting as *diuretics*. There are two measures, however, which demand special notice in relation to ascites, namely, *paracentesis abdominis*, and the employment of *pressure*. It has been the custom to look upon paracentesis as an operation which should only be performed as a last resource, when the fluid has collected to such a degree as to cause urgent symptoms. When the ascites is a part of general dropsy from cardiac or renal disease the amount of fluid is not often so great as to need its removal by operation, nor could this really serve any beneficial purpose, except in affording temporary relief, although occasionally ascites from cardiac disease has been thus cured. The last remark applies also to many cases in which it is merely a local dropsy, as when ascites is associated with cancer of the liver; but there is one class of cases in which paracentesis may not uncommonly be performed as a curative measure, so far as the ascites is concerned, namely, when it is dependent upon *cirrhosis of the liver*. In such cases I have for some years had recourse to *paracentesis* as a systematic method of treatment. In some cases one such operation has sufficed for a cure; usually it has to be repeated, and I maintain that the fluid may be taken away again and again should it re-accumulate, due care being of course exercised in the performance of the operation, and in the subsequent management of the case. My own results have been highly satisfactory, and other observers have also recorded favourable results from this operation; therefore it appears to me justifiable to insist upon the employment of *paracentesis abdominis as a means of cure*, in connection with ascites from uncomplicated cirrhosis of the liver, should the fluid be at all abundant, and show no signs of being removed by other methods of treatment. I am aware that cases have been recorded in which recovery has followed merely general tonic and other modes of treatment, but this is such a rare event that sole reliance cannot be placed on these remedies, though they may aid materially the treatment by operation, and some of them may be adopted as adjuncts after paracentesis. *Pressure* may also be of service, the abdomen being tightly bound by a broad roller, as soon as all danger of undue irritation has ceased, from which much benefit often results. I may state that seldom has any injurious consequence followed the operation within my experience; and in some almost hopeless cases

permanent recovery has been brought about. The employment of poultices of digitalis leaves, along with pressure, has appeared to me to do good in some instances.

---

## CHAPTER XXXII.

## DISEASES OF THE STOMACH AND INTESTINES.

## CLINICAL CHARACTERS.

SYMPTOMS referable to the alimentary canal are of such common occurrence, that it has properly become a matter of routine in the examination of a patient to make inquiry concerning them. The succeeding general sketch will indicate the clinical phenomena which may be met with, and the course to be pursued in their investigation.

1. **Morbid sensations** are very commonly experienced over some part of the abdomen, the principal being pain or tenderness; heat or burning in the epigastrium: a sense of sinking, dragging, or tightness; discomfort, weight and fulness after food, or, on the other hand, a feeling of emptiness even after a full meal, with constant craving for food; and abnormal movements within the abdomen. *Cardialgia* or *heartburn* are terms applied to a peculiar sensation of heat or burning in the epigastrium, which extends upwards, as if along the œsophagus, to the throat; or which in some cases spreads more or less over the chest. With regard to *pain*, it is very important not only to investigate it carefully in all the usual particulars, but also in many cases to ascertain whether and in what way it is influenced by food or drink in general, or by special articles of diet; by vomiting or eructation; defæcation or the passage of flatus; posture or movement; coughing or deep inspiration; mental disturbance; or, in certain instances, by the periods of menstruation. In determining whether there is tenderness, it is well to take off the patient's attention, and its site and extent, degree, and apparent depth must be made out as accurately as possible; while it must be noted whether it appears to be connected with any evident morbid condition, such as a tumour. These remarks apply to all kinds of abdominal pain or tenderness. When the stomach is affected, uncomfortable or painful sensations are often referred to the back, between the shoulders; to the front of the chest; or round the sides, especially the left.

2. The sensations as regards inclination for **food and drink** are often altered. *Appetite* may be deficient or lost—*anorexia*—in some cases the feeling amounting to a complete disgust for food; excessive, both as to quantity and frequency—*bulimia*; attended with a desire or dislike for special articles; or altogether depraved—*pica*. *Thirst* is a frequent symptom, and there may be a particular inclination for certain drinks; on the other hand, an antipathy to fluids is sometimes observed.

3. The process of **digestion** is frequently interfered with. Hence decomposition or fermentation is set up in the contents of the alimentary canal, leading to the production of gases; occasionally of alcohol; of



various acids (lactic, butyric, acetic, &c.); or of vegetable growths (*sarcinæ ventriculi* and *torulæ*). Great discomfort may thus arise from flatulent distension; abdominal gurgling or rumbling—*borborygmi*; gaseous eructations; or acidity.

4. **Expulsive acts** are excited in connection with the stomach, with the view of getting rid of offending materials, namely, *vomiting* and *retching*, which may or may not be attended with a feeling of nausea; *regurgitation* of food; or *eructation* of gases, liquids, and other substances. With regard to the mechanism of these acts, *vomiting* is not only attended with contraction of the muscular coat of the stomach, but also of the abdominal and thoracic muscles, while the cardiac end of the œsophagus is relaxed. *Retching* is the same act, but ineffectual, merely air being expelled, either because the stomach is empty, or because the lower portion of the œsophagus is spasmodically closed. *Regurgitation* and *eructation* are simply due to contraction of the stomach, and some individuals can regurgitate their food at will. In infants the act of vomiting appears to be much of this character. A special form of eructation or regurgitation has been named *pyrosis* or *water-brash*, in which, often after painful sensations in the epigastrium, especially a sense of burning, a quantity of clear watery fluid rises into the mouth, generally tasteless and neutral, but in some cases sour or acrid and acid in reaction. This fluid has been supposed by some to be mainly saliva; others have considered it to be pancreatic juice; but probably most of it comes from the stomach. Dr. Clifford Allbutt regards pyrosis as being for the most part a pure neurosis, probably vaso-motor.

5. Blood may be poured out into the alimentary canal, and either rejected from the stomach—*hæmatemesis*; or passed by the bowels—*melæna*.

6. The bowels are very commonly irregular in their action, either in the direction of **constipation** or **diarrhœa**. It is frequently desirable to make particular inquiry into this matter, as patients offer general statements which may easily mislead. The chief points to be ascertained are the frequency of the act of defæcation; whether attended by any straining; whether any unusual sensations precede, accompany, or follow it; and the quantity and characters of the materials discharged. In many cases it is imperative to make a personal examination of the stools, noticing their amount; colour; general appearance; consistence; the form and size of any solid fæces; odour; if there are any signs of fermentation or aëration; general composition, the materials to be specially looked for, in addition to ordinary fæces, being various articles of food, either unaltered or more or less digested; foreign bodies introduced from without; calculi, especially hepatic; intestinal worms or hydatids; blood or altered blood; mucus or pus; fatty matter; fibrinous flocculi or casts; epithelial shreds; vegetable, animal, or mineral poisons; or, rarely, sloughs or portions of the intestines. Occasionally a chemical and microscopic examination of the fæces is necessary, especially for the detection of poisons and parasites, or even merely to determine their composition.

7. The **tongue** gives important information as to the state of the digestive organs, the particulars to be noted being:—*a.* Its size and shape, and whether it is marked by the teeth. *b.* The colour of its mucous covering, especially at the tip and edges. *c.* Its condition as to dryness or moistness. *d.* The state of the surface, whether smooth, glazed, fissured, furrowed, &c. *e.* The size, shape, and colour of the

various papillæ. *f.* The presence, extent, and characters of any fur over the dorsum. It may also be mentioned here that the mouth and throat are frequently affected when the stomach is out of order; while a slimy, bitter, or otherwise disagreeable taste is often experienced; and the breath has an unpleasant odour.

8. In some cases **abnormal sensations** are experienced about the lower part of the **rectum** and **anus**, such as pain, either constant or only felt before, during, or after the act of defæcation; fulness, weight, heat or burning, itching, constriction, dragging, or frequent inclination to go to stool, with straining. Certain of these sensations are included under the term *tenesmus*. Hæmorrhoids are also of frequent occurrence.

9. The methods of **physical examination** applicable to the alimentary canal have been previously indicated, and these are particularly useful in making out flatulent distension; a tumour or growth in connection with the stomach or intestines; accumulations in their interior; permanent dilatation of the stomach; and displacement, spasmodic contraction, or obstruction in the course of any part of the alimentary canal.

10. It will readily be understood that any derangement of the digestive organs is very likely to affect the **general system**. Hence numerous symptoms arise, varying much in their exact nature in different cases, the most important being wasting, often accompanied with a sallow or anæmic aspect; a sense of debility, general discomfort, languor, malaise, and fatigue, with incapacity for effort, especially in the mornings and after meals; more or less pyrexia, with a dry and harsh skin, or on the other hand a depression of temperature, with cold extremities and sweats; nervous symptoms, namely, congestive or neuralgic headache, or a feeling of weight and oppression in the head, giddiness, irritability and petulance, depression of spirits and apathy, inaptitude for any mental effort, confusion of ideas and failure in intellectual vigour, hypochondriasis, wakefulness, or drowsiness, with restless and unrefreshing sleep attended with disagreeable dreams, timidity and nervousness, pains in the limbs and back, chilliness or even rigors, especially in the evenings, creeping sensations over the body, or convulsions in children; disturbance of the heart's action, in the way of palpitation or irregularity, feebleness, sometimes accompanied with faintness or actual syncope, as well as with uncomfortable sensations in the cardiac region, the pulse being weak; dyspnœa, hiccup, or asthmatic attacks; oppression across the chest, and cough; changes in the urine, especially indicated by excess of lithates or sometimes of phosphates or oxalates, excessive or deficient acidity, and deficiency of chlorides; menstrual derangements; and skin-eruptions, such as urticaria, herpes, or psoriasis.

11. A tumour or solid accumulation in connection with the stomach or intestines may press on **neighbouring structures**, and may thus originate different symptoms.

## CHAPTER XXXIII.

## ON CERTAIN GASTRIC SYMPTOMS AND FUNCTIONAL DISORDERS.

IN this chapter the chief symptoms and functional affections connected with the stomach will be considered; the diagnosis, prognosis, and treatment of the more chronic complaints will, however, be referred to in a subsequent general chapter on the subject.

## I. GASTRODYNIA—GASTRALGIA.

**ÆTIOLOGY.**—Dr. Clifford Allbutt, in his admirable lectures on Visceral Neuroses (*Gulstonian Lectures*, 1884), discusses gastralgia very fully, and to these lectures the reader is referred for complete information on the subject. The term implies a painful neuralgic affection of the stomach, and Dr. Allbutt states that this organ is “by very far the commonest seat of abdominal neuralgia.” Gastralgia is chiefly met with among females, but also occurs in males. Allbutt gives the proportion as two to one. He states that in men and women gastralgia is by no means confined to middle life, but is found at all ages, between 14 and 60, being most common between 20 and 45. It comes on earlier in women than men, apparently by some ten years. “A gastralgic man is rare before the twenties; girls often begin in their teens.” In females it seems to occur especially about the time of puberty, or when the menstrual functions are declining. It tends to die out in middle life. The conditions with which the complaint is mainly associated are physical exhaustion and debility; anæmia; hysteria; hypochondriasis; nervous exhaustion from depressing emotions, anxiety, or excessive or prolonged mental effort; gout or rheumatism; and uterine or ovarian derangements, including pregnancy. Sedentary habits, with habitual constipation; and excessive use of hot tea have appeared to me to have had considerable influence in originating this affection in some cases. Occasionally it results from the action of malaria; and in rare instances depends on central nervous disease. The “gastric crises” in locomotor ataxy are of a gastralgic nature. Allbutt traces an affinity of gastralgia with the “dartrous” diathesis, and phthisis, as well as with gout and rheumatism.

**SYMPTOMS.**—The prominent symptom of gastralgia is epigastric pain, varying much in its severity and characters, usually paroxysmal, and coming on either at regular or irregular intervals, though in many cases there is never complete relief. The attacks are not unfrequently nocturnal. During the paroxysms the suffering may be extreme, especially in cases of hysteria or gout. Food frequently gives decided relief, the pain returning as the stomach becomes empty. Sometimes indigestible substances afford more ease than those which are digestible and soothing. Some patients, however, suffer intensely when they take anything, or after particular articles, such as hot tea, and the suffering after food may be so great as to lead to abstinence, and then to distaste



and aversion to food—*anorexia nervosa*. Pressure generally relieves, especially when made firmly and continuously, but there may be much superficial tenderness or hyperæsthesia. The pain often radiates along the spinal nerves, and these may even be chiefly affected, indicated by cervico-brachial and intercostal neuralgia. Various curious sensations are often complained of in the epigastrium, such as sinking and the like. During the severe attacks of pain spasmodic movements of the stomach and bowels may be observed, with cramps of the abdominal muscles. Symptoms usually regarded as dyspeptic are habitually present in most cases, such as acid and gaseous eructations, voluminous flatulency, borborygmi, heartburn, or pyrosis; Allbutt, however, looks upon these phenomena as frequently of a neurotic character. The tongue may be fairly natural. In certain cases chronic vomiting is sometimes a very distressing symptom; and not uncommonly a morbid craving exists for improper and indigestible articles of food. Vomiting is sometimes a climax-symptom in gastralgia, and may remain after this complaint has passed away. The bowels are generally very constipated. Frequently other neurotic disturbances are observed, such as migraine, asthma, cardiac disorder or angina pectoris. In some instances there is considerable emaciation, especially if food is not taken; but it is remarkable what a slight degree of wasting may attend the chronic vomiting of hysterical cases. Aortic pulsation is often present.

## II. SPASM OR CRAMP OF THE STOMACH.

**ÆTIOLOGY.**—This complaint differs from gastralgia in being an acute affection, attended with spasmodic contraction of the walls of the stomach, which may be excited by indigestible or irritating food or drink, or in some individuals by special articles of diet, ordinarily quite harmless; drinking excess of cold water, or indulging too freely in ices, especially when the stomach is empty; acrid secretions in the stomach; flatulent distension; mental emotion; and gout.

**SYMPTOMS.**—There is intense pain, which comes on suddenly in a series of paroxysms with remissions, being of a griping, constrictive, or twisting character. It is most marked near the pylorus, but may be felt running across the epigastrium, or even up along the œsophagus. Pressure gives marked relief, the patient either sitting up and making firm pressure over the stomach, lying upon the abdomen, or tossing and rolling about. Often a feeling of sickness is experienced, and the pain may be eased by vomiting. More or less prostration is frequently observed, and occasionally even severe collapse, with cold and clammy sweats, a very feeble and slow pulse, and fluttering of the heart, which condition may actually terminate in death. Sometimes the spasmodic movements of the stomach can be felt externally. If they continue for some time a little soreness and tenderness remain, but these sensations soon pass off.

**TREATMENT.**—Should there be any irritating materials in the stomach, an *emetic* of sulphate of zinc or mustard, with plenty of lukewarm water, should be given immediately. A combination of spirits of ammonia, spirits of chloroform, and tincture of opium, with some carminative, will generally relieve the pain. If there is acidity, carbonate of soda or magnesia may also be administered. A little brandy or gin with hot water is often very beneficial. The continuous external

application of dry heat over the abdomen, by means of hot plates, an india-rubber hot-water bottle, or a bag containing hot bran or salt, is most soothing. After the attack it may be well to clear out the alimentary canal by means of a brisk *purgative*.

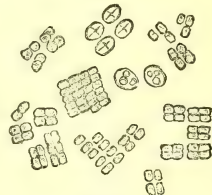
### III. VOMITING--EMESIS.

**ÆTIOLOGY.**—The act of vomiting is excited either through some reflex irritation; or by a direct disturbance of the brain, affecting the stomach through the vagus nerves. Its numerous causes may be classed thus:—1. *Those immediately acting upon the stomach*, namely, gastralgic attacks; irritating materials in its interior, whether introduced from without or formed there; organic diseases of its coats; obstruction at the pyloric orifice; external pressure upon the organ; or its displacement, for example, hernia of the stomach through the diaphragm. 2. *Reflex irritation from other sources*, particularly the throat; intestines (hernia, worms); peritoneum; female genital organs (especially in connection with pregnancy; and testicles. Reflex vomiting also accompanies the passage of a gall-stone or renal calculus, as well as other complaints attended with severe pain. It may arise in susceptible persons from any unpleasant smell, taste, or sight; or even from a sudden light. The vomiting which follows severe fits of coughing, especially in phthisis, comes mainly under this head, but is partly mechanical. 3. *Centric or cerebral vomiting*. The chief causes coming within this group are injury to, or disease of the brain or its membranes, especially meningitis; cerebral anæmia or congestion; a poisoned state of the blood, the poison being either introduced from without (for example, alcohol, and its products, tobacco, tartar-emetic, chloroform, opium and its constituents, lobelia), or being generated within the body, as in various febrile disorders, especially at the outset, uræmia, or from the inhalation of a hot and tainted atmosphere; mere nervous shock or fright; hysteria and other functional nervous derangements, the vomiting being then probably the result of disordered circulation; and the thought of unpleasant things. With regard to *sea-sickness* and other allied forms of vomiting, such as that brought on by swinging, these certainly come mainly within the *cerebral* class, but several theories have been propounded to explain the occurrence of this symptom under these circumstances. The peculiar movements, the appearance of objects in motion, and the unpleasant odours and sights usually present, probably all aid in inducing sea-sickness, though some authorities regard it as entirely due to a peculiar disturbance of the cerebral circulation. Vomiting is a prominent symptom in migraine or sick-headache. Morning sickness is often associated with chronic alcoholism, being partly the result of the presence of deleterious materials in the blood; partly of catarrh of the throat and stomach, the former giving rise to fits of cough. It must not be forgotten that malingerers can sometimes excite vomiting at will.

**CLINICAL CHARACTERS.**—It is frequently requisite to make a thorough investigation with regard to vomiting, in order to arrive at a correct diagnosis as to its cause, the following particulars being noted:—1. The *times* and *frequency* of its occurrence. 2. The *circumstances* under which it takes place, whether spontaneously; only when the stomach is empty; after any food or drink, or only after certain articles or meals, it being

important also to ascertain the quantity necessary to induce vomiting, and how soon it follows the introduction of the exciting materials; in connection with some obvious reflex or centric cause, such as cough, irritation in the throat, severe pain, a bad smell or taste, smoking, drinking, or mental disturbance; in certain positions, or on change of posture. It must not be forgotten that many poisons excite vomiting, and suspicious cases might come under observation needing complete and cautious investigation as to substances which had been taken into the stomach, or some of these might even be required for chemical examination. 3. The *sensations preceding and accompanying the act*, especially noting if there is any feeling of nausea, as well as its degree; giddiness; prostration; or pain. 4. The *manner in which the act is performed*, this being determined by personal inspection, if possible, especially remarking if it appears to be originated voluntarily; and whether it is performed easily, or with more or less straining and retching. 5. The *after-effects*, particularly as regards the relief of gastric pain or its intensification; and the influence upon cerebral symptoms. It may be mentioned here that the mere violence of vomiting may occasion serious lesions, such as rupture of the stomach or of a vessel, apoplexy, or hernia, and it often leaves a sense of soreness over the abdomen. 6. *Examination of the vomited matters*. This is of the utmost importance, and ought never to be neglected, and the same remark applies to materials discharged by regurgitation or eructation, or brought up by the stomach-pump. The chief points to be noticed are:—*a. The quantity rejected.* *b. The taste*, as perceived by the patient. *c. Odour.* *d. General physical characters*, as to colour, and as to the materials of which the vomited matters consist, whether of different kinds of food, unaltered or in various stages of digestion, decomposition, or fermentation; unusual substances introduced from without; blood or altered blood; gastric juice; watery fluid; mucus; biliary matters; fæces; morbid products, such as calculi, worms, hydatids, portions of growths, or pus. It is also desirable to observe whether the vomit is frothy or yeasty-looking. *e. Chemical characters.* The reaction should always be taken; and in certain cases it may be desirable to make a chemical analysis, in order to determine the presence of products of fermentation, gases, bile, sugar, urinary compounds, or inorganic or organic poisons. Of course in any case of suspected poisoning a complete analysis must be performed. *f. Microscopic characters.* The chief microscopic elements to be looked for in vomited matters are blood-corpuscles, pus-cells, cancer-cells, echinococci, and sarcinæ or torulæ. The microscope is also of use in detecting certain poisonous substances. *Sarcinæ* (Fig 23) are vegetable growths, and appear as little oblong rectangular bodies, in shape resembling minute wool-packs, being divided into four equal parts by cross lines which correspond to dissepiments, these being again subdivided by fainter lines, so that in all they make up 64 divisions, each ultimate particle consisting of an elementary square cell. Sarcinæ are only found in acid vomit, which usually presents well-marked signs of having undergone fermentation, and they are most frequently observed in connection with pyloric obstruction.

FIG. 23.



Sarcinæ.

**DIAGNOSIS.**—By attention to the particulars just considered, aided by the history of the case, and the other symptoms present, the cause of



vomiting can generally be satisfactorily made out. It is requisite, however, to point out the chief distinctions between *cerebral* and *gastric* vomiting. 1. Nausea usually precedes and attends the latter; but is often absent in the case of the former. 2. The accompanying symptoms in the one case point chiefly to the alimentary canal, and especially to the stomach; in the other to the brain, head-symptoms being prominent. 3. The act of vomiting generally relieves any nausea, giddiness, or headache which may precede it, when it is gastric in origin; such is not the case with cerebral vomiting.

TREATMENT.—Vomiting occurs under such a variety of circumstances that little more can be done here than to indicate the general principles upon which its treatment should be conducted. 1. The *cause* must be sought out, and removed if possible. Thus an *emetic* is not uncommonly one of the best remedies, in order to clear out the stomach of irritant matters. Any reflex excitement must also be subdued. Patients should be told to aid voluntarily in suppressing vomiting as much as they can; being also warned against bringing it on by coughing or any such act. 2. Attention to *diet* is all important. By withdrawing food altogether, or only giving very small quantities of cool or iced liquids, especially milk with lime-water or soda-water, or brandy with weak beef-tea or beef-juice, sickness may often be effectually stopped. It is particularly necessary to inquire into the feeding of children, as vomiting in these subjects is so commonly due merely to errors in this respect. Koumiss is of unquestionable service in some cases of vomiting. The administration of artificially digested food proves of great benefit in some cases. 3. It may be useful in treating vomiting to attend to certain *general matters*, such as position, rest, and free ventilation. Especially is this the case with regard to cerebral vomiting and sea-sickness, against which absolute rest in the horizontal posture, with a free supply of fresh air, may afford some protection. Pressure by means of a girdle across the abdomen has been recommended to prevent sea-sickness. 4. The chief *direct remedies* for the relief of vomiting are the sucking of small lumps of ice; effervescent draughts with hydrocyanic acid, or the latter with mucilage; iced champagne or brandy with soda-water; opium, either in the form of pill, as the tincture or liquor opii sedativus along with other remedies, or in an enema with starch; morphia in pill, by hypodermic injection, or sprinkled on a blistered surface over the epigastrium; chloroform; creosote in drop doses in the form of pill; sulphurous acid, sulphite of soda, or hyposulphites, should the vomiting depend on vegetable growths, or carbolic acid or sulpho-carbolates under the same circumstances; nux vomica or minute doses of strychnia, the last-mentioned proving wonderfully efficacious in some instances, after all other remedies have failed. Salts of bismuth, magnesia, liquor potassæ, carbonate of soda, or oxalate of cerium, are also valuable under certain conditions. Dr. Ringer recommends in many forms of vomiting drop doses of ipecacuanha wine, every hour, or three times a day, according to circumstances; in others he finds arsenic useful. In some forms of chronic vomiting, oxide or nitrate of silver in minute doses may prove beneficial. It is desirable to make all draughts as small and as agreeable to the taste as possible. 5. *External applications* over the epigastrium are sometimes beneficial in treating sickness, especially sinapisms, a small blister, cold by means of the ice-bag, and friction with chloroform or belladonna liniment.

## IV. HÆMATEMESIS.

**ÆTIOLOGY.**—Blood may find its way into the stomach under a variety of circumstances. As a rule it comes from the vessels of this organ, being usually capillary in its origin, but sometimes due to the erosion or rupture of a large vessel; the blood may, however, be derived from other sources. The causes of hæmatemesis may be thus classified:—

1. *Traumatic*, from external violence over the epigastrium.
2. *Diseased conditions of the blood*, especially in yellow fever.
3. *Vicarious*, particularly in connection with deficient menstruation.
4. *Injury* by foreign bodies or destructive chemical agents which have gained access into the stomach.
5. *Abnormal conditions affecting the stomach itself*. Thus hæmorrhage may be the result of violent vomiting and retching; congestion from any cause; inflammation; ulceration; cancer; or rarely atheroma of the vessels, embolism or thrombosis, or varicose veins in the stomach.
6. *Diseases of other organs and structures*, especially those in the vicinity of the stomach. These chiefly act by inducing extreme mechanical congestion, which may follow any great obstruction of the portal circulation, but especially that due to cirrhosis of the liver, thrombosis of the portal vein or its branches, pressure upon the portal trunk or vena cava inferior, and long-continued cardiac or pulmonary affections. Acute atrophy of the liver is often attended with hæmatemesis, which is then partly due to the state of the blood. Splenic disease may originate this symptom in both ways. Sometimes a neighbouring disease, such as cancer of the pancreas, destroys the coats of the stomach, and thus opens its vessels. Occasionally an abdominal or thoracic aneurism bursts into this organ, or more commonly a thoracic aneurism opens into the œsophagus. It is stated that an omental hernia may drag the stomach downwards, and thus lacerate the mucous membrane.
7. It must not be forgotten that blood may be *swallowed*, coming either from the œsophagus, mouth, throat, nose, or respiratory organs. The blood of animals also is purposely swallowed sometimes, either by hysterical girls or by malingerers, being afterwards rejected.

**SYMPTOMS.**—Hæmorrhage into the stomach may not be attended with any external indications, either because the blood is poured out so abundantly as to kill instantly; or, on the other hand, because it is in very small quantity. In the majority of cases, but not always, there is either some obvious cause of the hæmorrhage; or it is preceded by symptoms referable to the stomach, or by signs of organic disease in its vicinity. Usually the blood is rejected, either by a mere act of regurgitation, or in most cases by more or less violent vomiting, though it must be remembered that this act may be the cause of the bleeding. The quantity of blood discharged necessarily varies much, and it is generally more or less mixed with food and other materials. Its characters are in the majority of cases very distinctive, it being non-aërated: brown or black in colour; grumous, often resembling “coffee-grounds,” soot, or tar; and acid in reaction. Should the blood be coagulated, the clots are broken up, irregular, firm, and heavy. On microscopic examination the red corpuscles are seen to be much altered in shape or destroyed, and pigment granules are abundant. Most of these characters depend

upon the action of the gastric juice on the blood. If the blood is discharged immediately or soon after its escape into the stomach, it may be quite bright and unaltered, or only slightly changed. Commonly some of it passes on into the bowels, giving rise to tarry stools. The general symptoms indicating loss of blood will of course be present in proportion to the extent of the hæmorrhage.

DIAGNOSIS.—The most important matter is to distinguish between *hæmatemesis* and *hæmoptysis*, which can usually be done by a consideration of the following points:—1. The *age of the patient*, hæmatemesis being more frequent later in life than hæmoptysis, except in the case of young women who are the subjects of perforating ulcer. 2. The *previous and existing symptoms*, as indicating some condition likely to give rise to one or other form of hæmorrhage; and also the symptoms immediately premonitory to the attack, in the one case pointing generally to the stomach, in the other to the lungs. 3. The *mode of discharge of the blood*, whether by coughing or vomiting. It must be remembered, however, that vomiting may be excited by the cough in hæmoptysis; or some of the blood may be swallowed and afterwards rejected from the stomach. 4. The *characters of the blood*, as already described, with reference to colour, aëration, general aspect, reaction, and microscopic appearances. 5. In hæmoptysis some blood usually *continues to be discharged in the expectoration* for a certain time after the main bulk has been expelled; which is not the case in hæmatemesis. 6. Along with hæmatemesis altered blood is usually seen *in the stools*. 7. Careful *physical examination* will often reveal some organic cause likely to give rise either to pulmonary or gastric hæmorrhage; and, in connection with the former, there may be *râles* indicating the presence of blood in the bronchial tubes.

As regards the *cause* of hæmatemesis, this can only be made out by a thorough consideration of the case in all its details. Blood coming from above may be usually detected by local examination of the throat and nose. It is necessary to warn against mistaking the colour due to altered bile or iron for that of blood.

TREATMENT.—The principles of treatment in hæmatemesis are the same as for other hæmorrhages. In addition to bodily rest, the stomach must be kept in a state of absolute repose in severe cases, nutriment being administered only by enemata; in less dangerous cases very small quantities of cool or iced liquids being alone permitted. The patient should swallow small lumps of ice at frequent intervals. The most efficient medicines are gallic acid or acetate of lead in full doses, combined with opium; pyrogallic acid; hamamelis; oil of turpentine; tincture of steel; ergotine, ergotinine, or sclerotic acid subcutaneously. It is necessary to warn against giving any medicines by the stomach if they are not actually required, or if they seem to cause irritation. Ice may be applied carefully over the epigastrium. It is very important to check any violent efforts at vomiting, by means of hydrocyanic acid with mucilage, morphia internally or by subcutaneous injection, or an enema containing tincture of opium; at the same time a sinapism being applied over the epigastrium. In cases of capillary hæmorrhage, dependent on congestion of the stomach from portal obstruction, a *saline purgative* is useful, or an aperient enema. Should stimulants be required, they are best administered by enemata. Vicarious hæmorrhage must be treated according to ordinary principles.



## V. DYSPEPSIA—INDIGESTION.

**ÆTIOLOGY.**—Difficulty and imperfection in the digestive process arise under a great variety of circumstances, either in connection with the stomach, the intestines, or both; and affecting all articles of diet alike, or only special elements of food. In ordinary language the terms *dyspepsia* or *indigestion* have a very indefinite meaning, but are supposed to signify a group of symptoms depending upon interference with the *gastric* digestion, and in this sense the subject will at present be alone considered. In many instances such symptoms are merely due to *functional* disturbance of the stomach, or at all events no obvious organic disease can be detected, and it is to this class of cases that the terms are often limited; the same symptoms, however, are commonly associated with different forms of *organic* mischief, and in the subsequent remarks on this subject it will be impossible to avoid alluding to these lesions. It will thus be obvious that *dyspepsia* is not an independent disease, and to regard it as such is a serious error.

The causes of dyspepsia in general may be conveniently grouped under certain heads, according to the following arrangement:—

1. **Disorders connected with the diet**, namely, excessive eating; too rapid eating; insufficient mastication and ensalivation, this being especially associated with the habit of “bolting” food, or being due to absence or irregularity of teeth, particularly in old people; irregularity in meals, or their being taken too frequently, or the reverse; and improper quality of food. The last may depend upon the nature of the food itself; the manner in which it is cooked; or upon its having undergone fermentation or decomposition. Liquids not uncommonly cause indigestion, and special mention must be made of the habit of taking much soup; of drinking large quantities of cold water or other drinks with meals, by which the gastric juice is much diluted; of excessive indulgence in tea, or sometimes in coffee; and of abuse of alcohol, particularly when spirits are taken at frequent intervals, strong or but little diluted. Injudicious use of sharp condiments with food sometimes originates dyspepsia. Idiosyncrasy causes some individuals to suffer after special articles of diet, which are usually easily digestible, such as milk or eggs.

2. **Alterations in the gastric juice.** This secretion may be in excess; deficient, even to complete suppression; or of morbid quality. The principal changes in quality are the presence of excess of acid; deficiency of acid, pepsine, or both; admixture with abundant mucus secreted by the stomach, which may even render the gastric juice alkaline; and the addition of abnormal ingredients. These alterations result from:—*a. Organic affections of the stomach*, especially mechanical congestion; inflammation; degeneration and atrophy of the secreting glands; degeneration of the vessels; ulceration; and cancer. *b. Morbid conditions of the blood*, as in renal disease, diabetes, pyrexial conditions, gout, anæmia. *c. General want of tone, with debility.* *d. Nervous disturbance.* Dr. Fothergill believes that dyspepsia not unfrequently has a reflex origin, in connection with ovarian irritation. Dr. Allbutt regards many cases of so-called dyspepsia as being of a neurotic character, the neurosis causing disordered work and secretions in connection with the stomach.

3. **Changes affecting the movements of the stomach.** The expulsive power of the stomach may be interfered with, in consequence of want of muscular or nervous tone, dilatation, or pyloric obstruction; or its movements are irregular; or the food passes into the duodenum too speedily, before it is properly digested, either in consequence of undue excitability of the stomach, or of imperfection in the pyloric valve.

It is by influencing the *secretory* and *motor functions* of the stomach that many of the ordinary causes aid in inducing dyspepsia, such as sedentary habits; undue exertion either just before or after a meal; habitual constipation; abuse of narcotics, tobacco, tea, or alcohol; excessive study, emotional disturbance, or any form of mental shock; and venereal excesses. Most important is it to bear in mind also that dyspeptic symptoms may be entirely due to disease of some other organ than the stomach; and in any case not yielding to proper treatment, the condition of the principal organs should be thoroughly ascertained.

**SYMPTOMS.**—In the first instance it will be well to give a general outline of the clinical phenomena which are, in different combinations, observed in cases of dyspepsia; and then to indicate the special characters presented by the main varieties of the complaint.

Uncomfortable or painful sensations are experienced over the epigastrium, chiefly after meals, either due to the state of the stomach itself, or to its being irritated or distended by the materials formed as the result of the imperfect digestion. Not uncommonly these sensations are also complained of over the front of the chest, or between the shoulders. There is no tenderness as a rule. In the great majority of cases appetite is impaired or lost; some patients, however, have an inclination for food, but cannot take any, or they are obliged to confine themselves to certain articles of diet; while others, on account of the discomfort which is produced, are soon satisfied. Thirst is generally absent, but may be a prominent symptom. From the decomposition and fermentation of food result flatulent distension, with a sense of fullness and weight in the epigastrium; acidity; heartburn; and eructations. It is very important to ascertain the characters of the eructations. They consist of gases, various liquids, and undigested food. The gases are either tasteless and odourless, resulting from fermentation; or they have some peculiar smell and taste, of which the chief are those resembling fish or rotten eggs, both being associated either with deficiency or arrest of secretion, and the last being directly due to decomposition of food, and the formation of hydric sulphide. The principal liquid eructations include the watery fluid of pyrosis; and matters having an acid, acrid, rancid, or bitter taste. Acid eructations indicate either that excess of gastric juice is formed; or more commonly that the contents of the stomach have undergone acid fermentation. Butyric acid imparts the rancid characters. Bitter eructations are probably due to the presence of bile. Nausea is felt in many cases, but vomiting is not a frequent symptom, though some patients endeavour to excite the act after taking food, in order to relieve their discomfort. The bowels are usually disturbed in their functions, as indicated by constipation, or in some instances by diarrhœa; colicky pains; flatulence and borborygmi; and the passage of fœtid gas. The tongue, mouth, and throat are generally in an abnormal state, but they present different appearances in the different varieties of indigestion. The breath is also frequently offensive.

The *general* and other remote symptoms previously described as being associated with disorders of the alimentary canal are often present in variable combinations in dyspeptic cases.

VARIETIES.—The forms of dyspepsia which are usually recognized are as follows —

**I. Acute Dyspepsia.**—This may come on in an individual habitually quite free from dyspeptic symptoms; or it may be merely an exacerbation of a previously existing morbid state. It is difficult to determine precisely what the morbid condition is in many cases of acute dyspepsia, but unquestionably in a good number of such cases there is gastric catarrh; while others are merely instances of *migraine* or so-called *sick-headache*. Some cases are, however, true examples of *simple dyspepsia*, arising either from some error in diet; or as the result of interference with the secretion of gastric juice, owing to nervous disturbance from emotion, over-exertion, or other causes.

The symptoms differ much in intensity and duration, but are liable to be particularly severe in children. They come on shortly after a meal, usually in about three or four hours, and are more or less of the following nature:—Uneasiness or pain in the epigastrium, with a feeling of heaviness and fulness, or sometimes cramp-like sensations, but no tenderness; complete distaste for food; thirst; nausea, or vomiting of undigested food and other matters, such as mucus, acids, or bile, which affords relief; eructations of gases, either tasteless and odourless, or like rotten eggs, as well as of acids; heartburn; a large and moist tongue, covered with a thick white or yellow fur, and sometimes presenting enlarged and red papillæ; disagreeable taste and breath; constipation usually, but occasionally diarrhœa with colicky pains. The *general* symptoms are usually very pronounced, and there is not uncommonly a sense of extreme illness and depression, with a certain degree of pyrexia, the skin being dry. Herpes about the face or general urticaria may break out. The urine is generally concentrated, and deposits lithates; occasionally there is slight albuminuria. In infants there may be high fever or convulsions. Probably many of the cases of so-called *gastric remittent fever* in such subjects are merely examples of acute dyspepsia, attended with febrile symptoms assuming a remittent type.

TREATMENT.—The treatment of acute dyspepsia is similar to that of the slighter cases of gastric catarrh, which will be presently described. It is important to remove speedily all irritant matters, by means of *emetics* or *aperients*.

**II. Chronic Dyspepsia.**—1. *Atonic*.—Most of the ordinary cases of dyspepsia belong to this variety, being associated with general debility; anæmia; want of tone in the coats of the stomach; or sometimes with degeneration of the peptic glands. The gastric juice is deficient, and muscular activity is impaired. The sensations in the epigastrium are mainly those of weight, fulness, and discomfort after food, without actual pain or tenderness, pressure often affording relief. Not uncommonly there is in the intervals a constant sense of sinking in the epigastrium. Occasionally œsophagismus is experienced. There is a disinclination for food, and also not unfrequently for drink. Digestion is much delayed, and a quantity of foul gas is formed, as well as acids and rancid matters, there being hence much flatulence, with various eructations. The tongue is large and marked with the teeth, pale, flabby, moist, and usually more or less furred, but it may be quite clean. The



mouth and throat are also often pallid, flabby, and relaxed; and the breath is generally disagreeable. As a rule there is habitual and obstinate constipation, the stools being firm, pale, deficient in bile, and offensive. The general symptoms are well-marked usually, the pulse being feeble, wanting in tone, and easily hurried; the skin cool, soft, and clammy, with a tendency to coldness of the feet and hands: and the urine often abundant and watery. The nervous symptoms incline chiefly to languor, apathy, and indisposition for any effort. Oppression across the chest, shortness of breath, cough, and palpitation, are often complained of.

2. *Irritative*.—Probably in the form of dyspepsia thus named a condition of *chronic gastritis* is present to a greater or less degree. Actual pain or a sense of burning is experienced in the epigastrium, increased by food, and generally accompanied with a little tenderness. Heart-burn and acidity are also common symptoms. Appetite is impaired, but thirst is usually felt, especially for cool drinks. Occasionally vomiting takes place, or it is excited in order to relieve symptoms; while nausea is often felt. Eructations are frequent, but are not offensive as a rule. The tongue tends to be contracted and red, especially at the tip and edges, with enlarged papillæ: it may either be furred or clean. The throat also is frequently in an irritable condition, being reddened and granular, or sometimes presenting follicular ulcers. Though constipation is the rule, from time to time diarrhœa with colicky pains is apt to set in. The skin tends to be hot and dry, the palms and soles having a burning sensation; and sometimes a cutaneous eruption breaks out. The pulse is frequent. The urine is often concentrated and deficient in quantity, depositing lithates on standing. The nervous disturbance is chiefly in the direction of irritability and petulance, with restlessness. There may be considerable emaciation.

3. *Nervous*.—A variety of dyspepsia has been described by this term, in which the prominent symptom is pain after food, supposed to be associated with hyper-secretion of gastric juice, and observed chiefly in young women. It seems to be merely a form of gastralgia; and may either exist alone, or associated with other symptoms of dyspepsia.

4. One form of indigestion deserves special notice, which is by no means uncommonly met with, especially in out-patient hospital practice, in which there is an excellent appetite, and no particularly unpleasant sensations are felt after meals, but almost as soon as food is taken it seems to pass out of the stomach, either owing to this organ being in an irritable condition, or to incompetency of the pylorus; then rapidly traverses the intestines, giving rise to borborygmi and colicky pains; and is speedily followed by diarrhœa, the stools consisting chiefly of undigested food. Hence there is a constant craving for food, and a sense of considerable exhaustion or prostration is often experienced after the passage of a stool. In some instances this course of events only occurs the first thing in the morning; in others it follows every meal, and may thus cause serious loss of flesh and weakness. In some cases which have come under my notice the symptoms have been apparently due to the habit of excessive smoking; or to over-indulgence in hot tea.

TREATMENT.—This part of the subject will be discussed in the chapter on the treatment of chronic gastric affections.

---

## CHAPTER XXXIV.

## ACUTE GASTRIC CATARRH—ACUTE GASTRITIS.

**ÆTIOLOGY.—Exciting causes.**—1. The ordinary exciting cause of acute inflammation of the stomach is some *direct irritation* of its mucous surface, either mechanical or chemical, and set up by food or drink, foreign bodies, or poisons. Food may excite inflammation in any of the ways mentioned under acute dyspepsia. Certain irritants require special notice, namely, very hot or cold substances; alcoholic liquids, either taken in excess or insufficiently diluted; sharp condiments; tartar emetic and arsenic. It must not be forgotten that the last two have been frequently administered for poisoning purposes, and arsenic may be inhaled from certain papers used for papering rooms. 2. More or less gastric catarrh commonly supervenes in the course of various *diseases*, especially in many of the exanthemata, cholera, and yellow fever; and sometimes in diphtheria, pneumonia, puerperal fever, gout, acute rheumatism, and other febrile complaints. 3. The membrane lining the stomach may be affected, along with other mucous surfaces, as the result of *taking cold*. 4. Drinking a large quantity of *cold water while the body is heated* has been regarded as a cause of gastric catarrh. 5. It has been stated that this affection occurs occasionally as an *epidemic*, attended with pyrexia. 6. Gastritis arises in connection with *starvation*, but is then probably originated indirectly.

As regards *predisposing causes*, it may be stated that children, elderly or feeble persons, and those who habitually suffer from a disordered stomach, are more liable than other individuals to attacks of gastritis from errors in diet and other slight causes.

**ANATOMICAL CHARACTERS.**—Acute gastritis presents very different degrees of intensity, and special forms are also described, under the names *catarrhal*, *erythematous*, and *phlegmonous* gastritis. The following is a brief account of the changes which may be met with in different degrees and combinations. Hyperæmia of the membrane has been observed during life in cases of gastritis where a fistulous opening into the stomach existed, but it may completely disappear after death. The redness is punctiform or capillary, and usually in isolated patches; in cases of irritant poisoning, however, intense redness may be seen over the entire surface, though generally more marked on the top of the rugæ. Small extravasations are not uncommon. There is the usual cloudiness or opacity observed in mucous inflammations, with swelling and thickening of the membrane, and diminution in consistence. Superficial erosions or ulcerations, and follicular ulcers are visible in many cases. In exceptional instances, when the inflammation is very intense, sloughs form, or suppuration is set up in the submucous tissue. Very rarely croupous or diphtheritic deposits have been observed. The secreting structures undergo important changes. The cells and nuclei of the tubuli become enlarged and multiplied, while numerous granules and fat-globules form, so that the tubuli are distended. The solitary and lenticular glands are much increased in size. Gastric juice is not properly secreted, but the surface of the membrane is usually covered with a thick ropy

mucus, alkaline in reaction, and containing a large number of young cells. Between the glands also there is a multiplication of cells, the lymphatic tissue being increased.

It will be readily understood that the appearances vary greatly, according to the intensity and cause of the inflammation. When gastritis is the result of irritant poisoning, it often presents special characters, and deposits of the poisonous substance may be observed, or actual destruction of the coats of the stomach may take place. For a description of the appearances characteristic of the several poisons reference must be made to toxicological works.

**SYMPTOMS.**—The symptoms attending inflammation of the stomach are usually *local* and *general*, but they present wide differences in their intensity and gravity in different cases. The morbid change may vary from a slight superficial catarrh of the mucous lining to an extensive and violent inflammation, and the clinical phenomena present corresponding variations in degree.

*Local.*—Pain over the epigastrium is almost invariable; and may be very intense. In characters it is often hot and burning; or it shoots in different directions, especially towards the back. In some cases there is merely a sense of aching and soreness, or uneasiness and weight. These sensations are increased by food, by the act of coughing, or by a deep inspiration. They are often relieved by vomiting, but in some cases this act aggravates the suffering, especially if accompanied with violent retching. When the pain is very intense there may be spasm of the abdominal muscles. Tenderness is always present, even when pain is not complained of spontaneously, or there may be a sense of soreness. Nausea, vomiting, and retching are prominent symptoms, anything that is swallowed being usually rejected at once in severe cases. The vomited matters contain much mucus, saliva, often bile, and not uncommonly a little blood or “coffee-ground” material. There is complete anorexia, with urgent thirst, particularly for cool drinks. The tongue is frequently small, red, and irritable, especially at the tip and margins; or it may be furred in the centre, and smooth, with a tendency to dryness; or large, moist, and covered with a white fur, the papillæ being enlarged. The mouth is slimy, and tends to dryness; while an unpleasant taste is experienced. Constipation or diarrhœa may be present, according to the state of the intestines. The lips are sometimes the seat of herpes.

*General.*—In some forms of gastritis premonitory symptoms are observed, such as chills or slight rigors, feverishness, and general malaise. During the attack pyrexia is frequently present, though seldom to a marked degree, except in children, with restlessness, headache, nervous depression, and sleeplessness. In severe cases, and especially when the inflammation is the result of poisoning, there is often great prostration and collapse, with a cold and clammy skin, pinched and anxious features, and a very rapid, weak, and small pulse. Hiccup is sometimes a most troublesome symptom, and the breathing may be much hurried.

**DIAGNOSIS.**—The symptoms above described, if present to any marked degree, are quite characteristic of gastritis; but in mild cases, or when mere gastric catarrh occurs as a complication of febrile diseases, it may be difficult to diagnose this affection positively. The tongue often gives useful indications under these circumstances. When there is much pyrexia, constituting one form of so-called *gastric fever*, typhoid fever may be simulated at first. An important matter bearing upon the diagnosis of gastritis is the determination of its *cause* in any particular case.



When the characteristic symptoms of this complaint are present in an intense degree, it must always be specially borne in mind that they may be due to the action of some irritant poison, either accidentally introduced into the stomach, or wilfully administered.

PROGNOSIS.—Generally this is favourable, except when the gastritis is the result of poisoning; or when it assumes a severe type, and attacks persons who are weak, very old or young, or suffering from acute febrile diseases. In some cases gastric catarrh tends to become chronic.

TREATMENT.—1. If there is anything in the stomach causing irritation, the first thing to be done is to get rid of this *exciting cause*, by means of an *emetic* of sulphate of zinc, mustard, or ipecacuanha, with plenty of lukewarm water; or by the stomach-pump, if necessary. A *purgative* at the outset is often useful, such as a dose of calomel, followed by a black draught, castor-oil, or a draught containing sulphate and carbonate of magnesia; in some cases an enema may be advantageously employed. It is decidedly objectionable to administer purgatives repeatedly, but, if necessary, an enema may be given from time to time.

2. The patient should be kept *quiet in bed*, in cases of any severity, and it is most important to allow the stomach to remain in a state of rest, either complete or partial according to the severity of the attack. In dangerous cases no food should be taken by the mouth, but nutrient enemata administered instead. If food is permitted, it must be entirely of a liquid character, or only thickened with some farinaceous substance, and given in small quantities at regular intervals. Milk diluted with lime-water or soda-water, or mixed with a little arrow-root or corn-flour, weak beef-tea, or mutton or chicken-broth, are the most suitable articles of diet. The patient must be prevented from drinking large quantities of water, which is usually much craved for, but may suck small lumps of ice at frequent intervals, and this gives much relief. As a rule *stimulants* are not required, but sometimes brandy in small quantities, well-diluted, or mixed with soda-water, milk, or beef-tea, seems to be decidedly beneficial; or a little champagne with soda-water may be given. Should there be much prostration, considerable quantities of alcoholic stimulants may be called for, and if the stomach will not bear them, they must be administered by enema.

3. The prominent *symptoms* in gastritis are most effectually alleviated by the administration of *antacids* and *sedatives*. Among the most serviceable remedies may be mentioned a combination of bismuth with hydrocyanic acid and opium; iced effervescent draughts, containing carbonate of ammonia, potash, or soda, with hydrocyanic acid and tincture of cardamoms; solid opium, gr.  $\frac{1}{2}$ -i, or, better still, morphia, gr.  $\frac{1}{6}$ - $\frac{1}{4}$ ; hydrocyanic acid,  $\mathfrak{m}$  iij-v. with a little mucilage; magnesia and the alkalis, alone or in combination with some of the other remedies. One or other of these should be given at intervals of from two to four hours, according to circumstances, and it is desirable to make each dose of the medicine as small in quantity as possible. The alternation of *effervescent*s with an opium or morphia pill is frequently attended with the best results. In treating children of course due caution must be exercised in employing the powerful drugs mentioned above.

4. *Local treatment*.—In cases of severe gastritis it might be advisable to apply a few leeches to the epigastrium, but venesection is never required. The constant application of heat and moisture over the abdomen, by means of poultices, fomentations, or spongio-piline, is highly beneficial. Cold is preferred by some practitioners. Sinapisms

are sometimes of use, but more severe forms of counter-irritation are of doubtful efficacy. When gastritis arises from retrocedent gout, an attempt should be made to excite inflammation in the joints.

5. Much care is needed during *convalescence* after gastritis, as regards diet, hygienic management, and medicinal treatment. Various remedies employed in the more chronic complaints, which will be hereafter considered, are of much service if given with due precautions, such as the vegetable bitters, alkalies, acids, pepsine, and preparations of iron. The state of the bowels must be attended to, and mild *aperients* administered if required. Vichy and Seltzer waters are often beneficial, when taken in moderation.

---

## CHAPTER XXXV.

### CHRONIC DISEASES OF THE STOMACH.

#### 1. CHRONIC GASTRITIS—CHRONIC GASTRIC CATARRH.

DIFFERENT pathological conditions are included under these terms. Thus there may be merely *mechanical congestion* and its consequences; or a true *catarrhal inflammation*, which is of very common occurrence; or in some instances probably an *erythematous* condition exists. For practical purposes these different states may be conveniently discussed together. Allusion may also be made here to a fibroid change in the walls of the stomach which is sometimes met with, occasionally of considerable extent, but usually localized.

ÆTIOLOGY.—In its more or less chronic form gastric catarrh is met with:—1. Occasionally as the *sequel of an acute attack*. 2. As the result of *constant or repeated irritation of the stomach*, particularly by indigestible food, tea, alcohol, purgatives, stimulant and bitter medicines, hot condiments, and arsenic. 3. In connection with *chronic organic diseases of the stomach*, especially cancer, ulceration, and albuminoid disease. 4. From *interference with the portal circulation*, which leads to persistent mechanical congestion of the stomach. 5. Associated with *constitutional disorders*, particularly phthisis, renal disease, gout, syphilis, or any low general condition of the system.

ANATOMICAL CHARACTERS.—The colour of the mucous lining of the stomach is changed, there being increased vascularity, and the vessels may be permanently distended; frequently, especially if there has been mechanical congestion as well, portions of the surface are seen to be grey, slate-coloured, or almost black, from altered blood-pigment. Small hæmorrhagic erosions are not uncommon. The surface is often covered with a thick tenacious mucus. One of the most important changes is a thickening of the membrane, accompanied with increased firmness and toughness, sometimes so marked that it has a leathery feel, although there may be at the same time slight superficial softening. More or less opacity is also observed, some spots appearing quite opaque and white from fatty degeneration. Mammillation is a common appearance in the vicinity of the pylorus. The intimate changes which occur in chronic gastritis have been described by Wilson Fox, Fenwick,

Handfield Jones, and other observers as an increase in the interstitial tissue, including the lymphatic elements; distension of the solitary glands; alterations in the gland-structures here and there, in the way of fatty degeneration and destruction of their epithelium, thickening of their membrane, contraction and puckering, formation of minute cysts, or atrophy; and sometimes fatty degeneration of the entire membrane in spots, including the small vessels. The mammillation may be due to the enlarged glands; or to contraction of the muscular fibres which surround them.

**SYMPTOMS.**—The symptoms referable to the *alimentary canal* which are most characteristic of chronic gastritis are considerable uneasiness over the stomach, amounting in some cases to actual pain, though this is never severe, generally increased soon after meals, especially after taking hot or spiced articles; a certain degree of epigastric tenderness; a sense of heat and burning, sometimes extending over the chest; frequent heartburn with acidity, and acid or gaseous eructations; impaired appetite, the patient being soon satisfied, though there may be a feeling of emptiness and longing for food; thirst, especially for cool drinks, often particularly marked between meals and in the evenings; a small, bright-red, raw-looking, and sore tongue, with enlarged and red papillæ, or the last condition alone, there being usually more or less fur as well, though in some cases the tongue seems abnormally clean; an irritable or catarrhal condition of the lips, mouth, and throat, sometimes accompanied with aphthæ or follicular ulceration; hot and disagreeable breath; intestinal disturbances, in the way either of constipation with pale and dry stools, or of diarrhœa with lenteric stools, as well as flatulence, and colicky pains. There is often a feeling of sickness, but actual vomiting is only common in certain forms of gastric catarrh, namely, when it is associated with chronic alcoholism, renal disease, or portal congestion, sickness being then frequently a prominent symptom in the mornings and after meals. In some cases a large amount of alkaline mucus is brought up, when the affection is termed *gastrorrhœa*.

The *general system* suffers more or less as a rule, there being the various nervous and reflex symptoms previously described, often accompanied with loss of flesh and debility; sallowness or slight jaundice; a little pyrexia, especially in the evenings and after food or stimulants, accompanied with a dry and harsh skin, a sense of burning in the palms and soles, and flushing of the face. The urine is frequently disordered, depositing urates abundantly, or in some cases phosphates or oxalates. Cutaneous eruptions are not uncommon. Sometimes signs of premature decay are evident.

## II. ULCER OF THE STOMACH.

Some writers describe all ulcerations of the stomach under one group, but it is better to distinguish two chief forms, namely, (*a*) the *perforating ulcer*, characterized by its tendency to perforation; and (*b*) the *chronic ulcer*, which is attended with much thickening of tissues. There are other varieties of less importance.

**ÆTIOLOGY.**—On the whole females are more subject to gastric ulcer than males. It is most common between 18 and 25 or 30 years of age; and in advanced life. The *perforating ulcer* is most frequent in *young females*; the *chronic* in *old males*. Among the causes to which gastric ulceration has been mainly attributed are intemperance, bad living,



mental anxiety, tuberculosis, various lowering diseases, disorders of menstruation, suppression of hæmorrhoidal flux, pregnancy, and the rapid healing of cutaneous ulcers. Much doubt exists, however, on this matter, and often the morbid condition cannot be traced to any definite cause. I have known gastric ulcer follow dilatation of the stomach from pyloric obstruction.

With respect to the *pathological cause* of the disease, gastric ulcer is considered by most authorities to originate in an interference with the supply of blood to a portion of the mucous membrane of the stomach, the vitality of which becomes thus impaired, so that it is acted upon by the gastric juice and destroyed, the deeper tissues being subsequently attacked in the same way. This imperfect vascular supply may result from extensive extravasation into the tissues of the stomach; embolism; degeneration or narrowing of the arteries; or, rarely, submucous suppuration. Some pathologists regard ulceration of the stomach as being invariably the consequence of inflammation.

**ANATOMICAL CHARACTERS.**—The *perforating ulcer*, or, according to some authorities, the *early stage of ulceration*, may be seen in various stages of destruction of the coats of the stomach, beginning with the mucous membrane and extending towards the peritoneum. Its edges are even and clean-cut, as if punched out, without any thickening, and as each subsequent coat is destroyed over a smaller area than that above it, the ulcer has a somewhat conical shape, the apex being next the peritoneum, and the margin of each layer being well-defined. The floor is smooth, but may be sloughy or covered with extravasated blood.

After the *chronic ulcer* has existed for some time, its margins and floor become greatly thickened and indurated, owing to the formation of a nucleated and granular substance, which subsequently develops into imperfect fibrous tissue. The different layers become matted together over a variable extent, but the ulcer remains distinctly conical or funnel-shaped, the mucous membrane being inverted. Granulations are sometimes seen on its surface. It is important to notice that in this condition of the ulcer firm adhesions tend to form between the stomach and neighbouring organs, by which the evils of perforation are prevented.

The superficial form of gastric ulcer is usually circular or oval at first, but it may become irregular, either from extension or from the coalescence of two or more ulcers. The size generally varies from  $\frac{1}{4}$  inch to 1 or  $1\frac{1}{2}$  inch in diameter, but it may reach as much as 5 or 6 inches in length. Usually there is only one ulcer, but two or more are sometimes found, or cicatrices of former ulcers may be observed.

The most frequent seats of gastric ulcer are the posterior surface, the neighbourhood of the smaller curvature, and the vicinity of the pylorus; it is rare on the anterior surface, near the greater curvature, or at the cardiac end. Chronic ulcer is most frequent near the pylorus.

The mucous membrane around the ulcer may be quite healthy; or it presents signs of hæmorrhage and extravasation, polypoid vegetations, or acute or chronic catarrh.

*Cicatrization* frequently occurs, generally by granulation, and the cicatrix may be either smooth or puckered, or it may give rise to much thickening, contraction, and alteration in the form of the stomach, in some instances leading to stricture, especially at the pyloric end, or causing the organ to assume various distorted shapes. Sometimes a gastric ulcer does not completely cicatrize; or it heals at one spot, while it extends to another.

*Perforation* is very liable to happen if there is no thickening or adhesion, especially when the ulcer is so situated that it is subject to much disturbance by movement and distension of the stomach, or where adhesions cannot easily form, as in the anterior wall or near the smaller curvature. When perforation takes place, the peritoneum forms a small slough, and then gives way by a small sharply defined or slightly torn opening. If adhesions have formed, the coats may be destroyed completely without any immediate harm resulting, and ultimately even considerable portions of contiguous organs, such as the pancreas. In some cases the thickened peritoneum becomes distended in the form of a pouch.

**SYMPTOMS.**—Occasionally gastric ulcer is unattended with any characteristic symptoms, and its existence is only revealed by some serious event, such as perforation, or the opening of a large vessel. In many instances the clinical phenomena are for a time more or less ill-defined and obscure, especially in the chronic form of ulceration. The symptoms which are suggestive of ulcer may be thus summarized:—1. Severe localized pain in the epigastrium, of aching, gnawing, or burning character, or attended with a feeling of sickness and prostration; persistent, but increased after food, especially after certain articles, such as hot tea. 2. Local tenderness on pressure. 3. Vomiting, particularly after taking any food or drink, this act not being attended with much nausea or retching as a rule, and generally affording relief to the pain: while the vomited matter sometimes contains *sarcinæ ventriculi*, or fragments of the stomach-tissues. 4. Hæmatemesis, either due to capillary rupture or to the opening of a large vessel, and generally followed by melæna. 5. Various dyspeptic symptoms, such as flatulence, eructations, pyrosis, deranged appetite, and constipation or occasional diarrhœa. 6. More or less general wasting and debility, which may be accompanied with a dull, earthy, cachectic aspect, or in young females with a marked anæmic or chlorotic tint; the menstrual functions being also usually much disturbed in these subjects.

There are some points of importance which require comment. The exact site of the pain will vary with that of the ulcer, but it is most commonly felt a little to the right of the epigastrium; if the ulcer is on the posterior surface, the pain may be referred to the back, on one side of the spine. Movement and posture often influence the degree of suffering, and it is frequently aggravated by mental emotion, or in females during the menstrual periods. In the chronic form of gastric ulcer pressure not uncommonly gives marked relief, and hence some patients voluntarily press against the epigastrium. Occasionally food also affords ease, instead of increasing the pain. Vomiting is chiefly observed when an orifice or its vicinity is affected, especially the pyloric. The interval which elapses between the taking of food and the subsequent occurrence of sickness or aggravation of suffering will often indicate the situation of the ulcer; thus, if it is near the cardiac opening these effects are produced immediately; if about the pylorus, they only follow after some time. In some instances the pyloric orifice is permanently obstructed, and the stomach becomes consequently dilated, the signs of which will be presently indicated. No distinct tumour can ever be felt, but occasionally, when there is much thickening and induration about the pylorus, this can be made out by careful manipulation. The tongue is often abnormal, but has no special characters. Salivation is said to occur sometimes, the saliva being deficient in sulpho-cyanides.

The character and severity of the *general* symptoms will depend mainly on the intensity of the pain; the degree of interference with digestion and nutrition; and the amount and frequency of hæmorrhage. In exceptional instances of perforating ulcer pyrexia has been noticed.

The *course* and *duration* of cases of gastric ulcer are very variable. As a rule they are of a chronic nature, but occasionally the perforating variety appears to be rather acute in its progress. Many cases terminate in cicatrization and recovery; but death is also not an uncommon event, taking place either suddenly or rapidly from perforation or hæmorrhage, or gradually from asthenia.

### III. CANCER OF THE STOMACH.

**ÆTIOLOGY.**—Among *general predisposing causes* of gastric cancer age is the most important. The majority of cases occur between 50 and 60 years of age, but the complaint may be met with from 30 to 70, and, exceptionally even beyond these extremes. The male sex; hereditary tendency; a high social position; and mental anxiety are also believed to predispose to gastric cancer. As *local causes* leading to the development of the disease have been mentioned long-continued pressure over the epigastrium; injury; and the repeated action of irritants upon the stomach, such as hot spices or strong spirits.

**ANATOMICAL CHARACTERS.**—All forms of cancer are met with in the stomach, but *scirrhus* is by far the most common. It is in this organ, however, that the *colloid* variety is usually observed, and *villous* cancer has been rarely met with in the stomach. The pyloric orifice and its vicinity is the part of the stomach generally involved, but the cardiac end, curvatures, fundus, or body may be attacked. The cancer may be very limited in extent, especially *scirrhus*; or widely-spread, implicating a great portion of the walls, which is especially the case with *colloid*, and when the body of the stomach is implicated. In some cases it passes from the stomach to the œsophagus, but shows no tendency to invade the duodenum. The submucous tissue is usually the primary seat of the deposit, and it subsequently involves the deeper coats, as well as the mucous membrane partially. *Colloid*, however, according to Dr. Wilson Fox, seems to begin in the glandular structures. In most cases the morbid growth infiltrates the coats of the stomach, but encephaloid cancer is prone to form nodular masses in the submucous tissue.

The actual characters of the cancerous part will necessarily vary with the nature and amount of deposit. In most instances it will be found hard, dense, thickened, contracted, and whitish on section; but each variety presents its own peculiar characters. Not unfrequently the mucous membrane becomes destroyed, and an ulcer forms, but there may be extensive cancer without any ulceration. The ulcer has thick ragged margins, and an uneven floor, which presents cancerous masses. Adhesions often form with other organs, which may further become involved by extension; or occasionally perforation takes place into hollow viscera or other parts.

The seat of the cancer influences materially the shape and size of the stomach, and the condition of its walls. When the disease involves the pylorus, the organ becomes much dilated, and its walls are hypertrophied. On the other hand, it is contracted, shrunken, and small when



the cardiac orifice is affected. If the middle of the body of the stomach is alone implicated, the cavity is greatly constricted at this part, so that the organ assumes an hour-glass form. Cancer along the curvatures distorts the stomach in various ways by its contraction, often drawing the orifices near together. In some instances the organ is displaced considerably, owing to a mass at the pyloric end having fallen in the abdominal cavity by its own weight, and subsequently become adherent in some abnormal position. Such a mass may press on various structures, and thus lead to other morbid conditions, for instance, on the portal vein, causing ascites.

Acute or chronic gastritis and glandular degeneration are generally observed, to a greater or less extent, associated with gastric cancer. Dr. Fenwick lays particular stress on the extent of the degeneration of the gastric glands in connection with cancer. The muscular tissue is also more or less altered in its characters.

Cancer of the stomach is almost always *primary*, but it tends to involve other abdominal organs and structures, either by extension or by originating secondary deposits, the latter being particularly common in the liver.

**SYMPTOMS.**—For some time, and in some cases even to the last, the symptoms of gastric cancer are merely those of dyspepsia, with wasting; or the disease may be entirely latent. As a rule, however, there are prominent *local* and *general* symptoms.

*Local.*—Pain is generally present in some part of the epigastrium, varying with the seat of the cancer, and though at first amounting merely to a sense of weight and uneasiness, it usually becomes very intense. It may be continuous or intermittent, and is often paroxysmally increased. Food aggravates the pain as a rule, but not so distinctly as in cases of gastric ulcer, and it may even be relieved by food. In character it is frequently described as aching, burning, or gnawing, as well as lancinating, shooting towards the hypochondria, back, or shoulders.

Tenderness is almost invariable, with a feeling of soreness, even when there is no spontaneous pain, the slightest touch being sometimes unbearable. This may be associated with some evident tumour or thickening.

Nausea and vomiting are rarely altogether absent, usually becoming more frequent and distressing as the case progresses. Vomiting is particularly observed if the orifices are involved, or if there is ulceration, and the time of its occurrence with reference to the taking of food varies according to the seat of the cancer, in the same manner as has been described when speaking of ulceration. The rejected matters not uncommonly contain numerous *sarcinae ventriculi* and *torulae*, as well as occasionally cancerous elements; when there is ulceration they may be very offensive.

Hæmatemesis is a very frequent and early symptom of gastric cancer, but usually to a small amount. Large hæmorrhages are stated only to occur in the later stages, and not to be so common as in connection with ulcer, but the former statement is certainly not always true, according to my experience. Melæna is often observed at the same time, or even where there is no hæmatemesis.

Appetite varies, but tends to become deficient or lost from an early period. The tongue has no constant characters. Among other frequent symptoms must be mentioned flatulence; gaseous eructations, at first

odourless, afterwards often foetid; acidity; gastrorrhœa; obstinate constipation; and hiccup.

*Physical examination* may reveal one or more of the following conditions, and it should in all suspected cases be carefully and repeatedly made, especially when the stomach is empty:—1. A sensation of *fulness and resistance over the epigastrium*, perhaps not uniform, detected by manipulation and percussion, the sound produced by the latter being somewhat dull and muffled. This indicates extensive infiltration of the walls. 2. A *distinct tumour*, especially in connection with scirrhus of the pylorus. Its site is usually the right hypochondrium or epigastrium, but it may be felt in the iliac fossa owing to displacement of the stomach, or in females near the umbilicus. The tumour is small, circumscribed, dense, hard, and irregular. Not usually movable on manipulation, it sometimes alters its position with change of posture, and with varying degrees of distension of the stomach. There is dulness on percussion over the tumour, which may, however, be modified by the stomach-sound. Aortic pulsation may be transmitted through it. It is said that a cancerous tumour of the stomach may disappear completely by sloughing or ulceration. 3. *Dilatation of the stomach*, due to pyloric obstruction. 4. *Retraction of the abdomen*, which may even assume a concave form, and when this condition is present, a tumour can be more easily detected, and may even be visible. 5. On the other hand, in exceptional instances a pyloric tumour presses on the portal vein, and thus causes *ascites*.

*General*.—The constitutional symptoms are as a rule very pronounced in gastric cancer, namely, early and rapidly-progressing emaciation and debility, ultimately often becoming extreme; signs of the cancerous cachexia, the skin being dry and harsh, with a dirty, sallow, or earthy hue, and the features sunken and pinched; marked anæmia, especially if much blood has been lost, with a tendency to œdema of the legs, or sometimes to thrombosis; great weakness of the heart and pulse; lowness of spirits, with a melancholic and anxious expression, or irritability and moroseness; and disturbed sleep. Occasionally jaundice is observed, owing to pressure on the common bile-duct. In the later stages the temperature is now and then a little elevated, but pyrexia is absent as a rule. The urine has been found to contain excess of indican, and also peptones in some cases.

The *course and duration* of gastric cancer are subject to some variations. Generally the progress is continuous and rapid; sometimes there are slight or even marked remissions in the symptoms, but these seldom last for any length of time. Cases rarely extend beyond two years from the first appearance of symptoms; and the average duration is said to be a little over a year.

#### IV. PYLORIC OBSTRUCTION—DILATATION OF THE STOMACH— PYLORIC INCOMPETENCE.

*Ætiology*.—The pylorus may be obstructed either from some morbid condition of the stomach itself, inducing stricture or stenosis; or from external pressure. The following list includes the main causes:—1. *Cancer of the pylorus*, especially scirrhus, which is by far the most frequent cause. 2. *Cicatrization of an ulcer*. 3. *Corrosive poisoning* and its results. 4. *Hypertrophy of the coats*, with thickening of the sub-mucous tissue, and fibroid changes involving the walls. 5. *Spasmodic*

contraction of the muscular coat, due to an ulcer in the vicinity. 6. *External pressure* from—*a*, tumour of the pancreas; *b*, cancerous masses projecting from the liver; *c*, enlarged glands in the vicinity; *d*, very rarely a tumour connected with the gall-bladder. 7. *Displacement* of the stomach by adhesions, and dragging down of the pylorus.

Pyloric obstruction causes the stomach to become dilated, while its walls hypertrophy, especially the muscular coat, in the endeavour to overcome the interference with the passage of the food, the intestines at the same time being contracted.

Dilatation of the stomach may also result from obstruction of the duodenum, or an ulcer just beyond the pylorus; or, rarely, from constriction of the upper part of the jejunum. A certain degree of distension is due sometimes to deficient tone of the muscular coat of the organ, from weakness, want of proper innervation, or chronic catarrh; and a form of *acute* dilatation is met with in rare instances. In one case it was attributed to drinking a large quantity of effervescing liquid. As exceptional causes should be mentioned paralysis of the gastric walls, interfering with the expulsion of food, due to injury to the splanchnic nerves, or to a fibroid change in the muscular coat; hernia of the stomach through the diaphragm, or its displacement by an omental hernia; and accumulations of foreign substances in its interior, such as hair. Dilatation of the stomach has been attributed to the presence of *sarcinæ*, but this is extremely doubtful.

Probably a condition of *pyloric incompetence* is sometimes present, in consequence of which the food passes too readily out of the stomach, either immediately or before it is properly digested. This incompetence may be due to destruction of the tissues about the pylorus by malignant disease or ulceration; or to paralysis of the sphincter.

**SYMPTOMS.**—In cases of pyloric obstruction there may be evidences of some organic disease of the stomach about the pylorus, or of some morbid condition in its vicinity causing pressure; but the only positive clinical indications of chronic dilatation of the stomach are derived from the *characters of the vomiting*, and of the *matters rejected*; accompanied with the physical signs of a *dilated stomach*. The vomiting comes on some hours after food, or may only occur at intervals of a few days, a great quantity being then discharged. The vomit never contains bile, but is strongly acid, presents numerous *sarcinæ* and *torulæ*, and readily ferments. The stomach may be more or less enlarged, occasionally so much so as to cause general distension of the abdomen. The *physical signs* of this enlargement are as follows:—(i.) The *shape of the stomach* may be retained, and made out by careful examination: while movements of the organ can sometimes be felt or excited by the hand. (ii.) On *succussion* a splashing sensation is often experienced, when the stomach contains liquids. (iii.) *Percussion* reveals extension of the stomach-note upwards, as well as downwards, if the organ is empty. If it contains food or fluid, however, there is dulness below, as after taking a good draught of water; and this dulness may be made to alter its position by changing the posture of the patient. (iv.) If a *probang* is passed by the œsophagus, it may reach the bottom of the stomach, and be then felt through the abdominal walls. (v.) Emptying the stomach by means of the *stomach-pump* may afford some aid in diagnosis. (vi.) *Auscultation* may reveal a splashing sound on succussion; the sound of food or liquid falling into the stomach, when swallowed; or loud heart-sounds reverberating through the space. (vii.) The heart may be dis-



placed upwards. Occasionally the patient experiences a sensation as if the food passed too low down in the abdomen.

In acute gastric dilatation the abdomen enlarges rapidly, and the physical signs of the dilatation are discovered on examination. There is usually severe and abundant vomiting.

*Pyloric incompetence* leads to digestive disorders in the intestinal canal, usually accompanied with diarrhœa, undigested food being passed in the stools; while the general nutrition is liable to become much impaired. It is for the detection of this condition that the plan already mentioned, of introducing into the stomach substances which cause effervescence and the formation of gas, is resorted to.

## CHAPTER XXXVI.

### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT OF CHRONIC GASTRIC AFFECTIONS.

#### I. DIAGNOSIS.

It will be most convenient to consider the diagnosis of stomach disorders according to certain groups in which they are presented in ordinary practice.

1. Many cases come under observation evidently belonging to the class of **dyspeptics**. It is then requisite to determine what *form* of dyspepsia is present; and to what *causes* this is due. The two chief varieties to be distinguished from a clinical point of view are the *atonic* and *irritative*, which are mainly separated from each other by the difference in the intensity and characters of the sensations in the epigastrium; the conditions of the tongue, mouth, and throat; the absence of thirst in the atonic form; the minor degree of general disturbance in the same variety, this being also of a different nature. Further, diarrhœa is often present in the irritative form; while in atonic dyspepsia constipation is generally a prominent symptom. With regard to the *causes* of any dyspeptic symptoms, these must be ascertained by a satisfactory inquiry into the history and habits of the patient; and a thorough investigation as to the condition of the various organs, as well as of the general system. It is particularly necessary to recollect that such symptoms may depend upon a state of gastric catarrh kept up by portal congestion, or upon some disease affecting the constitution, especially Bright's disease; or that they may mark the early stage of some serious organic affection of the stomach. In order to see what elements of food are not properly digested, it has been proposed to evacuate some of the contents of the stomach at certain intervals by the aid of the stomach-pump, and thus to obtain them for personal examination.

2. It is frequently difficult to diagnose between mere **functional dyspepsia** and the less serious **organic affections**, namely, glandular degeneration, and chronic gastric catarrh or gastritis. The opinion has

been already expressed that many cases of so-called irritative dyspepsia are merely those of chronic gastritis, and it is scarcely practicable to draw a line between them.

3. Young women not unfrequently complain of *severe gastric pain*, in whom it is difficult to determine with certainty whether they are suffering from mere **gastralgia** or **nervous dyspepsia**, or from **perforating ulcer**. In any really doubtful case it is safer to diagnose the latter affection. The chief distinctions are that in gastric ulcer the pain is usually more localized, and is almost always much increased by food; there is a sense of soreness and deep tenderness, but often less superficial hyperæsthesia than in the other affections; vomiting occurs after food, usually affording relief, and there may be hæmatemesis; emaciation is generally well-marked; while there are none of the neuralgic pains in other parts, or signs of hysteria, so commonly associated with gastralgia and nervous dyspepsia. It may be mentioned here that the *gastric crises* which occur in certain cases of locomotor ataxy are liable to be mistaken for mere gastralgia.

4. In persons advanced in years more especially, but occasionally in younger individuals, symptoms are not uncommonly present which render the diagnosis between mere **functional disorder** and **grave organic disease** of the stomach—either *chronic ulcer* or *cancer*—for a time very doubtful. Persistent disturbance of digestion without any evident cause, and not yielding to proper treatment, should lead to the suspicion of the existence of serious organic disease, though it must be remembered that the symptoms may be due to glandular degeneration. The diagnosis would be rendered more positive by the presence of marked pain in the stomach, increased by food; localized tenderness; vomiting; hæmatemesis; and progressive emaciation. Some of these symptoms, however, especially pain, I have known to be very intense in connection with mere functional disorder in females and gouty subjects. With regard to hæmorrhage, it is important to observe that the blood, if in small quantity, is sometimes not vomited, but passed entirely by the stools, and therefore in any doubtful case it is desirable to examine the fæces.

5. The diagnosis of **chronic ulcer** from **cancer** is frequently very difficult at first. The circumstances in favour of the latter are:—the patient being a male and advanced in years; a hereditary history of cancer; pain more constant, and less influenced by food and vomiting; hæmorrhage not occurring in the earlier stages, but late in the case, and being on a small scale; marked and early digestive disturbances, appetite being much impaired; considerable and rapid wasting, especially if independent of vomiting or loss of blood; and evidences of the cancerous cachexia. Later on, the discovery of a tumour, especially near the pylorus; signs of obstruction of the pyloric orifice, with dilatation of the stomach; inefficiency of treatment; the almost continuous and speedy progress of the case; and perhaps signs of cancer in other parts, usually serve to render the diagnosis certain. It is necessary to guard against mistaking a contracted rectus abdominis for a tumour.

With regard to the *part of the stomach* involved, this can often be made out by noting the exact seat of the pain and tenderness; the relation of pain and vomiting to food and posture; the state of the stomach, as to whether the organ is contracted or dilated; and the locality of any physical signs which may be discovered.

6. Affections of the stomach may simulate **neighbouring diseases**, or *vice versâ*, especially disease of the duodenum or head of the pancreas; cancer in the small or large omentum; hepatic affections; the passage of a gall-stone; disease of the absorbent glands in the vicinity; affections of the transverse colon; and painful conditions of the abdominal walls. In any doubtful case a correct diagnosis can only be arrived at by a careful consideration of all its details.

7. Occasionally **rare conditions** in connection with the stomach give rise to much difficulty in explaining symptoms associated with this organ. Of such which have come under my own notice I may mention abscess in the walls of the stomach; and a hernial protrusion of this organ through the diaphragm. On the other hand, it must be remembered that extensive organic disease may exist, even cancer involving a considerable portion of the gastric walls, without any, or with only slight local symptoms, there being merely profound interference with the general nutrition.

## II. PROGNOSIS.

1. The prognosis in any case of **dyspepsia** will depend mainly on the time the symptoms have lasted; their causes, and whether these can be removed; and the ability and willingness on the part of the patient to submit to appropriate treatment. When indigestion has become a chronic and habitual condition, the complaint is frequently very difficult to cure, especially if it is associated with permanent organic changes in the coats and glands of the stomach, with conditions keeping up venous congestion, or with some general disease; or if the patient persists in pernicious habits. Most cases may, however, be restored to health if properly attended to, or at all events they may be much improved. If there is reason to believe that the mucous coat of the stomach, with its glands, has undergone serious morbid changes, especially after the abuse of alcohol, the condition is highly dangerous, owing to the interference with digestion and nutrition thus brought about, and the patient may gradually sink from marasmus. Persistent dyspepsia occurring in the course of various chronic diseases often materially increases the gravity of their prognosis.

2. **Gastralgia** is frequently difficult to get rid of, and may prove serious, especially in elderly persons, on account of the severity of the pain, and because this prevents them from taking food.

3. **Gastric ulcer** is obviously a dangerous lesion. The variety which occurs in young women is the more immediately grave, on account of its tendency to perforation and hæmorrhage. A large proportion of presumed cases of perforating ulcer, however, recover, the ulcer being supposed to cicatrize. The chronic ulcer shows much less disposition to heal, but is not nearly so liable to lead to a speedily fatal issue, death usually taking place slowly by asthenia. In proportion to the severity of the stomach-symptoms—pain, vomiting, and hæmorrhage—is the prognosis more grave. The effects of cicatrization may also prove serious.

4. **Cancer** is necessarily a fatal disease, and the question can merely be one of duration. This must be determined in each particular case by the characters of the symptoms; and the progress of the malady. Rarely does the duration extend beyond two years, and generally it is much shorter than this.



## III. TREATMENT.

There are certain obvious indications to be followed in the management of all chronic gastric affections, and these will now be considered, anything calling for special notice being pointed out in the course of the remarks.

1. Regulation of the **diet** evidently demands the first consideration in all cases. This involves not merely directions as to what kinds of food should be taken, but also with regard to quantities, intervals between meals, proper mastication, and other matters. In many cases of dyspepsia this regulation of diet is the chief thing needing attention, without which all other means are usually unavailing. It would occupy too much space to point out all the modifications of diet required in different forms of stomach derangement, and, indeed, this has generally to be determined by the experience of each individual patient. There are, however, certain broad rules which may be laid down. In all cases substances which are known to be indigestible, such as pastry, cheese, many fruits and vegetables, fresh bread, and most made-dishes, must be avoided, plain food only being taken. Meat is decidedly beneficial in atonic dyspepsia, fresh beef and mutton, not too fat and properly cooked, being the kinds of meat which are most suitable. Pork, veal, and salted meats must be forbidden. In proportion to the degree of irritation present, and especially should there be distinct evidence of chronic gastritis, does it become requisite to order a more bland and easily digestible diet. In such cases white fish, light soups, chicken, game, jellies, calves' feet, sweetbread, the yelk of eggs, milk-puddings, and articles of this kind answer best, and it is often better to give food in small portions at rather frequent intervals, than in any considerable quantity at a time. Care must always be exercised in the use of hot condiments.

The question of *drink* calls for special notice. Many patients suffer from abuse of tea, and it is frequently desirable to forbid this beverage absolutely, and order cocoa or milk instead, the latter being advantageously mixed with lime-water or soda-water. Instructions must also be given against taking large quantities of cold water, especially during meals. Any abuse of alcoholic drinks must of course be at once put a stop to. A glass of good bitter ale or stout with meals is often beneficial in atonic dyspepsia, provided it does not produce flatulence; a moderate quantity of wine may also be useful, just before and with food, especially dry sherry, champagne, claret, or hock. In some cases a small quantity of good brandy or whisky, well-diluted, answers best. When there is an irritable condition of the stomach much care must be exercised in the use of stimulants.

In *functional disorders* of the stomach attended with *severe pain after food* it is sometimes requisite to insist upon patients taking their meals properly, as they will otherwise go without food, and are thus only aggravating the mischief; underdone meat is beneficial in many of these cases, and it may be pounded. Where mastication is impracticable, food must be artificially divided before it is taken; and in the case of old people and others who have lost their teeth, as well as in some instances where individuals have very irregular teeth, it is often extremely serviceable to provide them with an artificial set.

In treating *gastric ulcer*, especially the *perforating* variety, diet is all-important. In order to promote the healing process, and to guard against untoward events, one main indication is to keep the stomach in as absolute a condition of rest as possible. Hence anything which is in the least degree liable to irritate this organ, or to give rise to flatulent distension, must be entirely avoided. Further, the food should consist of liquids or pulpy materials, such as thick soups, pounded underdone meats, or meat-extracts; milk, either alone or thickened with corn-flour or arrow-root; and the yolk of eggs beaten up or soft-boiled; and even these must be given only in small and regulated quantities at stated intervals. In treating *perforating ulcer* it has with good reason been recommended that the patient should be kept in bed for some weeks, so that less nutriment may be required, and thus the stomach be less disturbed. Some have even gone so far as to advocate that the system should be entirely supported by nutrient enemata, but this is rarely required, unless serious symptoms should supervene, such as uncontrollable sickness or hæmatemesis.

In connection with *cancer* no definite rules can be laid down, but as digestible and nutritious food as possible must be given, each case being managed on its own merits. It may not be altogether useless to remember that, both in cancer and ulcer, the position assumed during and after the taking of food has sometimes a decided influence in the way of relieving pain and other symptoms.

In some cases of gastric disease it is requisite to be particular as to the *elements of food* which are permitted. Thus, if there is much tendency to acid fermentation, starchy substances are contra-indicated. A most valuable element in the treatment of many cases now recognized is to subject the food to *artificial digestion*, or to *peptonize* it, either partially or completely, before it is taken. Many foods specially prepared in this way are also now sold. This artificial digestion is also of great importance in relation to *nutrient enemata*, the materials being peptonized before they are injected into the rectum. *Liquor pancreaticus* is chiefly used for the purpose, as recommended by Dr. William Roberts. This subject will be again referred to.

2. The next matter requiring attention is **general hygienic management**. Many cases of functional disorder of the stomach, as well as of chronic catarrh, and even of ulceration, are greatly benefited by attention to various matters coming under this head, of which only the chief can be here mentioned, without entering into details, namely, the taking of a proper amount of exercise, though not immediately before or after a meal; avoidance of undue mental work, harassing anxiety, and brooding over symptoms; mingling in cheerful society; change of air and scene; abstinence from injurious habits, such as intemperance or excessive smoking; promotion of the action of the skin by cold bathing or douching, if this agrees, or by an occasional warm bath or Turkish bath; and the wearing of warm clothing, with flannel next the skin.

3. Coming, in the next place, to **medicinal treatment**, first, those therapeutic agents require notice which *act directly upon the stomach*. These must not be given indiscriminately, and much care is required in their administration. They act by increasing the appetite; by giving tone to the stomach and aiding its muscular contractions; by promoting the secretion and improving the quality of the gastric juice, or, on the other hand, checking excessive secretion; or by producing a sedative effect upon the stomach. The chief remedies include *alkalies*, namely



liquor potassæ and the carbonates of soda, potash, or ammonia; *mineral acids*, especially hydrochloric, nitro-hydrochloric, and phosphoric; tincture or extract of nux vomica, or strychnia; cinchona or quinine, which, however, must be used with particular caution, as they are apt to disagree; *vegetable and aromatic bitters*, namely, calumba, gentian, orange-peel, quassia, chiretta, cascarrilla, chamomile, absinthe, rhubarb, aloes in small doses, and hop; *carminatives and stimulants*; carbonate, nitrate, or oxy-chloride of bismuth, or liquor bismuthi. These may be variously combined, and among the most useful combinations may be mentioned bicarbonate of soda with tincture or infusion of calumba or gentian, and aromatic spirits of ammonia; dilute hydrochloric acid with the same bitters, or with tincture or infusion of orange-peel, to which tincture of nux vomica (m v-x) may often be very advantageously added; bismuth with carbonate of soda and some bitter infusion. Hydrocyanic acid is in many cases a useful addition to these mixtures. When giving the vegetable bitters it is desirable to begin with a moderate dose, as they are sometimes apt to disagree at first. Medicines which promote secretion are best administered shortly before or during meals. Alkalies certainly act in this way; acids may be used to check excessive secretion, when given just before meals, but their continued administration also seems to increase and improve the quality of the gastric juice, by improving the condition of the lining membrane of the stomach and its glands. Ipecacuanha, in doses of gr.  $\frac{1}{2}$  to i in pill, has been recommended as a promoter of secretion; and also hot condiments and other stimulants, especially capsicum. The last-mentioned are useful in some cases, but the habit of always taking alcoholic stimulants before meals is decidedly to be deprecated.

When there are evidences of much *gastric irritation* most benefit is usually derived from the use of preparations of bismuth with alkalies and hydrocyanic acid, to which solution of morphia (m iij-x) may be added, should there be much pain. Sometimes, however, mineral acids act well in this condition; and occasionally, even when there have been distinct signs of subacute gastritis, I have known strychnine to be the only remedy affording relief. In cases of continued gastrorrhœa some of the most efficacious medicines are oxide of zinc, oxide or nitrate of silver in minute doses, and *vegetable astringents*. Arsenic is also recommended. Opium or morphia are of great value in some morbid states of the stomach, particularly in that condition which is due to chronic alcoholism. Dr. Wilson Fox has found the compound kino powder very useful in these cases. In that variety of indigestion in which the food passes immediately out of the stomach, I have found most benefit from the administration of a salt of bismuth shortly before meals, combined with tincture of opium (m iij-vi).

The employment of remedies as *substitutes for the gastric juice*, or so-called *digestants*, has been now well-established as a method of practice, chiefly by the experiments and observations of Dr. William Roberts, from whose writings on the subject full information may be obtained. The principal agents thus used are pepsine in various forms, hydrochloric and lactic acids, liquor pepticus and liquor pancreaticus (Benger), pancreatine, ingluvin, lactopeptine, papayine, and maltine or malt extracts. They are either taken along with or after the food; or this may be more or less digested beforehand, especially by means of liquor pancreaticus. As already stated, materials employed as nutrient enemata may also be digested before administration, or liquor pan-



creaticus may be merely mixed with them, and digestion allowed to take place in the rectum. Suppositories made of artificially digested meat, originally introduced by Dr. Spencer of York, may be employed with advantage in many cases.

The drugs thus far considered are often of service in cases of *cancer* and *chronic ulcer*, and either of them may be tried, should symptoms seem to indicate a necessity for its administration. With regard to medicines which directly promote the *healing of an ulcer*, nitrate and carbonate of bismuth are supposed to act thus (gr. x every four or six hours), and also nitrate or oxide of silver; either of these may be combined with opium or morphia. There is no known remedy which has the least direct influence upon cancer of the stomach.

4. **Symptomatic treatment** almost always demands attention in the various stomach-complaints. The chief symptoms which call for interference are *pain*; *nausea* and *vomiting*; *heartburn* and *acidity*; *flatulence*; *eructations*; *pyrosis*; *constipation* or *diarrhœa*; and, in certain cases, *hæmatemesis* or *perforation*. The treatment of most of these has already been fully considered. Pain may be relieved by opium, morphia, hydrocyanic acid, belladonna, conium, spirits of chloroform, or chloral internally: with external applications over the epigastrium, namely, dry or moist heat; cold in some cases, especially of cancer; anodyne fomentations; turpentine stupes; sinapisms; or, if the pain is continuous, a small blister, which may be dressed with morphia, or a belladonna or opium plaster. Should the pain be severe, subcutaneous injection of morphia may be required. Frequently painful sensations are associated with flatulence or acidity, being relieved on removing these conditions. Flatulence and eructations are usually much diminished by the use of the medicines already considered, which act upon the stomach and food. Charcoal alone, or a combination of bismuth with freshly-prepared charcoal, given after meals, are often efficacious in preventing or relieving flatulence; as well as such remedies as assafœtida, galbanum, musk, valerian, sumbul, spirits of ammonia, oil of rue or cajeput, and peppermint. Sometimes small doses of creosote, carbohc acid, sulphocarbonate of soda (gr. x-xv), or hyposulphites may be given with benefit. Acidity and heartburn are best relieved by the bicarbonate of soda or potash, or by magnesia or carbonate of magnesia before meals. Frequent acid eructations are generally an indication for *mineral acids*, when these depend on excessive formation of gastric juice; they may, however, be due to fermentation of food from deficiency of this secretion, and the consequent formation of various organic acids. Pyrosis is usually checked by bismuth in full doses, combined with alkalis; or compound kino powder may be employed with advantage in some cases. *Aperients* are often required, but should there be habitual constipation, it is very important to avoid the constant employment of strong purgatives, if possible. Vichy, Seltzer, Friedrichschall, Hunyadi Janos, Esculap, Rubinat, and other mineral waters are often very serviceable for relieving this symptom.

5. It must not be forgotten that the state of the **general system**, as well as that of the main **organs**, needs due consideration in all cases of stomach disorder. Many of the remedies already alluded to act as general *tonics*, and thus aid in improving digestion. Among the conditions requiring particular notice are anæmia, which must be treated by mild ferruginous preparations; hepatic derangements, calling for an occasional dose of some mercurial preparation or podophyllin; gout;

hysteria; malarial affections; and renal disease. Gastralgia is often much benefited by the administration of iron, strychnia, and various other *nervine tonics*.

6. In the management of those conditions in which the stomach is much dilated, it has been recommended to use the stomach-pump systematically; and also to wash out the organ, according to Kussmaul's method, using various injections, such as strong alkaline solutions, Vichy water, or antiseptic solutions. Similar treatment has been advocated in cases of chronic catarrh. The results of experience during recent years seem to point to the possibility of considerable advantage being derived in suitable cases from the removal by operation of limited cancer of the stomach.

## CHAPTER XXXVII.

### ON CERTAIN INTESTINAL SYMPTOMS AND FUNCTIONAL DISORDERS.

#### I. ENTERALGIA.

**ÆTIOLOGY.**—Enteralgia is one of the recognized forms of visceral neuralgia, and is defined by Dr. Wardell as “a painful affection of the intestines, of neuralgic character, generally accompanied with constipation and flatus.” Dr. Clifford Allbutt, in his *Gulstonian Lectures*, also speaks of this malady as “one of singular character and marked identity”; but he further remarks, “as a matter of speculation, I hesitate to place the seat of enteralgia in the bowel at all.” The *pre-disposing* and *exciting causes* of the complaint are in the main similar to those which originate gastralgia. Local causes, such as flatulence, undigested food, accumulated fæces, &c., are mentioned by Wardell and other writers, but it certainly appears that in the painful attacks thus produced there is an element of intestinal colic, as well as in those resulting from lead-poisoning. Wardell states that females are more prone to enteralgia than males, but Allbutt's experience is opposed to this statement, who found that of fifteen cases, eight occurred in men, and seven in women. He accounts for the greater relative frequency of enteralgia in men than of gastralgia by its being more directly allied to gout, and by its dependence on the weightier affairs of men. He observes that “enteralgia is aroused rather by the strife of public life than by the teasing of homely worries.”

**SYMPTOMS.**—The essential symptom of enteralgia is abdominal pain, coming on in paroxysms, usually sudden, in some instances more gradual. The attacks are generally irregular in their onset, sometimes distinctly periodic. The pain is said to begin in the majority of cases in the umbilical region, or in the right flank, but it moves about to other parts of the abdomen. It varies in its character, being described as sharp, shooting, piercing, twisting, drawing or tightening, rolling, aching, &c.; and in some instances it has been of the most agonizing intensity. In the severer cases more or less collapse and subsequent prostration remain. Wardell states that enteralgia is frequently accompanied with constipation, accumulation of fæces, and flatulence,

but Allbutt regards the complaint in typical cases as being uncomplicated with any intestinal derangement. Diarrhoea is occasionally observed; and also vomiting in rare instances. There may be other associated visceral neuroses; and also neuralgia affecting the spinal nerves.

TREATMENT.—The treatment of enteralgia consists in relieving the painful paroxysms by means of *anodynes*, with the application of heat and other measures which alleviate pain; in the intervals attending to the alimentary canal and to the digestive functions, and adopting the general treatment suitable for neurotics, with change of air, and tonics. Dr. Allbutt recommends quinine and belladonna; arsenic is not so useful as in gastralgia.

## II. INTESTINAL COLIC.

ÆTIOLOGY.—Intestinal colic is a painful affection, attended with irregular spasmodic contractions of the muscular coat of the bowel. Its causes are:—1. *Direct irritation of the bowels* by improper or undigested food; cold drinks or ices; irritant, acrid, or poisonous substances; excessive or morbid secretions, especially bile; retained fæces, colic being often associated with constipation and flatulence; and foreign bodies, such as fruit-stones, gall-stones, or worms. 2. *Organic diseases* of the intestines; and the different forms of *intestinal obstruction*. 3. *Reflex irritation*, as from ovarian and uterine affections; or during the passage of a renal or hepatic calculus. 4. *Morbid conditions of the blood*, especially in gout, and perhaps rheumatism. 5. *Lead-poisoning*. 6. *Disorders of the nervous system*, particularly in connection with hysteria, or as the result of strong emotion. 7. Occasionally *exposure to cold*, either generally or locally.

SYMPTOMS.—The symptoms of intestinal colic are usually quite characteristic. Paroxysmal pain is felt in the abdomen, often coming on quite suddenly, and presenting remissions or intermissions. It generally begins and is most severe about the umbilical region, but may spread over the entire abdomen, and is liable to change its site constantly. As a rule the pain is of considerable intensity, being sometimes most excruciating during the exacerbations, while in character it is more or less twisting, pinching, or constricting, what is commonly termed *gripping*. Pressure almost always gives marked relief, the patient either bending forwards and pressing with the hands, or lying upon the abdomen; at the same time being very restless, rolling and tossing about from time to time. Should the spasm continue for a long time, a little soreness may be left. Ordinarily the bowels are constipated, and distended with flatus; diarrhoea may be present, however, in some conditions. Occasionally vomiting takes place, but then probably the stomach is affected. *Physical examination* generally reveals flatulent distension, except in lead-colic; while the spasmodic movements of the bowels, and rolling about of flatus, can often be felt. The abdominal muscles are also commonly in a state of rigid contraction, or they may be knotted here and there.

The patient presents an expression of suffering, and if the pain is very severe and prolonged, there may be signs of more or less collapse. Pyrexia is absent. The attack lasts a variable time, and



usually ends abruptly, being followed by a feeling of great relief and comfort.

**TREATMENT.**—The first thing to be attended to is to find out the cause of an attack of intestinal colic, and to get rid of this. A free *aperient enema* is generally useful, to which may be added some turpentine or assafoetida, if there is much flatulence; or a brisk *purgative* may be given by the mouth in less urgent cases, such as a full dose of castor oil, either alone or preceded by calomel, a black draught, or one containing sulphate and carbonate of magnesia with peppermint-water. Opium is the chief remedy for the relief of pain and spasm; it is best given in the form of tincture or liquor opii sedativus, which may be combined with spirits of chloroform and tincture of cardamoms. In severe cases subcutaneous injection of morphia may be employed. Warm *carminative* drinks are also beneficial; or a little hot spirit and water may be given. Should the attack be associated with hysteria, a draught containing tincture of valerian or assafoetida is indicated. The patient should be kept warm; and the assiduous application of dry heat over the abdomen, with friction, will usually afford great relief. In some cases hot fomentations answer best. Any signs of collapse must be combated by *stimulants*. It may be remarked that infants probably often suffer from intestinal colic, on account of improper feeding. This may be prevented by careful attention to their diet; but should it arise, *carminative waters* may be given, along with magnesia or a little castor oil, and heat applied over the abdomen.

### III. CONSTIPATION.

**ÆTIOLOGY.**—The immediate causes of this very common symptom may be summed up as:—1. *Mechanical obstruction* in some part of the alimentary canal, directly interfering with the passage of the fæces. 2. *Deficient peristaltic action* of the intestinal muscular coat, especially of that of the large bowel, generally due to impaired excitability of the nerves. 3. *Deficiency of secretions*, particularly of the intestinal secretion and bile; or, as some believe, their *excessive absorption*; the fæces being consequently too solid, while at the same time the peristaltic action is diminished.

The first class of cases will be separately considered. The other two classes may be associated with *organic diseases*, but are very frequently the consequence of mere *functional disturbance*. This may arise from a great variety of causes, of which the chief are habitual neglect of the act of defæcation, either from carelessness, want of time or undue modesty; indulgence in astringent articles of diet; habitual use of opium; excessive smoking; sedentary habits, especially if combined with much mental work; enervating habits, particularly lying in bed to a late hour; anæmia, debility, and want of tone from any cause; hepatic derangements; most acute febrile diseases; various chronic affections, especially those connected with the nervous system; uterine and ovarian derangements; and the presence of lead in the system.

Undoubtedly some individuals are predisposed to constipation, particularly those who are of a slow, lethargic temperament. This disorder is more common in females than males, and is more liable to arise as age advances, though it is of very frequent occurrence in young women.

**SYMPTOMS.**—Constipation simply means that the stools are not passed with sufficient frequency, being at the same time generally deficient in quantity, as well as too dry and solid. In many instances it is a mere temporary derangement; but in others the bowels are habitually confined. Some individuals state that their bowels are regular, simply because they go to stool every day, but in reality they suffer from habitual constipation, as they only pass small lumps of hard fæces; hence the necessity for making close inquiry in any doubtful case. The degree of constipation varies much, but it is not uncommon to meet with patients, especially females, whose bowels are only moved once or twice a week; and sometimes the intervals are even longer than this, being in exceptional cases quite extraordinary. Hence fæces may accumulate to an enormous amount in the intestines, distending them greatly; and when discharged, they are firm, often extremely hard, dry, in the form of scybalous lumps or large masses, frequently pale, and unusually foetid. Hard excrement may cause irritation, setting up a kind of diarrhoea attended with the discharge of mucus or pus, and thus may mislead as to the actual conditions present, the fæces being in reality retained. The passage of indurated fæces may give rise to a great deal of pain about the anus, with straining, and sometimes discharge of blood; and may also cause piles. When retained, excrement is very liable to undergo decomposition, thus giving rise to much painful flatulence; the secretions are also still more interfered with, as well as the motor functions of the bowels, and dyspepsia, usually of an atonic kind, is set up. The mechanical effects of accumulated fæces are often very serious, and they may cause complete intestinal obstruction, or may lead to ulceration and perforation. Not uncommonly an accumulation can be detected by *physical examination* of the abdomen, and it may simulate various abdominal tumours. As a rule tumours due to accumulation of fæces correspond in position and shape to the cæcum, or to some part of the colon; they often have a doughy feel, yielding to pressure, by which they are sometimes much altered; and percussion over the corresponding part of the abdomen generally elicits a combination of dulness and tympanitic sound. In some cases, however, these accumulations produce extensive, irregular, solid enlargements, greatly resembling masses of cancer. Therefore the possibility of any doubtful abdominal tumour being due to fæces should always be borne in mind, and the effects observed of a thorough clearing-out of the bowels by means of aperients and enemata, or in other ways before a positive opinion is given.

Upon the general system the effects of habitual constipation are frequently very marked. It produces a state of nervous depression; and by interfering with digestion and nutrition, may cause much wasting and anæmia.

**TREATMENT.**—It is needless to enumerate here the ordinary remedies employed in the treatment of accidental and temporary constipation, as these are discussed in treatises on *THERAPEUTICS*, and are sufficiently alluded to in other parts of this work. A few remarks are, however, necessary regarding the management of habitual constipation. 1. It is most important to impress upon patients the necessity of attending to the *habit* of going to stool daily, at the same hour, and of having a proper evacuation, because, if this is neglected for a long period, it becomes extremely difficult to restore the bowels to their normal activity. 2. *Change in diet* may assist in removing constipation. Astringent articles of food must be avoided. Bran-bread, oatmeal cakes,



and porridge certainly prove efficacious in not a few cases; and figs or somewhat acid fruits are also useful in some instances. Any injurious habits which tend to confine the bowels must be avoided; and a proper amount of daily exercise should be taken. Cold bathing, with douching of the abdominal walls, is often beneficial; and in women in whom these walls are relaxed, the plan of wearing a broad bandage or elastic support round the body, firmly applied, is exceedingly serviceable. 3. It must not be forgotten that the inactivity of the bowels may be due to a general want of tone, and hence *tonics* are frequently useful, particularly those which improve the condition of the alimentary canal. The most beneficial are the non-astringent preparations of iron; mineral acids with bitter infusions or tinctures; strychnia, or extract or tincture of *nux vomica*. Should there be any lead in the system giving rise to constipation, iodide of potassium is the essential remedy. 4. Various *aperient* medicines have usually to be employed, but it is highly desirable to avoid falling into the habit of relying upon these agents, if possible, especially the stronger purgatives, and, therefore, as soon as the desired effect has been produced in any case, and the bowels have been properly emptied, purgatives should be stopped, and the patient impressed with the importance of trying to keep up a regular action, by attention to the matters already indicated. Among the most efficacious *aperients* in these cases are confection of senna or sulphur, taken early in the morning; castor-oil or olive-oil; compound rhubarb pill; sulphate of magnesia, gr. xx to 3i three times a day, which is often beneficially combined with sulphate of iron; sulphate of soda; Seidlitz powders; sulphate of potash, or phosphate of soda, particularly recommended for children; aloes, in the form of watery extract or decoction, or aloin, especially valuable if the colon is torpid; jalapine; and extract of belladonna in doses of gr.  $\frac{1}{4}$ th to  $\frac{1}{2}$ th once daily, gradually increased. The last mentioned drug has deservedly come into high repute, and has been particularly recommended by Trousseau; a combination of this remedy with aloes and extract of *nux vomica* is very serviceable in some cases, the *nux vomica* giving tone to the bowel. Powdered ipecacuanha in small doses is also a useful addition in some cases, by causing increased intestinal secretion. Cascara has of late come into repute in the treatment of habitual constipation, and in many cases it proves of service. Not uncommonly it becomes necessary to use the stronger purgatives from time to time, such as extract of colocynth, blue pill, calomel, jalap, or gamboge. If the bile appears to be deficient, podophyllin, euonymin, and other cholagogues are valuable; or some recommend inspissated ox-gall. Some of these remedies may be given in different combinations with advantage, made up into pills with extract of gentian or extract of hyoscyamus. It seems best to administer them just before or during a meal. Various *aperient mineral waters* are often serviceable, such as Friedrichshall, Hunyadi Janos, Æsculap, Rubinat, Vichy, or Seltzer waters.

The employment of *simple enemata* in cases of habitual constipation is not carried out to the extent which it deserves. Unquestionably a morning injection of water, soap and water, or a solution of salt, will often prove highly efficacious; if necessary a little castor-oil may be added. The use of a suppository of soap is a popular remedy in some parts, especially in the case of children. The application of cold to the abdomen, or supporting the walls by means of an elastic apparatus may sometimes aid in curing habitual constipation. It has been recommended to galvanize the abdominal muscles in obstinate cases.



Occasionally, as the result of long-continued accumulation, the rectum becomes greatly distended with solid and dry excrement, which has to be mechanically scooped out. Enemata may be used for the purpose of aiding in softening this hardened fæces, and breaking it down.

#### IV. DIARRHŒA.

**ÆTIOLOGY.**—Diarrhœa results either from increased peristaltic action of the intestines; from an unusually liquid state of their contents, especially when this depends on excessive secretion; or, most commonly, from a combination of these conditions. The *exciting causes* of these morbid phenomena may be thus arranged:—1. *Irritation of the intestines* by food, either taken in excess, of improper quality, undigested, or having undergone decomposition; impure water or other liquids; purgative medicines and irritant poisons generally; excessive or unhealthy secretions, especially bile; worms, trichinæ, and other parasites, possibly vegetable as well as animal; or retained fæces. 2. *Mechanical congestion of the intestinal vessels*, owing to some obstruction in the portal circulation. 3. *Organic affections of the intestines*, namely, enteritis, either acute or chronic; albuminoid disease; and ulceration. 4. Occasionally mere *nervous disturbance*, such as strong mental emotion; or reflex irritation in connection with dentition. 5. *Certain diseases* in which diarrhœa is a prominent symptom, especially cholera, typhoid fever, and dysentery. By many it is then regarded as *eliminary* in its character, serving to carry off some poisonous material; and the same theory is applied to its occurrence in renal disease, gout, pyæmia, and various fevers; or when it takes place as a *critical discharge* at the close of pyrexial affections. Colliquative diarrhœa not unfrequently sets in during the course of certain wasting chronic affections, especially towards their termination, aiding in bringing about a fatal result, especially in phthisis, cancer, splenic or supra-renal disease, and Hodgkin's disease. 6. The rapid suppression of discharges, or the absorption of dropsical fluid, when the diarrhœa is termed *vicarious*. 7. Causes of a more *general* character, namely, exposure to changes of temperature, or to excessive cold or heat; foul air, overcrowding, and other anti-hygienic conditions; excessive fatigue; emanations from decomposing animal matter; and malarial influence. The combined action of some of these causes, along with improper diet, gives rise to the *summer* and *autumn diarrhœa*, or so-called *English cholera*, so prevalent during those seasons. 8. Very rarely the *escape of some fluid accumulation* into the intestines, such as the contents of an abscess, peritoneal effusion, or the fluid portion of a hydatid tumour.

**CHARACTERS.**—In all cases of diarrhœa it is requisite to ascertain its duration; the number of stools passed in the twenty-four hours; and their relation to the introduction of food, if any: and also to inspect specimens of the excreta, if practicable, as frequently as may be desired. The principal varieties of loose stools are *faculent*; *lienteric*, when they contain cognizable fragments of food, in some cases scarcely at all changed; *bilious*; *serous* or *watery*, also called a *flux*; *mucous* or *gelatinous*; *bloody*; *fatty*; *purulent*; *chronic* or *white flux*. As a rule the materials are more or less mixed, and by an examination of the characters of the stools the cause of the diarrhœa may often be determined. Various other digestive disturbances are usually associated with

this symptom, indicated by griping or other pains in the abdomen, sickness, borborygmi, straining at stool, or an abnormal state of the tongue. The stools may irritate the anus considerably, especially when the diarrhœa is long-continued and of a watery kind. It must be remarked that patients sometimes state that they are suffering from looseness of the bowels, when on investigation it will be found that there is only some local discharge, especially in connection with *fistula in ano*. The association of mucous discharge with retained fæces has already been alluded to.

If diarrhœa is considerable or of long duration, it necessarily causes more or less debility and wasting, in some instances very rapidly and markedly reducing the patient.

**TREATMENT.**—The first matter relating to the treatment of diarrhœa is to determine whether it should be checked or not. In some instances this is not desirable, provided it is not excessive, the discharge by the bowels being preservative and beneficial, as, for instance, in connection with Bright's disease or portal congestion. Some even go so far as to promote diarrhœa in certain diseases, such as cholera and typhoid. As a rule it is necessary to check this symptom either entirely or partially. For this end the *diet* must be strictly regulated, and this may be the only thing needed, especially in the case of children. Milk with farinaceous articles, especially arrow-root and corn-flour; weak beef-tea, thickened with these materials; and milk puddings, constitute the best articles of diet. Milk with lime-water, if administered in small quantities, and at proper intervals, will often speedily put a stop to the diarrhœa of children. In some cases a little brandy-and-water, or a mixture of brandy with port wine, is beneficial. Not uncommonly an *aperient* is indicated at the outset, with the view of getting rid of irritant materials from the alimentary canal. Castor oil, calomel, a saline draught or Seidlitz powder, or a full dose of tincture of rhubarb act best in these cases, and they are often advantageously combined with a little opium. *Antacids*, such as carbonate of soda or magnesia, are beneficial when irritating secretions are present in the bowels.

Among the *direct remedies* used for combating diarrhœa opium holds the first place, given either alone or with other medicines, in the form of pill, tincture, confection, various powders, enema, or as syrup of poppies. An injection of  $\mathfrak{m}\text{xv}$ — $\text{xx}$  of laudanum with  $\mathfrak{z}\text{iss}$ — $\text{ij}$  of decoction of starch often acts most beneficially. The other principal medicines administered in *acute* cases are prepared chalk, aromatic confection, catechu, kino, logwood, krameria, alum, dilute mineral acids, especially sulphuric, tannic and gallic acids, carbonate or nitrate of bismuth, chloral, and chlorodyne; in *chronic* cases tincture of sesquichloride or solution of pernitate of iron, acetate of lead, sulphate of copper, or nitrate of silver. Ipecacuanha is invaluable in certain forms of diarrhœa. Among the most efficient combinations will be found chalk mixture with tincture of catechu and opium; compound chalk-powder, with or without opium; compound kino powder; decoction of logwood with lime-water, particularly valuable for children; dilute or aromatic sulphuric acid with laudanum; Dover's powder, alone or with carbonate of bismuth; and, in chronic cases, pills containing acetate of lead or sulphate of copper, combined with opium.

Creosote, carbolic acid, and other *antiseptics* have been employed in certain forms of diarrhœa, with the view of destroying vegetable organisms, upon which this symptom is supposed to depend.

*Local applications* to the abdomen are frequently very beneficial, in the form of poultices, fomentations, or dry heat. A flannel bandage worn round the abdomen is useful in some chronic cases. Occasionally a patient may by voluntary effort to some extent suppress diarrhœa, especially when this is due to emotional disturbance.

#### V. MELÆNA—INTESTINAL HÆMORRHAGE.

**ÆTIOLOGY.**—Most of the causes of melæna are similar to those which give rise to hæmatemesis, and it will be sufficient briefly to enumerate them thus:—1. *Traumatic injury*. 2. *Diseased conditions of the blood*. 3. *Vicarious*. 4. *Mechanical and chemical irritation or destruction of the bowel*, especially by violent purgatives, cantharides, turpentine, various irritant poisons, hardened fæces, and rough calculi. 5. *Organic diseases*, namely, enteritis, ulceration, especially in typhoid fever and dysentery, cancer, invagination, piles, prolapsus, fissures or fistulæ about the anus. 6. *Extreme mechanical congestion*, from portal obstruction or chronic heart or lung disease. 7. *A tumour eating its way through the wall of the intestine*; or an *aneurism bursting into its cavity*. 8. *Passage of blood from the stomach into the bowels*, following hæmorrhage into this organ.

**CHARACTERS.**—When blood appears in the stools, it is generally much altered in its characters, but this will depend upon its amount and source, and upon the rapidity with which it escapes. When in small quantity, coming from the upper part of the bowel, and being slowly discharged, it is usually more or less dark, being often quite black, and presenting a tarry or sooty aspect; occasionally it resembles coffee-grounds. If originating from the same source, being at the same time copious and speedily expelled, it may be but little altered, though it is usually of a very dark colour. When coming from the large intestines, especially near the anus, it is generally quite bright and unchanged. The quantity varies much, ranging from mere streaks in the fæces to an amount sufficient to cause speedy death. By attending to the quantity and appearances of the blood, its seat of origin may generally be determined, the diagnosis being further aided by the general history of the case; and by a consideration of the symptoms and physical signs referable to the abdomen, not forgetting to make an examination of the anus and its vicinity. Care must be taken not to mistake the dark colour due to bile or iron for that depending on the presence of blood in the stools.

**TREATMENT.**—The same remedies are useful in the treatment of melæna as have been recommended for hæmatemesis. Oil of turpentine is in much repute. Enemata of *iced water* are sometimes serviceable; as well as the application of ice-bags to the abdomen. *Astringent* enemata may also be indicated in some cases. If there is any morbid condition about the anus giving rise to hæmorrhage, such as piles or fistula, as well as in certain forms of internal disease, for example, cancer of the intestine, surgical interference may be required.

---



## CHAPTER XXXVIII.

## INFLAMMATION OF THE INTESTINES—INTESTINAL OR ENTERIC CATARRH—ENTERITIS—DUODENITIS—TYPHLITIS AND PERITYPHLITIS.

INFLAMMATION of the bowels presents considerable varieties as regards the coats which are involved, and the extent of the intestinal tract which is implicated; hence the clinical history of this disease is anything but uniform. The term *enteritis* has been used very vaguely, and several distinct conditions have been included under it. It will be convenient briefly to consider in the present chapter all forms of disease in which the intestines generally, or any portion of them, are inflamed, apart from special affections, such as dysentery or typhoid fever.

**ÆTIOLOGY.**—The causes of intestinal inflammation are very similar to those which induce gastritis. *Enteric catarrh* or *mucro-enteritis* is ordinarily due to some direct irritation of the mucous lining by food or other materials, or to a cold; it is also frequently associated with various exanthemata, and with dentition. Irritant poisons give rise to more severe inflammation. A very intense *local* form of enteritis follows obstruction of the bowels; this also sometimes results from ulceration, or from extension of peritonitis; and it is said to occur in rare instances idiopathically. The local variety named *typhlitis* or *inflammation of the cæcum* is generally due to the lodgment of hardened fæces or of foreign bodies in this part of the intestine, or in the appendix vermiformis, which often ultimately leads to ulceration and perforation. *Duodenitis*, followed by ulceration, is peculiarly liable to be set up after burns and scalds. *Chronic intestinal catarrh* remains occasionally after an acute attack, but usually results from the repeated action of local irritants; or the complaint is associated with ulceration, lardaceous disease, or other organic changes affecting the bowel.

**ANATOMICAL CHARACTERS.**—In the milder forms of intestinal catarrh the appearances resemble those observed in catarrh of other mucous surfaces, and need no special description. The secretions are abundant and often very irritating, being sometimes mixed with blood. Superficial erosions or slight ulcerations are not uncommonly observed. Occasionally a croupous or membranous deposit is formed over the surface more or less extensively, which indicates greater intensity of the inflammatory process. Should this be very violent, implicating the entire thickness of the gut, the colour is extremely deep red, sometimes purple or almost black, being accompanied with spots of extravasation; all the coats are thickened and softened, and generally infiltrated with serum, or occasionally with exudation or pus; while the intestinal contents are often mixed with blood. Gangrene occurs in some cases. The peritoneum may be involved by extension, exhibiting patches of lymph corresponding to the inflamed bowel.

In *simple catarrh* the entire mucous tract is frequently affected, the condition beginning above, and extending throughout the intestines; it may, however, be limited. The more severe forms are usually confined

to short portions of the bowel, which are generally much distended, particularly when the inflammation depends upon obstruction, the part beyond being contracted.

In *typhlitis* the cæcum or appendix vermiformis becomes much inflamed from local irritation, and this leads to ulceration and destruction of the coats, which is liable to end in rupture or perforation. The perforation may take place into the peritoneum, causing peritonitis; or into the surrounding cellular tissue, setting up inflammation in this structure—*perityphlitis*—which usually terminates in the formation of an abscess, and this, if not opened, may burst in a variety of directions. Possibly perityphlitis may be excited independently of any actual perforation. The cause of the irritation is usually some foreign body or hardened fæces. In the appendix some small foreign substance which has gained an entrance becomes a nucleus upon which fæces and secretions are deposited, forming concretions which come to resemble fruit-stones, for which they have been frequently mistaken. Of course it must be remembered that perforation may result from other forms of ulceration which are met with in the cæcum; from mere distension of this part of the intestine; or from its destruction by some extrinsic growth. The same course of events is now and then observed on the opposite side of the abdomen, in connection with the sigmoid flexure. A localized variety of inflammation has also been described in the colon—*colitis*—supposed to be distinct from dysentery, beginning in the sub-mucous tissue, but soon causing extensive destruction of the mucous membrane.

When intestinal catarrh becomes *chronic*, there are the usual changes in colour, the membrane being often dark or even black from pigment; thickening and induration of tissues; with degenerative changes in the gland-structures. Chronic catarrh may give rise to ulceration; or, on the other hand, it may result from this condition, or from some other organic change in the bowel.

**SYMPTOMS.**—1. Cases of *simple enteric catarrh* are generally characterized by uneasiness over the abdomen, with colicky and griping pains, especially about the umbilicus, where there may be a little tenderness, though pressure sometimes gives relief; formation of much gas in the intestines, causing gurgling and borborygmi; and diarrhœa, especially after taking any food or drink, the stools becoming in some cases very numerous, being at first fæculent, but soon assuming a watery, irritating character. These may be the only symptoms, but as the stomach is often implicated at the same time, this is indicated by a red, furred, and dryish tongue, impaired appetite, thirst, and a tendency to nausea or vomiting. In *duodenal catarrh* jaundice is frequently observed, owing to the closure of the common bile-duct by the swollen membrane; and if the duodenum is solely involved, there is corresponding localized pain and tenderness, with constipation instead of diarrhœa. Occipital headache is also said to be common in duodenitis. If diarrhœa has been severe for any length of time, the stools are apt to become somewhat dysenteric in character, containing mucus and blood, especially if the large intestines are mainly implicated, when there may also be much tenesmus and straining during defæcation.

The symptoms are more marked in proportion to the intensity of the inflammation, especially the pain and tenderness, and they are particularly severe in connection with *irritant poisoning*. Should there be any membranous deposit upon the mucous surface, shreds, larger

patches, or even intestinal casts of this material may be expelled in the stools.

*General* symptoms are in some cases entirely absent, except, perhaps, some feeling of exhaustion from excessive diarrhoea. In the more severe forms of enteritis, however, pyrexia is observed, with languor, general depression, and headache. In children there is frequently high fever, accompanied with much prostration, a greatly distended abdomen, and aphthous stomatitis. Sometimes convulsions or coma set in, and death may result from this cause, or from exhaustion. In cases of irritant poisoning the general symptoms are grave, there being often a tendency to collapse; and the same thing is occasionally observed in severe enteric catarrh from other causes, especially in persons constitutionally weak, or who are the subjects of some chronic lowering disease.

2. The *limited intense form* of inflammation which involves all the intestinal coats presents symptoms essentially different from those just described, and it is to this variety that many authors limit the term *enteritis*. Here the affected portion of the intestine, which is at first the seat of spasm, soon becomes paralyzed, so that the contents cannot pass along, but accumulate in the part above. The early symptoms include much localized pain, with tenderness, often referred to the umbilical region, and increased by movement; general colicky pains and tormina; obstinate constipation; constant nausea and vomiting; much thirst; a furred tongue; and pyrexia, preceded by rigors, the patient presenting a distressed and anxious expression. In a short time, if there is no relief, the abdomen swells on account of tympanites; while the painful sensations subside more or less, in some cases completely ceasing; the vomiting gradually becomes stercoraceous, at last the materials coming up without any effort; the tongue assumes adynamic characters; and signs of collapse set in, with a pinched countenance, and an extremely feeble and irregular pulse, the brain being either unaffected to the last, or death being preceded by low nervous symptoms. The urine becomes much diminished or suppressed. Hiccup is often a distressing symptom.

3. *Typhlitis* is generally indicated at the outset by pain and tenderness in the right iliac fossa, often severe; with, in some instances, distinct *physical signs* of an accumulation in the cæcum; and constipation, which may be followed by mucous or muco-purulent diarrhoea. *Sudden perforation* may take place into the peritoneum, even when there have been no previous symptoms of any moment. In other cases *perityphlitis* is set up, as evidenced by local redness; a firm swelling; oedema of the skin; increase of pain and tenderness; rigors and pyrexia; followed usually by signs of the formation of an abscess, which may open in various directions, either externally or internally, sometimes thus setting up peritonitis. The pus has often a faecal odour, and may be mixed with actual faeces or intestinal gas. If the case does not prove speedily fatal, a permanent fistulous opening may remain, death occurring gradually, preceded by hectic symptoms; or ultimately recovery may ensue, the abscess healing up. The same symptoms are observed in rare instances in the left iliac fossa, in connection with the *sigmoid flexure*.

4. *Chronic intestinal catarrh* is frequently attended with no other symptom than chronic diarrhoea, the stools being liquid, pale, fermented, and often very offensive or lenteric, varying much in number and quantity. In many cases uneasy griping sensations and gurgling are experienced from time to time; or there may be some degree of soreness over the



abdomen. Gastric symptoms are generally present, and the tongue often presents abnormal characters. Owing to interference with digestion and nutrition, more or less wasting is commonly observed, as well as slight pyrexia in some cases, especially towards evening.

DIAGNOSIS.—The chief affections for which the various forms of *acute intestinal inflammation* are liable to be mistaken are simple diarrhœa, or diseases attended with this symptom, especially typhoid fever and dysentery; intestinal colic; peritonitis; painful affections of the abdominal walls; or, in the case of typhlitis and its consequences, local inflammation or an abscess in the right iliac fossa due to other causes, and certain tumours.

There can be no doubt but that many ordinary cases of diarrhœa are the result of enteric catarrh, and it is often impossible to separate them. The characteristic symptoms of typhoid fever and dysentery are usually sufficiently distinctive. Simple colic is recognized by the characters of the pain; the absence of fever; and the presence of constipation. Peritonitis is readily separated from mere catarrhal inflammation by the intensity of the pain and tenderness, constipation, great constitutional disturbance, and other symptoms; but as regards severe localized *enteritis*, it is by no means easy to distinguish between the two diseases. Indeed, in most instances the peritoneum is involved along with the other intestinal coats, and this is more evident if the pain and tenderness are marked, superficial, and extensive. Colicky pains are suggestive of inflammation of the more internal portion of the wall of the bowel. It is important to bear in mind *duodenal catarrh* as a not uncommon cause of jaundice.

In cases of *chronic intestinal catarrh* the main point to be determined is whether this is of a simple nature; or if it is associated with ulceration or amyloid degeneration. The special characters of intestinal ulceration will be presently indicated. Lardaceous disease of the bowels is almost always preceded by distinct clinical evidences of other organs being affected; as well as by one of the known causes of, and the constitutional conditions accompanying this morbid state.

PROGNOSIS.—Ordinary *enteric catarrh* usually ends favourably, but it may become chronic. If intense, however, or if it occurs in children, in very weak subjects, or as a complication of acute or chronic diseases, it may become highly dangerous and end fatally. The severe form of *enteritis* is extremely grave. *Typhlitis* is also necessarily attended with many dangers. *Chronic intestinal catarrh*, especially if long-established, is often very difficult to cure, and may itself ultimately prove fatal; while it adds to the gravity of other chronic diseases, by interfering with the nutrition of the patient.

TREATMENT.—The remarks made with respect to *diet* in the case of stomach-disorders, apply with almost equal force to those affecting the intestines. In *acute catarrh* of the bowels, if there is anything causing irritation, it is desirable to get rid of this by means of a dose of tincture of rhubarb, castor oil, or some other simple *aperient*, or by an enema. As regards *internal remedies*, the most serviceable combination in my experience consists of bismuth, with alkalies, and small doses of tincture of opium. An enema containing laudanum is also very useful after all irritant matters have been evacuated; and the various other remedies recommended for diarrhœa may be had recourse to if required. In *duodenal catarrh* it is necessary to give small doses of some *saline aperient*, such as sulphate with carbonate of magnesia, which may be

preceded by a dose of calomel. *External applications* over the abdomen, especially heat and moisture, are often beneficial. Ordinarily there is certainly no necessity for removal of blood, but in the early stage of the more serious form of intestinal inflammation it may be permissible to apply a few leeches to the abdomen, provided the patient is in a fit state. When inflammation accompanies obstruction of the bowel, the main points in treatment are to avoid giving purgatives; to administer opium freely, either by the mouth or enema, or to employ subcutaneous injection of morphia; to support the patient, especially by enemata; and to treat the prominent symptoms, particularly pain, nausea, vomiting, and tympanites.

*Typhlitis* and its consequences need constant fomentation and poulticing; perhaps the application of a few leeches in some cases; and the administration of opium internally. If an abscess forms, it should be encouraged towards the surface, and the pus evacuated when the proper time arrives. If an accumulation can be felt in the cæcum, it may sometimes be squeezed out by gentle manipulation, but much care is necessary in practising this measure.

*Chronic enteritis* will probably require some of the more powerful *astringents* mentioned under diarrhœa. Powders containing carbonate of bismuth, gr. v-xx, with Dover's powder, gr. iij-vi, act very beneficially in some cases. Tincture of steel is also a valuable drug in this complaint, when given in full doses— $\mathfrak{m}$  xx-xxx. In obstinate cases *counter-irritation* over some part of the abdomen, especially over the right iliac fossa, by means of blisters, tincture of iodine, or croton-oil liniment, may prove of service.

## CHAPTER XXXIX.

### DYSENTERY—BLOODY FLUX.

**ÆTIOLOGY.**—Dysentery occurs as an *acute* and *chronic* disease, and may assume a sporadic or epidemic character. Different views are held as to its immediate *exciting cause*, namely:—1. That it is due to a malarial poison, originating in vegetable decomposition, and rising from the soil. 2. That though primarily originating in this manner, it may be afterwards propagated from one individual to another by means of a contagious specific poison; conveyed, as some suppose, only by the stools, especially through their being mixed with drinking-water; or, as others believe, by all the excretions and exhalations. Special micrococci have been described by Prior. 3. That it is independent of any specific poison, and merely results from certain general causes which tend to produce intestinal congestion and inflammation, such as exposure to cold, especially to night chills and dews; errors in diet, particularly want or improper quality of food; excessive use of salt meat; drinking impure water or irritating liquors; or indulgence in excess of, or in sour fruit. Those who regard the malady as *specific*, consider the causes just mentioned as merely *predisposing*, or as aiding in *propagating* the poison, but it is highly probable that they may at all events *excite* the sporadic form. Amongst other *predisposing* and *propagating causes*

are recognized a hot and moist climate, especially during the seasons when the nights become chilly, most cases occurring in the autumn, and particularly after much exposure to night air; over-crowding and filth; bad ventilation, especially if accompanied with exposure to emanations from any kind of decomposing organic matter; and physical exhaustion. Dysentery may complicate or follow certain diseases, particularly ague or remittent fever, scurvy, relapsing fever, cholera, or syphilis; or may supervene in the state of system resulting from the action of prolonged heat. Ague and chronic dysentery were not uncommonly met with together in the case of sailors admitted under my care at the Liverpool Northern Hospital; and occasionally the latter complaint accompanied scurvy. *Chronic dysentery* always originates in the acute form.

ANATOMICAL CHARACTERS AND PATHOLOGY.—In general terms dysentery may be said to be characterized anatomically by inflammation of the large intestines, ending in ulceration or gangrene; with exudation on the mucous surface. According to the view entertained as to the origin of the disease, the inflammation is considered by different authorities as *simple* or *specific*. Niemeyer regarded it as of a *diphtheritic* nature. So diverse have been the descriptions given of the morbid appearances, that it has been found necessary to start theories as to difference of type, &c., in order to explain the want of agreement, and it seems highly probable that the constitutional state of the patient will materially influence the pathological characters of the disease.

Usually only a portion of the *large bowel* is involved, especially the *rectum* and neighbouring part of the *colon*; sometimes the entire tract is affected, but the disease is then generally more advanced towards the lower end of the intestine. Occasionally it extends into the small intestines, even for a considerable distance, but this is usually observed only in scorbutic cases, or when patients are much debilitated.

In the early stages of dysentery the chief appearances are considerable enlargement of the solitary glands (which has been regarded by most authorities as the *primary lesion*), and of the tubular glands; increased vascularization, varying much in extent and intensity, but being especially marked around the glands; with some swelling and softening of the mucous membrane. The solitary glands form little rounded projections, and in many of them a minute spot can be detected, corresponding to the orifice. They are filled with a whitish exudation, containing abundant young cells. Some observers consider these prominences as at all events partly due to exudation outside the glands, and they affirm that these structures are not specially involved in dysentery. An exudation also forms in the intertubular tissues, and on the surface of the mucous membrane, which is of a diphtheritic nature. It covers the membrane more or less extensively and thickly, sometimes being uniform, but usually granular, often presenting an appearance like bran or sawdust, and being most abundant on the tops of the mucous folds. At first the colour is greyish or yellowish-grey, but this is soon liable to many alterations from various causes. The material is opaque, of some consistence, and can be detached, leaving a more or less red surface underneath, or the remains of extravasated blood. It appears to consist of a fibrinous substance, with abundant granules, nuclei, germs, epithelium cells, and young nucleated cells, some of which are elongated and fusiform, resulting mainly from proliferation. Occasionally the exudation undergoes a process of partial organization.



Ulceration appears to begin chiefly in connection with the enlarged glands, by a process of limited sloughing at the summit, the ulcer afterwards spreading. Sometimes, however, several glands, with the intervening tissues, are destroyed simultaneously; or the ulceration appears to be due occasionally to a process of disintegration of the exudation, involving at the same time the superficial part of the membrane. Very rarely it originates in submucous accumulation of pus or other morbid products. At the outset most of, but not all, the ulcers are small, circular, with rounded edges; by extension they become larger and irregular, often having a transverse direction, the margins being flattened, and the depth and appearance of the surface varying greatly, so that in time the ulcers are altogether wanting in uniformity. Not unfrequently the floor becomes covered with exudation. Now and then the coats are rapidly destroyed, so as to lead to perforation. Should the disease terminate favourably, and cicatrization ensue, this generally takes place with little or no puckering, the edges becoming rounded and adhering to the base of the ulcer, a layer of lymph then extending over the surface, and becoming organized. Now and then healing is attended with much thickening, irregularity, induration, and contraction, leading to serious consequences. There is never any fresh formation of glands in the cicatrix.

The rapidity with which the changes just described take place in the course of a case of dysentery differs much. In very severe cases the entire mucous lining of the large intestine is speedily converted into a slough. The affected part is generally dilated, and contains very offensive materials, similar to those passed in the stools, including fragments of exudation, and often blood.

When the small intestines are implicated, the appearances which they present are redness; exudation on the surface, sometimes extensive; and enlargement, or, rarely, ulceration of Peyer's and the solitary glands. The stomach may be more or less inflamed. The other morbid conditions observed in different cases associated with dysentery are enlargement, redness, and softening of the mesocolic glands, and, in some forms, of the mesenteric; serous inflammations, especially peritonitis, either corresponding to the affected part of the bowel, or due to perforation; hepatic derangements, particularly inflammation ending in abscess; enlargement of the pancreas and spleen, the latter being in rare instances the seat of abscess; renal disease, with destruction of the epithelium; extensive bronchitis or lobular pneumonia; and pyæmic abscesses. The relation of *hepatic abscess* to *dysentery* has been much disputed. Some regard the two affections as merely two effects of the same cause, being independent of each other; others think that the abscess is secondary to the dysenteric ulceration, being produced by an extension of phlebitis to the liver, or, more probably, by the convection of emboli and other deleterious substances into the liver. This complication seems to be much more common in hot climates.

When dysentery becomes *chronic*, the appearances presented are very diverse. Usually firm exudation collects between the coats, matting them together, and causing much thickening and induration, so that the intestine feels very firm and solid, and pieces will sometimes stand on end. The colour of the mucous surface alters, becoming dirty brownish-grey, or in parts black, on account of pigment derived from altered blood. The exudation undergoes a certain degree of organization, and often forms thick, warty, adherent masses. The surface

presents in some cases a bark-like aspect. Frequently, but by no means always, ulcers are observed, in every conceivable variety of stage and character, as well as cicatrices of former ulcers; some of these result from changes set up in the exudation, which extends into the tissue beneath. In some cases cicatricial bands and contractions alter greatly the calibre and form of the bowel, and sinuses may pervade its walls. In other instances there is extreme atrophy of the intestinal coats, including the glandular structures. An appearance of pseudo-ulceration may result from separation or cracking of the firm exudation, thus exposing the mucous surface, which is extremely red and irritable-looking.

**SYMPTOMS.**—**Acute dysentery** is presented in all grades of intensity, from a mild sporadic form to an epidemic of the most virulent type, but its symptoms are generally highly characteristic. Many cases, especially in temperate climates, commence with simple diarrhoea, slight colicky pains, thirst, loss of appetite, and some degree of constitutional disturbance, the special symptoms setting in after a short interval; sometimes, however, these are observed from the outset. A chill or rigor commonly ushers in the disease in severe cases.

The prominent *local* symptoms are griping pains in the abdomen, technically named *tormina*, irregular in site, but chiefly felt along the colon; often a sense of heat or burning along the colon and rectum, or, in grave cases, over the whole abdomen; tenderness, especially in the left iliac fossa; more or less tympanites; tenesmus, indicated by a sensation of fulness, weight, bearing-down, or of the presence of a foreign body about the lower end of the rectum, with frequent or constant desire to defæcate, the act being accompanied with much straining; and the passage of peculiar stools. The morbid sensations differ greatly in their severity and persistency; in most cases they are paroxysmal, increasing until a stool is passed, by which act they are temporarily relieved; sometimes, however, they become constant and most agonizing. The tenesmus is more marked when the lower portion of the rectum is implicated. At first the stools are often semi-fæculent, or consist of hard scybalæ, but they soon assume the dysenteric characters, becoming scanty, slimy or gelatinous from the presence of mucus, and bloody; also giving out a most offensive and characteristic odour. Usually the different matters are more or less mixed, the more so the higher the disease is situated, when the stools contain abundant depraved biliary secretions. If the rectum is chiefly affected, the blood is less intimately incorporated, while the stools are more muciform (Maclean). Not uncommonly scybalæ are passed from time to time, covered with mucus and blood. Mild cases do not go beyond this, but in grave forms of the disease, occurring in tropical countries, the stools change in their characters, becoming muddy-looking; brownish, brownish-red, or even black; often watery and copious; and containing shreds or masses of membrane, which look like “washed raw meat.” Sometimes a large quantity of pure blood is passed; or sloughs of the mucous membrane are expelled. At this time the odour becomes intolerable, and it has been compared to carrion, or to the “smell of a macerating tub in the dissecting-room.” Dr. Goodeve has recommended that the stools should be washed with water, so as to leave nothing behind but the sediment, which contains the products of the intestinal disease, and these can then be examined, the condition of the bowel being thus more accurately determined. Chemically the stools yield a large proportion of albumin,



even when there is little or no visible blood. They are alkaline in reaction, and contain much carbonate of ammonia. Microscopically they present abundant epithelium cells, blood, exudation and pus-cells, and remnants of the membrane. Peculiar cells and other bodies have also been described.

There are other indications of digestive disturbance in dysentery, in the way of anorexia; thirst, which may be intense; furred tongue; and sometimes nausea and vomiting. There may be irritability of the bladder, with strangury; or, on the other hand, paralysis with retention when the rectum is much affected. The urine is generally high-coloured, scanty, and prone to rapid decomposition; in low forms it becomes very offensive; occasionally it is suppressed. In females irritation of the vagina is complained of in some cases of dysentery.

The *constitutional* symptoms in the less severe cases are only those indicative of slight pyrexia. In the graver forms these are more marked, there being at the same time much nervous depression and irritability, with an anxious, distressed expression of countenance. When a case tends towards a fatal issue, or in the most severe types of the disease almost from the first, the symptoms assume an adynamic or typhoid character, with great prostration, the tongue becoming dry, red, brown, or blackish, with sordes on the teeth; the pulse being very rapid, feeble, or irregular; while the tympanites increases; the painful sensations cease; there is persistent hiccup; and low nervous symptoms set in, ending in coma. In the *malignant* type of dysentery speedy collapse occurs, resembling that of cholera; accompanied with hæmorrhage from the mouth and nose.

VARIETIES.—Several varieties of acute dysentery have been described, according to the severity and nature of the symptoms present, and the conditions with which the disease is associated. The chief are named:—

1. **Mild.** 2. **Sthenic.** 3. **Asthenic** or **Typhoid.** 4. **Bilious.**
5. **Malarious**, characterized by the periodicity of the febrile paroxysms, much gastric irritability, the serous character of the stools from the first, which contain but little blood, the greater frequency of hepatic complications, and the efficiency of quinine in treatment (Maclean).
6. **Malignant.** 7. **Scorbutic.**

DURATION AND TERMINATIONS.—Dysentery lasts a very variable time, and it may terminate in death or recovery; or may become chronic. Death may happen within two days, or not for two or three weeks or more. This event results either from collapse; the typhoid state; gradual exhaustion in prolonged cases; hæmorrhage; or occasionally from perforation. A favourable turn is indicated by the stools becoming fæculent, and losing their dysenteric characters; the cessation of the painful sensations; diminution of fever; and improvement in strength, in the state of the pulse, and in the expression of the face.

**Chronic Dysentery** is a most troublesome complaint, but the precise symptoms do not entirely depend upon the state of the bowels, being often modified by some constitutional diathesis, or by a diseased condition of some other organ. The tenesmus and other morbid sensations are less marked than in the acute form, or they may be absent. In some cases control over the sphincter ani becomes completely lost. The stools differ considerably in their characters, even in the same case from time to time. They may be formed, but covered with mucus or blood; usually, however, they are more or less liquid, presenting a mucous, serous, or bloody appearance, with an admixture of fæces;



sometimes they are either reddish-brown, pale and frothy, mucopurulent, or actually purulent. The peculiar odour is retained more or less, and may be very intense. Appetite varies much, but is usually impaired; the tongue is often red, glazed, or fissured. The general system necessarily suffers considerably in marked cases, as evidenced by emaciation; anæmia, or a sallow and cachectic aspect; shrunken features, with a distressed, weary, or aged expression; a sense of weakness and exhaustion; pyrexia, tending towards the hectic type, with night-sweats; and loss of hair. Death often results from gradual asthenia.

**DIAGNOSIS.**—The symptoms of dysentery just described are quite characteristic, and an examination of the stools, combined with the sensations of the patient, and the general symptoms, ought to leave no doubt as to the nature of the disease. All authorities lay much stress upon the peculiar odour of the evacuations. The fact of the disease being epidemic may also be of service in diagnosis. Dysenteric symptoms may set in in cases of long-continued intestinal catarrh, which might then be mistaken for true dysentery. I have also known a case of cancer of the rectum simulate chronic dysentery.

**PROGNOSIS.**—The prognosis of *acute dysentery* will depend upon whether the disease is sporadic or epidemic; the severity of the attack; the characters of the stools; the general condition of the patient; the progress of the case; and the presence or absence of serious complications, especially hepatic abscess. Epidemic dysentery, particularly when of a low type, is extremely fatal. Signs of collapse or adynamia are of course very unfavourable, and among specially bad signs are mentioned gangrenous stools; severe hæmorrhage; subsidence of the pain, while the other symptoms are becoming worse; and suppression of urine. An early return of the evacuations to their normal state is highly favourable. *Chronic dysentery* may often be improved by appropriate management, as I found from a tolerably extensive experience of this disease at the Liverpool Northern Hospital. Prolonged cases, however, are not much amenable to any treatment.

**TREATMENT.**—Early attention is of extreme importance in *acute dysentery*, and the patient should immediately take to bed. In the sporadic form resulting from a chill, some authorities advocate the use of a warm, vapour, or hot-air bath at the outset. In some cases also a small dose of castor oil with laudanum is beneficial at first. *The remedy*, however, in this disease, and one which seems to have almost a specific action, is ipecacuanha in full doses. Several gentlemen who have had much experience in the treatment of dysentery in India, have personally informed me of the marvellous effects of this drug. Dr. Maclean recommends the following plan of administration:—To give gr. 25 to 30 of the powder in a small quantity of fluid, with a little syrup of orange-peel, after which the patient must keep perfectly quiet, and take no fluid for at least three hours, if thirsty being allowed to suck a little ice occasionally. In from eight to ten hours a smaller dose may be given, this depending on the effect of the first, and the urgency of the symptoms, by which also the subsequent repetition of the drug must be guided, and it may be required for some days. It is well to administer 10 or 12 grains at bed-time for a night or two after the stools appear healthy. Some authorities recommend the ipecacuanha to be administered more frequently in smaller quantities; and others employ  $5\frac{1}{2}$  to  $5\frac{3}{4}$  every four or five hours, but these large doses seem unnecessary, and

are liable to produce much depression. It has also been advocated to introduce the drug by enema, but this is likewise objectionable. Many consider it desirable to make the stomach tolerant of the medicine beforehand, by giving a full dose of laudanum or Battley's solution, or a few drops of chloroform; or by applying anodyne poultices over the epigastrium. Perhaps a small subcutaneous injection of morphia might answer this purpose. Dr. Maclean states, however, that frequently no sedative is required, and that if vomiting is unmanageable after ipecacuanha, hepatic complication or overcharging of the system with malaria should be suspected.

*Local applications* over the abdomen are very useful in dysentery, especially warm poultices; fomentations sprinkled with turpentine, laudanum, or chloroform; and sinapisms. *Symptomatic treatment* is often required. The above applications will usually afford relief to the painful sensations, but if the tenesmus is very severe, warm emollient enemata, or a suppository of opium may be tried. Of course *diet* requires the utmost attention. Beef-tea, soups, arrowroot, sago, raw white of egg, jellies, and such articles should be given in small quantities, between the periods of administration of the ipecacuanha. *Stimulants* are to be avoided generally, but in the typhoid condition they are certainly required. As the patient improves, so must the food be cautiously altered. *Hygienic measures* also demand every care, especially as regards the immediate *disinfection* and *destruction* of the evacuations.

The evidence in favour of the treatment thus far considered seems quite conclusive; but in a treatise of this kind it is necessary to mention the other chief methods advocated for dysentery. These are:—  
 1. By *astringents*, especially opium. These are useful if diarrhœa holds on after the stools have lost their dysenteric characters. 2. By *purgatives*, such as castor-oil, sulphate of magnesia, or cream of tartar. 3. By *venesection* and calomel. Calomel has been given in large quantities; or in doses of gr. i to gr. ij with opium every three or four hours. This treatment had better be avoided, and the only removal of blood which seems justifiable is by the application of a few leeches in the left iliac fossa, should the pain be very intense, and the state of the patient be favourable. 4. By a combination of blue pill, opium, and ipecacuanha. 5. By *antiseptics*. 6. By large doses of tincture of steel. It is important to note that there are two forms of dysentery which require a modification of treatment, namely, the *malarious*; and the *scorbutic*. The former calls for full doses of quinine, alternating with the ipecacuanha; the latter demands fresh fruits, and Maclean and others recommend bael very highly in these cases.

In the management of **chronic dysentery** the most essential matters are to regulate the *diet*; and to attend to *sanitary measures*, with the view of improving the health. The late Dr. Harry Leach found from his experience at the Dreadnought Hospital, that rest for the bowels and body, with a bland nutritious diet, are mainly to be relied upon for a cure, and he considers that drugs are of little or no use. Certainly I think that I have seen much benefit follow the administration of Dover's powder, gr. iv-v three or four times daily; and still more from full doses of tincture of steel during the day, with a little Dover's powder night and morning. Dr. Reginald Thompson found ipecacuanha (gr. iij-v every three hours) most serviceable. The stronger *astringents*, such as gallic acid, acetate of lead, sulphate of copper, or nitrate of

silver are often employed, but they have never been of much permanent service in my experience. Small doses of bichloride of mercury have also been recommended. A dose of castor oil with a little opium may be taken from time to time. Enemata containing opium are sometimes beneficial, especially for removing unpleasant sensations. Other measures recommended for this purpose are the use of a water-compress over the anus, or gentle douching of this part; wearing a bandage or a water-belt over the abdomen; friction over the abdomen with anodyne or irritant liniments; or the application of a blister over the left iliac fossa. Among the *hygienic* matters needing special attention are change of air, particularly speedy removal from a malarial district, or from a tropical country to Europe; the wearing of warm clothing; and the use of cold baths, followed by friction, if they are well borne. Some practitioners employ baths containing dilute nitro-hydrochloric acid. If there is any malarial, scorbutic, or other morbid condition of the system, the treatment must be modified accordingly. Any acute or sub-acute exacerbation of symptoms calls for complete rest; and the immediate administration of ipecacuanha.

---

## CHAPTER XL.

### INTESTINAL NEW FORMATIONS AND ULCERATIONS.

It appears to me desirable to give a summary of the morbid conditions which come under the above headings, with some general remarks on their clinical features; and to consider briefly such of the more important of these conditions as have not yet been noticed. It is expedient to treat of *new formations* and *ulcerations* together, because the former often originate the latter.

#### I. GENERAL SUMMARY.

**A. NEW FORMATIONS.**—These include:—1. *Cancer*. 2. *Tubercle* and other materials formed in the so-called *tubercular disease*. 3. *Typhoid deposit*. 4. *Albuminoid material*. 5. Occasionally *fibroid infiltration*. 6. Rarely *villous growths*; *polypi*; *adipose*, *cystic*, *erectile*, or *glandular tumours*; and *calcareous deposits*. The *local* symptoms due to either of these formations, should any be present, are either indicative of obstruction of the bowel; or of irritation and catarrh of its mucous membrane. Local pain or tenderness may or may not be complained of. Frequently there are constitutional symptoms, or symptoms associated with other organs, which aid in determining the nature of the disease. In some cases *physical examination* may detect a growth.

**B. ULCERATIONS.**—Intestinal ulcers may be thus arranged:—

**I. Non-Specific.**—These comprise:—1. Ulcers due to *direct injury* of the mucous surface by foreign bodies, calculi, hardened fæces, and chemical destructive agents, including probably acrid secretions. 2. Those originating in *inflammation*. Simple catarrh, especially if it be of long duration, may end in ulceration, either catarrhal or follicular.



The separation of croupous or diphtheritic deposit may also originate an ulcer. Rarely this lesion results from submucous suppuration or gangrene. 3. *Perforating ulcer*. An ulcer similar to the gastric variety is now and then observed in the duodenum. The form associated with burns and scalds also needs to be mentioned. 4. Ulceration due to some *morbid condition outside the bowel* making its way into the interior, which is very rare.

II. **Specific.** These comprehend:—1. *Typhoid*. 2. *Tubercular*. 3. *Dysenteric*. 4. *Cancerous*. 5. *Syphilitic* probably. 6. Ulcers following *albuminoid disease*.

**SYMPTOMS.**—The *local* symptoms suggestive of ulceration of the bowels are frequent colicky pains; localized pain or tenderness, especially if the disease is extensive, or if the large intestine is involved; and persistent or troublesome diarrhoea, the stools often presenting very unhealthy characters, sometimes resembling pea-soup or gruel, and being unusually foetid, or containing blood, mucus, or pus. Cases come under observation, however, in which constipation is a conspicuous symptom. If the disease is limited to the small intestines, especially their upper part, diarrhoea is of a simple kind; and it is usually only in such a case that constipation is observed. If the large bowel is much implicated, especially the rectum, the symptoms tend to assume a dysenteric character. The diarrhoea is chiefly due to enteric catarrh set up by the ulceration. The circumstances under which ulceration occurs will generally aid materially in indicating this event, as in typhoid fever, dysentery, or phthisis. When *chronic*, it is in some instances very difficult to make out positively that there is ulceration, and to distinguish this lesion from mere chronic catarrh; but it may be strongly suspected should there be diarrhoea, either constant or easily excited, or not amenable to treatment, especially if the stools are of a very unhealthy character. The constitutional condition often affords aid in diagnosis. Intestinal ulceration itself tends to excite more or less pyrexia, which in chronic cases is frequently of a hectic type; and it also leads to impaired nutrition, consequently inducing emaciation, debility, and anæmia. It may cause peritonitis, perforation, or serious hæmorrhage; or stricture may result from cicatrization.

**TREATMENT.**—It need scarcely be remarked that attention to *diet* is all-important in treating ulceration of the bowels. At the same time it must be so ordered as to promote the nutrition of the patient; and to be adapted for any morbid diathesis present. Rest of the body is highly beneficial; and of course the affected part should be kept as quiet as possible. This object is best gained by administering opium in some form, if it is admissible; if not, other *sedatives* must be given, especially belladonna. Diarrhoea must be controlled by the various *astringents*, along with opium. The chief remedies which are believed directly to promote the healing of intestinal ulcers are nitrate of silver, sulphate of copper, acetate of lead, oxide of zinc, and bismuth salts. Much benefit often follows the use of carbonate of bismuth with Dover's powder in the ulceration which occurs during phthisis; as well as in other forms. It is not desirable to encourage long-continued constipation, but should this symptom be present, much care is necessary in the employment of aperients, which must be of the mildest kind; simple enemata are very useful under these circumstances. *Tonics* are often indicated, especially preparations of iron. It is well for the patient to wear a warm bandage round the abdomen, properly applied. *Local ap-*

*plications* may be required from time to time. Some recommend an occasional blister over the right iliac fossa, or other forms of counter-irritation.

## II. CANCER OF THE INTESTINES.

ANATOMICAL CHARACTERS.—*Primary cancer* of the intestines is very rare, and when the bowel is involved, which is not a common event, it is generally by extension of the disease, especially from the peritoneum and sub-peritoneal tissue, though even then the muscular and mucous coats often escape. The large intestines, particularly the rectum and sigmoid flexure, are far more frequently attacked than the small, of which the duodenum is the part usually implicated. All varieties of cancer are met with, even *melanosis*, but *scirrhus* is the ordinary form. *Epithelioma* has in rare instances invaded the rectum by extension from the uterus and vagina. Sometimes *encephaloid* grows on a basis of *scirrhus*, when the latter reaches the interior of the bowel.

The usual variations are presented as to the characters, arrangement, and extent of the cancer. It may be limited to one part, occasionally forming a rounded or lobulated tumour; widely spread; or disposed in scattered nodules, which are often secondary to some more localized and extensive deposit. In the progress of the disease the coats become matted together, and ultimately, if the mucous membrane is implicated, ulceration or sloughing ensues, the ulcer being either smooth and excavated, with thickened, indurated, and tolerably regular edges; or presenting an irregular aspect, with fungous growths over the floor and margins, which are often very vascular, and liable to bleed freely. Perforation may take place, not uncommonly a communication being thus established with some hollow organ. Frequently the affected part of the intestine is much constricted; while the part above is dilated, and its muscular coat hypertrophied, the portion beyond being contracted.

SYMPTOMS.—The following are the clinical phenomena to be looked for as indicative of cancer of the bowels:—1. Localized pain in some part of the abdomen, either constant or paroxysmal in character, dull and aching or lancinating, and accompanied with local tenderness. 2. Habitual constipation, with abnormal shape and size of the stools, ultimately culminating in complete obstruction. 3. The *physical signs* of a tumour situated deep in the abdomen; hard and irregular; tender on pressure; at first movable, but afterwards becoming fixed. 4. Marked and rapid wasting and loss of strength, often accompanied with signs of the cancerous cachexia, or of cancer in other parts. In some instances there is diarrhoea, especially after ulceration sets in, when the stools become extremely offensive, and occasionally symptoms of obstruction disappear, owing to a mass of cancer sloughing or ulcerating away. When the rectum is affected, the pain is referred to the sacrum, shooting thence towards the thighs and back, and being often extremely severe. There may likewise be intolerable irritation and itching within the anus. Symptoms of a dysenteric character are also generally complained of. Examination *per rectum* will usually reveal the disease. Cancer is liable to give rise to profuse hæmorrhage; or it may lead to intestinal perforation, or to extensive destruction of neighbouring organs. Death occurs gradually as a rule, but may be hastened by complete intestinal obstruction; perforation; peritonitis; or hæmorrhage.

TREATMENT.—In the large majority of cases of intestinal cancer all that can be done is to treat symptoms. In some instances life may be prolonged, and symptoms greatly relieved, by making an artificial opening into the bowel above the seat of the disease, should this be low down.

### III. TUBERCLE OF THE INTESTINES—TUBERCULAR ULCERATION—SCROFULOUS DISEASE OF THE INTESTINES.

ÆTIOLOGY AND PATHOLOGY.—There are the same differences of opinion as to the pathology of the so-called *tubercular* disease and ulceration of the intestines, as in the case of other affections of this class. Some pathologists explain all the phenomena by the formation of tubercle, and its subsequent destruction, along with the involved tissues. Others consider that true tubercle is but rarely formed, and that the morbid process usually consists in the proliferation of cells in the glands, which become caseous and break down, ultimately destroying the over-lying membrane, and forming ulcers, which spread by further cell-formation, and disintegration of the surrounding mucous and submucous tissues. Niemeyer acknowledged that *secondary tubercle* is met with sometimes in the immediate vicinity of ulcers, especially in the peritoneum corresponding to these lesions, but he maintained that as a primary condition intestinal tubercle is extremely rare.

It certainly is most difficult to distinguish definitely tubercle in the intestines. Niemeyer stated that it would be best recognized by its being deposited in separate nodules or groups, in parts where Peyer's patches do not exist.

As a *local* affection, implicating at the same time the mesenteric glands, this complaint is by far most frequent in scrofulous children; in adults it rarely occurs except as a complication of pulmonary phthisis.

ANATOMICAL CHARACTERS.—In the great majority of cases the solitary and Peyer's glands are chiefly implicated in tubercular disease, and hence the morbid appearances are observed in the lower portion of the small intestines, or are most advanced in this part, while they gradually cease towards the jejunum. Occasionally they extend into this portion of the bowel, or very rarely even to the duodenum. Not uncommonly the cæcum, appendix vermiformis, and colon are involved, to which parts the disease is sometimes chiefly or even entirely limited. The area affected varies greatly, and generally the morbid changes are visible in different stages in different parts. At first little firm, grey, projecting nodules are seen, which become yellow, and soften and break down, producing small circular ulcers. Apart from their situation, it appears impossible to distinguish the granulations of tubercle from those due to enlarged glands. The ulcers soon become larger, however, either by infiltration and destruction around, this process invading tissues far beyond the glands; or by coalescence. In course of time they come to present special characters, in which condition they are generally seen at *post-mortem* examinations. These characters include more or less irregularity in shape; a transverse direction as regards the bowel, the ulcer spreading mainly in the course of the vessels, and sometimes completely surrounding the gut with a band of ulceration  $\frac{1}{2}$  to 1 inch or more in width; thickening, irregularity, and induration of the margins and



floor, the latter presenting nodules; and but little proneness to heal. Imperfect or partial cicatrization is, however, often observed, with the formation of a dense tissue, sometimes pigmented, the edges of the ulcer being drawn together, contraction and irregularity of the gut being thus produced, or in rare instances even complete stricture.

During the process of ulceration *local peritonitis* is set up, giving rise to thickening and adhesion, and thus all the coats of the bowel are frequently destroyed without the occurrence of any symptoms of perforation, or sometimes a communication is formed between two portions of the intestines. *Secondary tubercles* are often observed in the affected portion of the peritoneum, which may spread along the lymphatics to the mesentery. The floor of an ulcer not uncommonly presents evidences of hæmorrhage. Niemeyer affirmed that true tubercular ulcers are not so extensive as those of non-tubercular origin.

**SYMPTOMS.**—Tubercular disease of the bowels is indicated in the child by the persistent or frequent occurrence of symptoms of intestinal irritation and catarrh; associated with the general signs of tuberculosis, marked wasting, and retarded development. In the adult ulceration may be suspected if in the course of phthisis the symptoms characteristic of this lesion should arise; especially if diarrhœa sets in, which will not yield to appropriate treatment, and if there is localized tenderness. It is in this class of cases, however, that constipation is most frequently observed, being sometimes due to peritonitis; while, on the other hand, it must be remembered that diarrhœa is often dependent upon other causes.

**TREATMENT** has been sufficiently indicated when discussing the treatment of intestinal ulceration in general.

#### IV. ALBUMINOID DISEASE.

**ANATOMICAL CHARACTERS.**—The entire alimentary canal may become the seat of albuminoid disease, and it will suffice to offer a few general remarks on this subject. In the intestines the change begins in the small vessels of the villi, and then spreads to the larger vessels; in course of time it involves the glands, the entire villi, and ultimately the whole of the mucous coat, or even the submucous and muscular coats. Extensive atrophy of the villi may follow. The morbid material is liable to undergo disintegration, being changed into a yellow substance; and finally small ulcers are sometimes formed, corresponding to the glands. It is very difficult in the early stages to recognize albuminoid disease of the alimentary canal. The mucous surface appears pale, anæmic, and glistening or shining, but the iodine-test is necessary to reveal the change, which shows that it affects the villi and small vessels. In more advanced cases the appearances are more characteristic, and enlarged glands or ulcers are seen, especially corresponding to Peyer's or the solitary glands. Peyer's patches sometimes present a reticulated aspect.

**SYMPTOMS.**—Should there be signs of albuminoid disease of other organs, implication of the alimentary canal may be fairly diagnosed if obstinate diarrhœa should set in, with liquid stools, especially if these are greenish, or in other respects of an unhealthy character. Hæmorrhage is liable to occur in the later stages, and it may be quite independent of ulceration. Implication of the stomach is indicated by persistent vomiting; with

signs of imperfect digestion. When the alimentary canal is affected with albuminoid disease, the general nutrition is necessarily gravely interfered with.

**TREATMENT.**—The treatment is that indicated for albuminoid disease in general; and for gastric or intestinal catarrh or ulceration.

---

CHAPTER XLI.

INTESTINAL OBSTRUCTION.

**ÆTIOLOGY AND PATHOLOGY.**—The numerous causes of obstruction of the bowels may be ranged under certain heads, namely:—

1. **Accumulations in their interior**, including hard fæces; indigestible matters, either taken in the food, such as oat-cakes, rice, seeds or stones of fruits, or swallowed purposely, especially by hysterical girls and children, for example, string, hair, dirt, sand; certain medicines which are apt to form concretions if taken for some time in considerable quantity, namely, magnesia and sesquioxide of iron; masses of worms; large or numerous and agglomerated gall-stones; concretions of phosphate or carbonate of lime. Gall-stones usually lodge high up in the small intestines.

2. **Stricture** resulting from morbid changes in the coats of the bowels, including:—*a. Congenital constriction*, usually situated about the anus, rarely in the duodenum. *b. Cicatrization of an ulcer*, especially if this has passed round the gut, or if it has been very extensive. *c. Fibroid infiltration* of the walls. *d. Cancer*. This class of causes is by far most common in connection with the large intestines.

3. **Compression, constriction, or traction**, due to morbid conditions external to the intestines. The late Dr. Hilton Fagge has drawn particular attention to some of these causes, which include pressure by displaced or enlarged organs and tumours of various kinds, particularly in connection with the uterus and ovaries; by growths or accumulations in the intestine itself; or by certain adhesions, agglutinations, or deposits in the peritoneum, sometimes after simple peritonitis, but more frequently associated with tubercle or cancer. The last-mentioned may lead to distinct constriction, or may cause a sudden bend or twist in the intestines; but usually they merely impede the peristaltic action, either by compressing the bowel somewhat for a considerable extent, binding it down, exerting traction upon it, or matting together several of its coils. Hence materials collect above, which press upon the portion below, ultimately inducing complete obstruction; this being frequently aided by a certain degree of spasm. These causes mainly affect the small intestines.

4. **Strangulation or incarceration**, either **external** or **internal**. Under this group come the different forms of *hernia*, the rarer varieties of which must not be forgotten. *Internal strangulation* results, in rare instances, from the passage of a portion of intestine into some normal opening, especially the foramen of Winslow; or into a perforation in one of the folds of the peritoneum, for example, the omentum or mesentery. Generally, however, it is due to peritoneal bands of adhe-

sion passing between different parts; or to the vermiform appendix, or diverticula connected with the ileum, becoming adherent at their free ends. Very exceptionally one portion of the bowel is strangulated by another portion; by the mesentery; or by its entrance into a rupture in the intestine or some other hollow viscus.

5. **Altered relation** of portions of the bowel, or of the intestinal coats. The most important form of obstruction coming under this head is that named *intus-susception*, *invagination*, or *volvulus*, in which one portion of the intestine is prolapsed into that next below. Another variety is named *torsion* or *rotation*, in which the bowel with its attached mesentery is twisted, though Dr. Bristowe considers that this twisting is in many cases not the cause of obstruction, but the effect of enteritis, which has been the primary mischief. *Prolapsus ani* also falls within this group, though it scarcely ever leads to complete obstruction. As very rare conditions have been mentioned *sacculatation* of a part of the bowel; and *hernia of the mucous membrane* through the other coats.

6. **Spasm and paralysis** of the muscular coat. Either of these conditions may aid in inducing obstruction; or now and then it may possibly be the sole cause.

With regard to the *determining cause* of intus-susception a few remarks are necessary. This condition depends upon the peristaltic action of the intestines, and is supposed to be chiefly the consequence of undue dilatation of a portion of bowel towards which the wave of contraction is advancing, from accumulation of gas or any other cause; and to this portion being fixed, so that the part above is driven into it by the force of its own contraction. In many cases some violent exertion, in which the muscles of the abdominal walls partake, determines the occurrence of intus-susception. It has also been stated to be originated by worms or polypi; and to arise frequently in connection with chronic diarrhoea. Once it has started, the invagination increases by a continuance of the peristaltic action, by which more bowel is driven in from above, at the same time the outer tube of intestine being inverted. Some of the other forms of sudden obstruction may also be immediately caused by violent effort.

Sex and age require notice as constituting important *predisposing causes* of certain varieties of intestinal obstruction. That resulting from impaction of gall-stones is by far more frequent late in life, and in females. Strictures are more common in males, and after middle life. Internal strangulation does not often occur under 30 years of age, except that form due to adhesion of the appendix vermiformis or of diverticula, which is observed in young persons most frequently, and chiefly in males. Ileo-cæcal intus-susception is remarkable for its frequency in children, but when it affects the ileum or jejunum it is almost limited to adults; on the whole intus-susception is twice more common among males than females.

**ANATOMICAL CHARACTERS.**—The appearances met with after death in cases of intestinal obstruction necessarily vary much according to the condition upon which it depends. *Intus-susception* is the only form which calls for special description. By far the most common form of invagination is that in which the *ileo-cæcal orifice descends into the cæcum*, and then passes on into the colon, bringing down more and more of the ileum. The condition is not very uncommon in the ileum or colon, but is rarely observed in the jejunum or rectum. Very exceptionally the end of the ileum passes *through* the ileo-cæcal



opening, the lips of the latter not being displaced. The portion of intestine which is the seat of invagination presents three layers, arranged concentrically in the following manner (Fig. 24):—The most internal layer is the part which has descended, or the *intus-suscepted portion* or *volvulus*; the outer one is the *sheath* or *intus-susciens*; and the middle layer unites these two portions, being derived from the continued involution of the sheath, and its surfaces are necessarily reversed, so that its serous coat is in contact with that of the internal layer, and its mucous coat with that of the external, while the mesentery belonging to the middle and external layers is drawn in, and lies between them. This exercises unilateral traction, whereby the intus-suscepted portion is curved, with the concavity towards the involved mesentery, while its lower opening looks towards some part of the wall of the outer tube, being elongated and fissure-like. The part of the intestine which forms the volvulus is more or less convoluted or twisted, especially the middle layer.

The extent of the invagination varies considerably, ranging from an inch or two to three or four feet or even much more than this, especially in the ileo-cæcal variety. Not uncommonly short intus-susceptions are found in the small intestine after death, which are easily reduced, and which have given rise to no symptoms during life; it is probable that these are originated during the act of dying, or even *post-mortem*. In the great majority of cases, whatever the length of the intus-suscepted portion may be, its lower end continues the same as at the commencement of the process. Ultimately it may reach the anus, or even protrude through this opening.

Certain important events are liable to happen in connection with the invaginated portion of intestine. 1. Of course the inner layers are more or less *compressed* by the outer tube, especially at the entrance or neck of the invagination; the canal is therefore narrowed, though not usually completely closed at first. 2. The return of blood is interfered with, and hence *mechanical congestion* is induced, often intense, leading to œdema of the tissues, or even to the escape of blood between the mucous surfaces or into the intestinal canal, where it is mixed with serum; consequently thickening and swelling arise, which increase the obstruction. 3. Soon *peritonitis* is set up in the contiguous layers of the serous coat, with exudation of lymph, and this may spread and become general; or the formation of adhesions may prevent any further descent of the intestine. 4. Violent *enteritis* is excited, and ultimately, owing to this condition being added to the congestion, *gangrene* not uncommonly results. 5. In some cases the mortified portion becomes *detached*, either completely or in part, and either in one mass or in fragments, and is *expelled per anum*. The dangers of this separation may be prevented by adhesions having formed between the top of the outer tube and the intestine above; but if these are not sufficiently firm, the structures give way, and the intestinal contents escape into the peritoneum. If the bowel is expelled and adhesions are complete, recovery may follow, but there is still a further danger of a stricture forming at the point of union; or sometimes only a part of the invaginated bowel comes away, while the upper portion remains and becomes adherent to the surrounding tube, and thus more or less permanent obstruction is

FIG. 24.

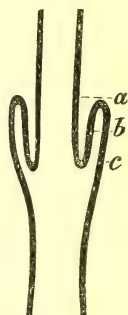


Diagram of Intus-susception:—a. Volvulus; b. Middle layer; c. Sheath.

established. The rapidity with which the changes above described are set up depends on the force of the compression, and therefore upon the part of the bowel which is involved. They are much more rapid in connection with the small intestines than the large, but are peculiarly speedy when the ileum passes through the ileo-cæcal orifice without disturbing it.

The portion of intestine above that which is intus-suscepted acts with undue vigour, and thus aggravates the mischief, while at the same time it drives on some of the contents of the bowel. For a time also the affected part itself contracts. By the pressure of its lower end against the wall of the outer sheath, ulceration of the mucous surface is often set up. In very rare instances double intus-susception has been observed.

Any sudden or acute intestinal constriction will necessarily lead to congestion of the bowel and its consequences; then to inflammation, involving also the peritoneum; and ultimately to gangrene and perforation, if the constriction is not relieved. In prolonged and chronic cases the part of intestine above an obstruction becomes much dilated, elongated, and hypertrophied, though these appearances are not always most marked directly above the impediment; while fæces and other matters accumulate, giving rise to catarrh or ulceration. The distal portion becomes contracted, empty, and atrophied.

**SYMPTOMS.**—The direct clinical phenomena which in the first instance indicate complete obstruction of the bowel, from whatever cause, may be stated generally as absolute constipation, usually accompanied with colicky pains, often severe; increased peristaltic movements of the intestines; abundant formation and accumulation of gas, leading to tympanites and borborygmi; nausea and vomiting, the latter ultimately becoming stercoraceous or fæcal. Not uncommonly *physical examination* of the abdomen and rectum reveals some abnormal condition. In many cases symptoms indicative of severe enteritis, peritonitis, or perforation are subsequently developed.

There are certain circumstances which have an important influence on the clinical history of intestinal obstruction, the most important being the pathological condition to which it is due; and its seat. Practically cases may be divided into two classes, namely:—1. Those in which the obstruction is established *gradually*. 2. Those in which it occurs *suddenly* or *acutely*. In the former class of cases there will be a history of constipation, sometimes alternating with diarrhœa, and often accompanied with alteration in the shape and size of the solid stools; colicky pains; nausea and vomiting from time to time, with other digestive disturbances; and perhaps occasional signs indicating complete temporary closure of the bowels. *Physical examination* may reveal some mechanical obstruction. These cases either terminate slowly by asthenia; or they culminate in a sudden attack of absolute stoppage of the bowels. The pain associated with intestinal obstruction is at first of a griping character, in some forms being sudden and severe, frequently starting from about the umbilicus, but it may radiate from some other locality which corresponds with the seat of the disease; after a time peritonic pain not uncommonly supervenes. Absolute constipation is not an invariable symptom, for when the small intestine is implicated, its liquid contents are able to pass along the canal unless there is a complete closure, and the same thing may happen when intus-susception affects the large intestine; further, fæces contained in the bowel below an obstruction are often expelled. Occasionally blood and mucus are

discharged, especially in cases of invagination of the large bowel. Vomiting is more easily excited, and is more severe, the nearer the stoppage approaches to the stomach. At first it is sympathetic in most cases, but soon the rejected matters have a distinctly fæcal odour, and present an appearance like pea-soup, consisting of materials which have either flowed back into the stomach from the bowels, or been forced through the pylorus by anti-peristaltic action, or by external pressure. In some instances there is more or less suppression of urine, especially if the obstruction is situated high up, this being most probably a sympathetic derangement. It is stated that indican is present in the urine in abundance when the obstruction involves the small intestine.

DIAGNOSIS.—The further elucidation of the clinical history of cases of intestinal obstruction will be best aided by considering the points on which their diagnosis is founded. This has to determine, first, the fact of the *existence* of an obstruction, and its *cause*; and, secondly, its *situation*.

In conducting the examination of any particular case the following course may be adopted:—1. The *age* and *sex* should be noted, their influence as predisposing causes of different varieties of obstruction being borne in mind. 2. Certain matters in the *past history* of the patient should be specially inquired into, namely, whether articles have been taken, either in the food or in any other way which might form concretions in the intestines; the habitual state of the bowels; and if there is any history of previous intestinal ulceration, peritonitis, the passage of gall-stones, uterine displacement, or any other condition which might give rise to pressure. 3. Any peculiar *constitutional condition* must be observed, and this may afford some aid, as, for instance, by indicating the cancerous cachexia: or the existence of phthisis, which is liable to be attended with ulceration and its consequences, or with tubercular peritonitis; of chronic dysentery; or of hysteria, in connection with which accumulations of fæces are common, and possibly intestinal paralysis might occur. 4. As regards the *history of the attack* itself, it should be first ascertained whether the obstruction has been gradual or sudden in its onset; and how long it has lasted. If it has been chronic, inquiry must be made as to what the state of the bowels has been; if any peculiar alterations in the stools have been observed; or if there have been previous attacks of complete obstruction, which have yielded to treatment. Should the obstruction be acute, it must be ascertained whether the attack can be traced to anything having been swallowed, or to any sudden effort or other cause; and if it has commenced with severe localized pain. 5. The precise *local* and *general* symptoms must of course be carefully noted, whether as indicating simple obstruction, partial or complete; or, in addition, enteritis or peritonitis; and also the rapidity with which stercoraceous vomiting sets in. 6. Thorough *physical examination* is essential, in conducting which attention should be paid to the following particulars:—*a.* All forms of *hernia* must be carefully searched for. *b.* Any *contraction* or *distension* of the *abdomen*, either general or local, must be noted, a view being also taken from behind; in the early stages this may help materially in fixing upon the seat of any obstruction. *c.* The situation may also be partly determined in some cases by observing the locality of any *violent peristaltic movements* of the intestines. *d.* Among the more important conditions discoverable by satisfactory exploration of the abdomen, which



may also point to the situation of a stricture, are *accumulations*, not forgetting impacted gall-stones, the onward progress of which can occasionally be traced; *tumours* of various kinds, either external to, or associated with the intestines; and *intus-susception*. It must be remarked, however, that even when these conditions exist, it is for many reasons frequently difficult or impossible to detect them. *e. Examination per rectum*, by means of the finger, hand, or bougie is often most serviceable. The amount of fluid or air which can be injected *per anum* has been stated, especially by the late Dr. Brinton, to aid materially in fixing upon the seat of any stoppage, but this must be by no means implicitly relied upon. *f.* Of course, should anything be *vomited* or *passed by stool*, the materials thus discharged should be properly examined. 7. In any doubtful case, it is necessary to *watch its progress* as regards its clinical course, rapidity, and termination, which may speedily afford considerable assistance in diagnosis; to observe the effects of treatment; or it might become not only permissible, but actually imperative, to have recourse to abdominal section for diagnostic purposes.

Having given this general outline of the method of investigation to be pursued, it will be well to add a brief summary of the chief clinical features presented by each class of cases of intestinal obstruction.

1. **Accumulations** are generally gradual in their progress, but in some instances, especially when due to gall-stones, the symptoms come on very suddenly. The physical signs and consequences of most of these collections have been already considered in a former chapter; and here it need only be remarked that impacted gall-stones are particularly liable to set up violent enteritis, while the course of these cases is usually very rapid.

2. **Strictures and compressions** of the bowel may be considered together. They are usually chronic in their progress, complete obstruction being preceded by gradually increasing constipation, sometimes, interrupted by attacks of diarrhoea; diminution in the size and change in the shape of the stools, should a stricture be seated near the lower end of the intestines; liability to colicky pains, sickness, and other digestive disturbances; and interference with nutrition. From time to time also there may be signs of temporary complete obstruction. There may be a history of some cause of stricture or compression; or *physical signs* may be detected, indicating some morbid condition likely to give rise to either of these mishaps. Commonly these cases linger on for a long while, even after absolute closure of the bowel has been established. Now and then, however, signs of obstruction come on suddenly, without any particular previous signs, these being followed by enteritis or peritonitis. Possibly some accumulation above the stricture may under such circumstances be the immediate cause of the symptoms.

3. **Strangulations** give rise to rapid and absolute obstruction; followed speedily by signs of severe enteritis, or even of gangrene of the intestines, perforation, and peritonitis. If not relieved, their issue is quickly fatal. Many cases belonging to this class can only be determined by exclusion, and frequently they can merely be guessed at. A previous history of peritonitis may help the diagnosis; while the immediate attack is often due to some violent exertion.

4. **Intus-susception** is also sudden in its onset as a rule, beginning with griping pain, more or less violent, usually referred to the umbilical region. Subsequently colicky pains occur from time to time, and the ordinary signs of obstruction set in, followed by those of enteritis or

peritonitis. The other important diagnostic evidences of invagination are the *passage of blood per anum*, in some cases mixed with mucus or decomposed tissues; the detection of a *sausage-shaped tumour* in the abdomen, corresponding to some part of the intestine, presenting peristaltic movements, and altering during the progress of the case as regards its direction, extent, and shape; and the *end of the intus-suscepted portion* being felt or seen on examination through the anus, or more or less of it being discharged in a gangrenous condition. In the latter case sudden perforation and its consequences may supervene. There are generally some important distinctions between invagination of the *small* and *large* intestines, namely, that in the former the symptoms are greatly more severe and acute in their progress; hæmorrhage is much more abundant, blood being also sometimes vomited; while in the case of the large bowel there is generally much tenesmus, with dysenteric stools. *Physical examination* may afford some aid in localizing the mischief. The presence of indican in the urine has been said to distinguish obstruction of the small from that of the large bowel. A large proportion of the cases of intus-susception end fatally, those in which the large intestine is involved sometimes lasting for many weeks or months. The several events which may happen in their course are indicated in the account of the morbid anatomy.

5. It is scarcely practicable to indicate the characters of obstruction from **spasm** or **paralysis** of the muscular coat. The occurrence of chronic constipation in a hysterical female, ending in complete obstruction, might suggest paralysis, though probably the previous accumulation of fæces actually originates the obstruction. It generally yields to treatment.

PROGNOSIS.—Without entering into details, it will be evident that all forms of obstruction of the bowels are exceedingly dangerous. The cases most speedily fatal are those due to strangulation or intus-susception. The chronic varieties are liable at any moment to end in complete closure. Accumulations may often be got rid of, and thus recovery be brought about.

TREATMENT.—The treatment must be separately considered, according as intestinal obstruction is *chronic* and *gradual* in its progress; or *sudden* and *acute*.

1.—In *chronic* cases the main principles are to regulate the diet strictly, allowing only liquid or pultaceous, highly digestible, and nutritious articles, in moderate quantities; to endeavour to keep the bowels acting comfortably, for which purpose mild enemata answer best, at the same time avoiding the use of strong purgatives; to remove, if possible, anything causing compression, as well as any accumulation; to support the strength of the patient, and improve the general condition; to treat troublesome symptoms referable to the digestive organs; and, in appropriate cases, to have recourse to certain operations.

Should there be a *stricture in the rectum*, it may often be dilated successfully by the cautious use of the *bougie*. In certain instances also it is desirable to make an artificial anus above an obstruction, as described in surgical works, which may prolong life considerably, at the same time giving marked relief.

2. In cases of *acute* obstruction, from whatever cause, a matter of prime importance is not on any account to excite the intestines by giving powerful purgatives. It is allowable to use enemata cautiously,

so as to clear out the bowel below the seat of obstruction. Of course little or no food should be taken by the mouth, and very soon the smallest quantity is immediately rejected; therefore all the necessary support, including *stimulants* when required, must be administered *per rectum*, and frequently considerable quantities of the latter are needed. *Digestants* must be added to the food given by enemata. The patient may be permitted to suck ice freely. The most important internal remedy is opium in full doses; or subcutaneous injection of morphia may be employed. Belladonna is also of much value, or this drug may be combined with opium. In the treatment of intus-susception, tobacco has been employed, usually in the form of an enema of its infusion, but it is a dangerous remedy. *External applications* over the abdomen, of dry heat, poultices, fomentations, turpentine stupes, or sinapisms are very serviceable to relieve symptoms. Vomiting and other symptoms require the usual remedies for their relief. In the treatment of intus-susception, the gradual injection of a large amount of liquid or air *per anum* has been frequently practised, and apparently with benefit. This injection may be effected by means of the syphon-enema, as in a case reported by the late Dr. Pearson Irvine (*Medical Times and Gazette*, Nov. 25, 1876), in which this method of treatment, followed by subcutaneous injection of atropine, proved successful.

The question of an *operation* presents itself in many acute cases of intestinal obstruction. Of course if there is any evident or suspected hernia, surgical interference is necessary. Another operation which might be indicated, and which has been successfully accomplished, consists in opening the abdomen, with the view either of removing some internal strangulation, or of reducing an invagination. If there is good reason to believe that the former condition exists, it is decidedly permissible to risk opening the abdomen, especially if the case seems otherwise hopeless. As regards intus-susception, it is considered by most authorities only allowable to attempt its reduction when the large intestine is involved. Under any circumstances the results are not very satisfactory.

---

## CHAPTER XLII.

### INTESTINAL WORMS—HELMINTHIASIS.

IN the present chapter it is intended to give a brief account of the main facts relating to those animal parasites which infest the alimentary canal of human beings; but it will be convenient, in noticing their life-history, to allude to another phase of their existence, as they are found in other organs of the body. For a complete account of this subject the reader is referred to Dr. Cobbold's valuable works. An excellent description of the natural history of tape-worm is also given by Dr. J. Davies Thomas, of Adelaide, in his recent work on Hydatid Disease.



The ordinary intestinal worms include:—I. **Cestodes** or **tape-worms**. *a.* *Tænia medio-canellata*. *b.* *Tænia solium*. *c.* *Bothriocephalus latus*. 2. **Nematodes**. *a.* *Ascaris lumbricoides* (*round-worm*). *b.* *Oxyuris vermicularis* (*thread-worm* or *seat-worm*). *c.* *Tricocephalus dispar* (*hair-headed* or *whip-worm*). Among rare varieties are mentioned *tænia nana* or *egyptica*, *elliptica*, and *flavo-punctata*; *bothriocephalus cordatus*; *ascaris mystax*; *dochmius duodenalis*; *distoma crassum* and *heterophyes*.

**ÆTIOLOGY AND DEVELOPMENT.**—It seems tolerably certain that no intestinal worm ever develops in the bowel directly from an ovum deposited there by a previous tenant, but that this must be first discharged and undergo metamorphosis, being afterwards conveyed by some means through the mouth into the alimentary canal in a certain stage of development, and when it reaches its peculiar habitat it grows into the adult animal. As regards the development of the *nematodes*, an embryo forms in each ovum after its discharge in the stools, or, in the case of the thread-worm, even while it is in the intestines; no further change occurs so long as the ovum is outside the body, though it may retain its vitality for a long period. In this condition it is supposed to enter the alimentary canal in various ways, such as in water, vegetables, fruit, or impure starchy substances. The ova of thread-worms may also probably be carried to the mouth by the agency of the fingers or nails of a person already infected, these having been previously used for the purpose of scratching the anus. Certain experiments seemed to show that the embryos of round-worms would not undergo any further change when introduced into the stomach in the free state, and it was suggested that they are taken up by some other animal, such as a small worm, or an insect or its larva, and then swallowed along with vegetable and other articles of diet. Hellier's observations, however, are in favour of the view that the round-worm completes its cycle of existence without having to pass through the body of any intermediary animal bearer. This is the opinion strongly held by Dr. Cobbold.

The development of *tape-worms* is better known. Segments of these, containing abundant ripe ova, separate and are discharged *per anum*, or even break up within the bowel; the ova escape and are scattered about in various ways; they are then swallowed by different animals, especially by pigs, oxen, and sheep, mixed with their food. In the alimentary canal of these animals the shell of the ovum ruptures, and then the embryo (*proscolex*) escapes, attaches itself to the mucous surface, and works its way into the tissues, until it reaches a suitable spot, where it settles down and undergoes further changes, presenting a head and neck with appendages like those of a tape-worm (*scolex*), from which a vesicular appendage or bladder hangs down. In this stage the worm is named a *cysticercus* or *bladder-worm*, such as is seen in the muscles, liver, brain, and other organs and tissues of different animals, sometimes in human beings. Each tape-worm has a special form of *cysticercus*; that of the *tænia solium* is named the *cysticercus cellulosus*; that of the *tænia medio-canellata* the *cysticercus medio-canellata*. This *cysticercus* may remain for some years, or may finally perish; if, however, it in any way reaches the alimentary canal of the particular class of animal which it infests in the adult condition, it becomes attached by the head, the vesicle falls off, and then a succession of segments form, constituting the ordinary tape-worm. The usual way in which these larvæ reach the stomach is in consequence of an individual eating

the raw or imperfectly-cooked flesh of the animals which they infest; thus the *tenia solium* comes from pig's flesh (measly pork); the *tenia medio-canellata* from beef; while the *bothriocephalus latus* is believed to be conveyed by fish or molluscs.

Different varieties of tape-worm are found in different parts of the world. In this country the *tenia solium* and *medio-canellata* are the forms met with, the latter quite as frequently as, if not more frequently than, the former. *Bothriocephalus* is prevalent in Eastern Europe as far as the Vistula, and in Switzerland, especially along sea-coasts and rivers. Tape-worm is by far most frequent in those countries where much pig's flesh is consumed, and individuals who do not eat this kind of food, such as Jews,

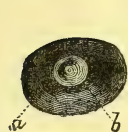


FIG. 25.†



FIG. 26.‡

are particularly exempt from the complaint. It is frequently observed among those who, in connection with their occupation, are in the habit of putting knives used for cutting raw meat into their mouths, such as butchers, cooks, &c.; and also among those who indulge in raw or very underdone meat, or in sausages and such articles, as then the parasites are not destroyed. It must be remembered that in this country beef is often the meat by which tape-worm is conveyed. The *bothriocephalus* is supposed to be taken along with drinking water. Women suffer from tape-worm more frequently than men; and usually the persons affected are between 16 and 40 years of age.

Round and thread-worms are principally found in children, especially if they are in bad health, or improperly fed and dirty. Round-worms are very prevalent in some parts of the world, namely, in the Southern States of America, Greenland, Iceland, Brazil, and in some parts of Holland, Germany, and France, especially in low and damp districts. They are common enough in this country. It is presumed that an unhealthy condition of the enteric mucous membrane, leading to the formation of much viscid mucus, favours the development of worms.

DESCRIPTION.—Only the main characters of the ordinary worms can be noticed in this work, so as to enable them to be recognized.

1. **Tape-worms.\***—In the adult form (*strobila*) tape-worms are elongated, narrow, flattened, or tape-like in form, consisting of a head, a neck, and a series of thin, flat, quadrilateral segments or links (*proglottides*), varying in number according to the length of the worm, united by a softer and more transparent tissue. The links grow from behind the neck by a process of budding, and then pass on, making room for those more recently formed, so that the oldest are the most distant from the head. At first they are very small, but enlarge considerably as they become more mature, at the same time altering in form somewhat, and presenting a more complicated organization. Tape-

\* A complete tape-worm is now regarded as a "peculiar animal colony, consisting of more or less numerous individuals temporarily connected together for the common advantage, but capable of leading separate, and quite independent existences."—(Thomas).

† Fig. 25.—*Cysticercus cellulosus*, from the human brain, of its natural size, and with a retracted anterior extremity (b).

‡ Fig. 26.—The same *cysticercus* extruded; a. The caudal vesicle of the cysticercus, which is nothing but the *receptaculum scolice* (or hinder end of a tænid embryo), distended into a vesicle by the accumulation of water. c. The transversely-wrinkled anterior extremity of the cysticercus. d. Its head and neck, which conjointly form the tænid scolex. (Von Siebold.)



worms are parenchymatous in structure, consisting of a soft, whitish, or yellowish-white contractile tissue; having no mouth or alimentary canal; but presenting a water-vascular system communicating between the segments, and well-developed sexual organs. These are not evident in the most recent links. The female apparatus appears first as a median tube with lateral branches, subsequently becoming more divided and developing ova, which almost completely fill the terminal links, rendering them opaque, and in these segments embryos may be visible. The male organs consist of tortuous seminiferous tubes, and a penis. Each segment is hermaphrodite, and the sexual orifice is either single or double, opening either laterally or on one of the surfaces. All the varieties of tape-worm inhabit the small intestines ordinarily; rarely one may enter the large bowel or the stomach. As a rule only a single worm is present; occasionally there are two or more.

1. *Tania Solium*.—Length varies from a yard to 100 or 150 feet or more, but the average is stated by different authorities at from 5 or 7 to 20 or 30 feet. *Head* very small, somewhat globular or bulbous, with a slightly prominent conical snout or *rostellum* in front, surrounded by a double row of curved silicious hooks, from 12 to 15 in each row; and further back 4 suckers, symmetrically arranged. *Neck* extremely slender; from  $\frac{1}{2}$  an inch to nearly an inch long; transversely marked. *Segments* in their earliest stage very small, and much broader than long; gradually become more flattened and altered in the relation of their diameters, so as to be first square, and afterwards oblong, being much longer than broad, with the ends narrowed, especially the anterior extremity. Mature links measure about  $\frac{1}{2}$  an inch long, and  $\frac{1}{4}$  inch broad. *Male* and *female* organs open by one orifice, which is situated laterally in a little projection, now on one side, now on the other, but not regularly alternating.

2. *Tænia Medio-cancellata*.—Has a general resemblance to *tænia solium*, with the following differences:—*Length* usually greater. *Head* larger, has neither snout nor hooks, being flattened in front, but its four suckers are very prominent and powerful. Leuckart describes a fifth smaller one between them. *Links* more numerous, broader, thicker, and firmer. *Sexual organs* more developed and divided; and orifice situated near the posterior border.

3. *Bothriocephalus Latus*.—Length very considerable. Head obtuse or club-shaped, having no hooks or prominences, but merely two longitudinal slits or grooved suckers, one on each side. Neck very short. Segments exceedingly numerous; not distinctly visible for a little distance from the head; at first nearly equal in diameters, but soon

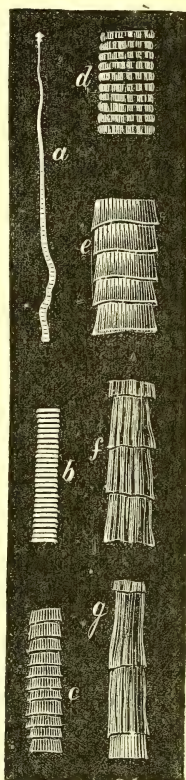


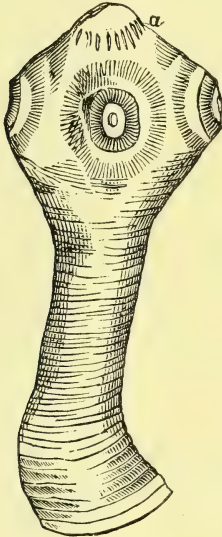
FIG. 27.\*

\* Fig. 27.—*Tænia solium armata* (of the natural size). Fragments taken at certain distances between the head and the posterior rings, in order to show the successive form of these rings; the order of the letters indicates their arrangement from before backwards. —(Duvaine.)



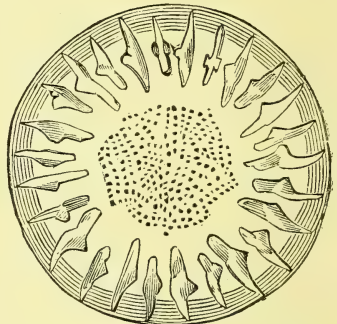
much broader than long; have a slightly brownish colour. *Sexual openings* in the middle of one surface of each segment, near its posterior border, and not lateral; they are distinct, that of the male apparatus being anterior. *Ova* of a brown colour.

FIG. 28.



Head and neck of *Tænia solium* magnified.  
a. Circle of hooks.

FIG. 29.

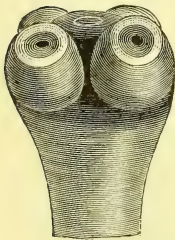


Circle of hooks, more highly magnified.  
(Leuckart.)

**2. Nematodes.**—The main characters of the chief *nematodes* are indicated in the following description:—

1. *Ascaris Lumbricoides*.—*Form* elongated and cylindrical, but tapering towards the ends, especially the anterior. *Length* from 6 to 12 or 16 inches; and diameter 2 to 3 lines. Appears reddish, greyish-red, or yellowish-white, semi-transparent, firm, and elastic. *Head* has three small prominences, with the mouth between them lined with numerous teeth. A circular depression separates it from the body. *Body* presents fine transverse markings. *Sexes* are distinct. *Male* shorter, and curved posteriorly, where the sexual organs are placed. *Female* straighter, and thicker at the hinder extremity; has the sexual opening about the end of the anterior third.

FIG. 30.



Head of *Tænia medio-canellata*, highly magnified.—(After Fritsch.)

*Habitat* small intestines, but often migrates into the large bowel, and out through the anus; or, rarely to the stomach, œsophagus, mouth, nares, frontal sinuses, windpipe, bile and pancreatic ducts or gall-bladder, peritoneum, vagina, urinary organs, and various other parts.

*Number* usually several; may be hundreds; sometimes only one.

2. *Oxyuris Vermicularis*.—Very small and delicate; fusiform; *males* measuring from 1 to 2 lines long; *females* about 5 lines. Whitish and

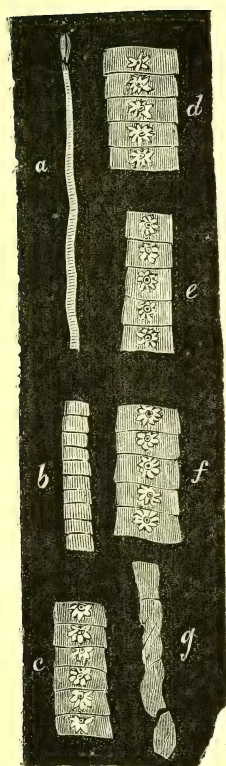


FIG. 32.†

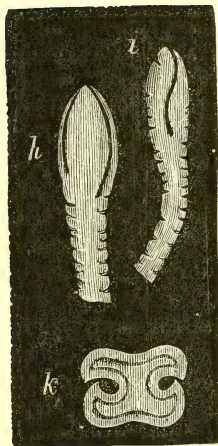


FIG. 31.\*

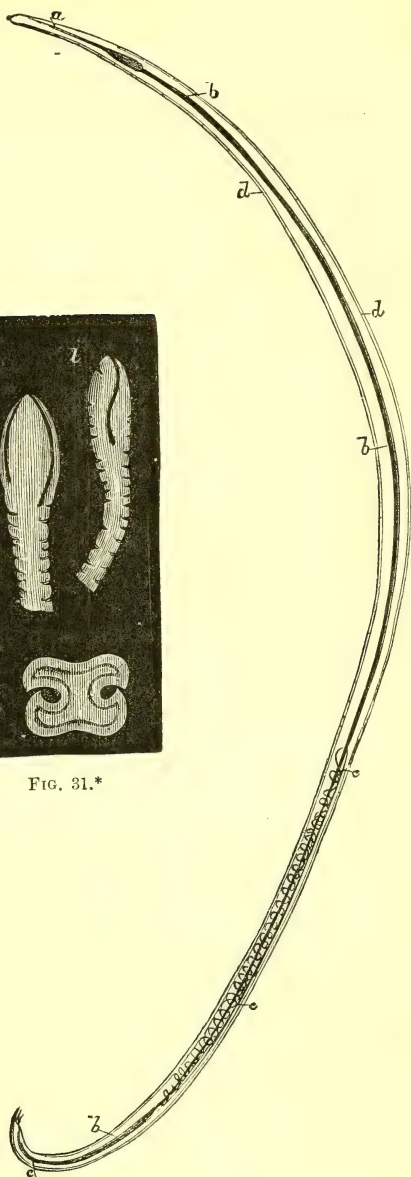


FIG. 33.‡

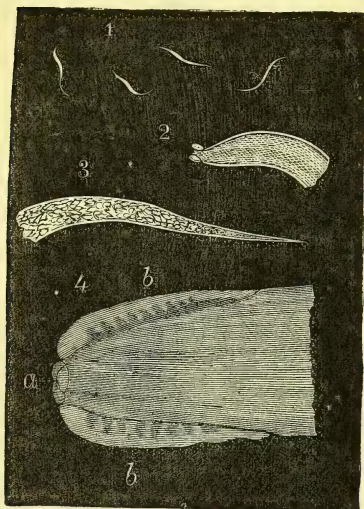
\* Fig. 31.—*i* and *h*. Head of the *Bothriocephalus latus*, enlarged six times and seen in two different positions. *k*. Transverse section of the head of the bothriocephalus found in the turbot, magnified twelve times; this is introduced into the figure for the purpose of showing the arrangement of the lateral suckers.—(*Davaine*.)

† Fig. 32.—The *Bothriocephalus latus*, of the natural size, the fragments being taken at certain distances; the order of the letters indicates their relative situation, from the head to the posterior extremity; in *c*, *d*, *e*, *f*, the genital pore is visible; *g*, some of the terminal rings shrivelled up, after the deposition of the ova.—(*Davaine*.)

‡ Fig. 33.—Male of *Ascaris lumbricoides*, of the natural size. *a*. Oesophagus; *b*. Intestinal canal; *c*. Spermatic ducts; *d*. Lateral longitudinal line.—(*Küchenmeister*.)

semi-transparent; surface presents fine transverse striæ. *Head* has a terminal mouth, with 3 scarcely evident lips, and a wing-like expansion on the dorsal and ventral aspects. *Male* is rolled up posteriorly, where the sexual organs are placed. *Female* is straight or but slightly bent, and has the vulva about the junction of the anterior and middle thirds.

FIG. 34.

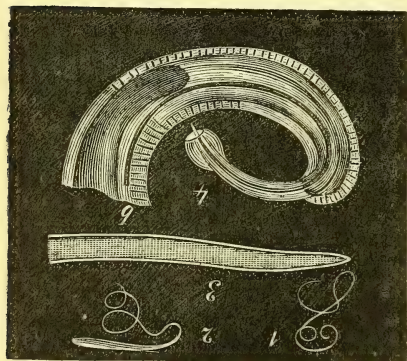


Female *Oxyuris vermicularis*: 1. Four oxyurides of the natural size. 2. The cephalic end magnified; the esophagus and stomach are shown. 3. The caudal extremity magnified. 4. The head greatly enlarged; *a*, the mouth furnished with three lips; *b b*, the lateral expansions of the integument.—(Dacaine.)

The head is generally imbedded in body moves freely.

*Number* usually not great, but may be hundreds.

FIG. 35.



The *Tricocephalus dispar*: 1. The male, of the natural size. 2. The female of the natural size. 3. The head magnified. 4. The tail, also greatly enlarged.—(Dacaine.)

*Habitat* the cæcum, but migrate to the lower part of colon and rectum. Often migrate around the anus, into the vagina, urethra, or under the prepuce. Have been seen in [the small intestines and stomach.

*Number* usually very great; hundreds or thousands.

3. *Tricocephalus dispar*.—Thread-like in form, being from 1 to 1½ or 2 inches long. Posterior end thicker than anterior, which is hair-like, and ends in a simple terminal mouth. *Male* is the smaller, and is spirally coiled posteriorly. *Female* is larger and thicker, only slightly curved; the uterus contains an immense number of ova.

*Habitat* usually the cæcum, rarely the colon, very rarely the ileum, the mucous membrane, while the

#### SYMPTOMS AND DIAGNOSIS.—

Worms frequently exist in the alimentary canal without setting up any evident symptoms. When present these are usually indicative of (1) *local irritation*; (2) *reflex disturbance*; and (3) more or less *constitutional disorder*. Occasionally intestinal worms lead to local congestion, inflammation, superficial erosion of the mucous surface, or even to slight ulceration; in exceptional cases they may even cause obstruction of the bowels; or, by migrating into the stomach, bile-ducts, liver, larynx, peritoneum, and other parts, may originate dangerous symptoms referable to either of these structures. It may be stated as a general rule

that the symptoms are more marked in delicate and weakly persons,



or in those whose nervous system is highly susceptible; and that they are proportionate to the number and size of the worms.

The *local* symptoms which may be produced by *tape* and *round-worms* are uneasiness, curious sensations, or actual griping pain in the abdomen, especially about the umbilicus; sometimes attacks of severe colic, attended with vomiting or retching, and faintness; capricious and variable appetite, often with craving for special and indigestible articles of food; furred tongue, and foul breath; nausea or vomiting; irregularity of the bowels, constipation and slight diarrhoea alternating from time to time, the stools sometimes containing mucus; and flatulence, with distended abdomen. The main *reflex* phenomena described include itching at the various mucous orifices, which causes the patient to scratch the anus, or pick the nose; salivation; grinding of the teeth during sleep, which is uneasy and disturbed; dull frontal headache, with giddiness; noises in the ears; squinting, dilated pupils, oedema of eyelids, flashes and specks before the eyes; twitchings of the limbs or facial muscles, or even violent general convulsions ending fatally; choreic, hysterical, epileptic, or maniacal attacks; deranged menstruation; palpitation; and a feeling of constriction in the throat. Among the *general* symptoms which may be observed are more or less wasting; pallor; a feeling of debility and languor; pains in the limbs; fretfulness and depression of spirits.

From a diagnostic point of view these symptoms are by no means characteristic of worms, and it is often a question how far they are originated by their agency; still when such phenomena are present, especially in children, worms should always be thought of. The diagnosis may be verified by the passage *per anum* of fragments of a tape-worm, or of entire round worms; and, if necessary, remedies may be given with the view of aiding their expulsion. Microscopic examination of the stools, for the purpose of discovering ova, is recommended in suspected cases. Portions of tape-worm sometimes escape spontaneously as the patient is walking along.

*Thread-worms* are very common in weakly and dirty children, and as they are often extremely numerous, they give rise to much local irritation, causing severe itching and tickling about the anus, which leads to constant scratching; this is especially intense towards night, and may gravely interfere with sleep. I have also met with cases in which they caused much annoyance in adult females. Occasionally these worms excite considerable dysenteric symptoms, and not uncommonly they originate *prolapsus ani*. They also frequently pass into the vagina, causing much irritation here, and inducing catarrh, undue sexual excitement and masturbation, or now and then severe hæmorrhage. By getting under the prepuce, thread-worms also promote the habit of masturbation in males. On examination they may often be seen moving in the vicinity of the anus; as well as in abundance in the stools. Various reflex symptoms are often attributed to thread-worms, but with doubtful propriety.

The *tricocephalus dispar* does not give rise to any symptoms.

PROGNOSIS.—Most intestinal worms can be readily got rid of, if properly treated. *Tape-worms* are sometimes difficult to remove completely, but with systematic management a cure may almost always be effected. It is the safest plan to see that the head of a tape-worm is discharged, else if this remains a further growth will probably take place; however, it is affirmed that if only the head and a small portion

of the neck is left, the worm will die; and, further, the nearer the head any portion is which is detached, the more easily will the rest be got rid of. Worms may now and then prove highly dangerous by their migrations, or by causing obstruction of the bowels; death may also occur in children from reflex convulsions excited by their agency.

TREATMENT.—1. If worms are present in the intestines, of course the first object in treatment is to get them expelled. The remedies for this purpose must vary with the nature of the parasite. For *tape-worm* the following plan of treatment is usually efficacious:—To make the patient take only liquids, such as milk and beef-tea, for a day; then to administer a full dose of castor oil in the evening; and, finally, early on the following morning, if the oil has acted well, to give a draught containing the liquid extract of male fern, in the dose of  $\mathfrak{m}$  x to  $\mathfrak{z}$ i or  $\mathfrak{z}$ iss, according to age. The draught may be made up with sugar, mucilage, and milk; or with the yolk of an egg and cinnamon water. The object of this plan is to clear out the bowels so as to expose the worm, and then the male fern acts upon it and kills it. Sometimes it is desirable to follow up the draught with another dose of castor oil, but generally this is not needed, as the drug itself acts as a purgative. Some authorities prefer giving the extract in smaller doses, frequently repeated; others employ the powdered fern. In order to see whether the head of the worm is discharged, each stool must be received into a separate vessel, then mixed with water, and filtered through coarse muslin.

Other *anthelmintics* employed for the destruction of *tape-worms* are kousso, followed by a cathartic; kamala powder ( $\mathfrak{z}$ i to  $\mathfrak{z}$ ij in treacle or syrup); decoction of the bark of the root of pomegranate ( $\mathfrak{z}$ ij in Oj, boiled down to Oss); powdered areca nut; oil of turpentine ( $\mathfrak{z}$ i to  $\mathfrak{z}$ ss); and petroleum ( $\mathfrak{m}$ xx to xxx). If the worm projects through the anus, it has been recommended to roll it gradually round a piece of stick, and thus draw it out; or to apply some poisonous agent to the protruded portion.

For *round-worms* the most efficient remedy is *santonin*, which is the active principle of the popular so-called worm-seeds. It is well before administering this drug to give an *aperient*, such as a little jalap with scammony; and to limit the diet to liquids for a day. *Santonin* may be employed alone, gr. i-v every morning, for two or three days, mixed with sugar or syrup, or made up into lozenges, or with gingerbread. It seems to be more efficacious when mixed with castor oil, and Kückenmeister advises that from gr. ij-iv be dissolved in  $\mathfrak{z}$ i of the oil, and  $\mathfrak{z}$ i taken every hour until it acts. Other preparations used containing *santonin* are an ethereal extract of worm-seeds, and *santonate of soda*. *Mucuna* and powdered tin are also employed for the expulsion of round-worms, which act by causing mechanical irritation. Some practitioners rely merely upon strong *purgatives*.

*Santonin* is also useful internally in the treatment of *thread-worms*, but these are decidedly best got rid of by means of *enemata*, of which many kinds have been employed. Any of the following will answer well, namely:—Common salt and other alkaline salts dissolved in water or gruel ( $\mathfrak{z}$ i to Oj); *santonin* with castor oil; infusion of quassia; infusion or decoction of wormwood; tincture of steel ( $\mathfrak{z}$ i to Oj of water or infusion of quassia); olive oil; lime-water; decoction of aloes; decoction of rue; turpentine with gruel; or even mere water, if employed freely for a few days. Cleanliness is of great importance.

The *tricocephalus* requires no special treatment.

2. In all cases of intestinal worms it is essential to look to the state of the *general health*, and to improve this by means of steel and other *tonics*, with cod-liver oil, if required; as well as by the regulation of diet, and attention to hygienic measures. The *alimentary canal* must also be attended to, and the bowels kept freely acting, so as to prevent accumulation of unhealthy mucus. Scammony, jalap, rhubarb, and castor oil are the best aperients in these cases, and either of the powders may be advantageously combined with carbonate of soda or magnesia.

3. The *prevention* of worms is a matter of considerable importance in some parts of the world, especially as regards tape-worms, and this can only be effected by taking every precaution against those habits mentioned under the *ætiology*, by which the ova are conveyed into the stomach, such as eating raw or partially cooked meat; putting knives into the mouth; or drinking impure water. Stools known to contain any kind of worms or their ova should be immediately destroyed. Of course meat that is measly ought on no account to be taken as food. In the case of children, important prophylactic measures against the development of worms consist in the maintenance of good health; the preservation of the digestive organs in a satisfactory condition; and attention to cleanliness.

#### TRICHINOSIS.

It will be convenient to consider in the present chapter a disease due to the entrance into the human body of a parasite named *trichina spiralis*. This complaint is very rarely met with in this country, but is not uncommon in some parts of the continent, where it occurs sometimes as an epidemic.

**ÆTIOLOGY AND PATHOLOGY.**—Trichinæ are introduced into the human body solely by eating pig's flesh in which they exist, either in a raw or imperfectly cooked condition, or in the form of pickled and smoked articles, sausages, and such articles. When this reaches the stomach and bowels, the parasites are liberated and develop with great rapidity, the females being by far the more numerous and the larger, and originating an immense number of young trichinæ, which perforate the intestinal wall, migrate along the mesentery to the spine, and pass thence to all parts of the body, entering into the substance of the muscles, penetrating even the sarcolemma. These structures constitute their *habitat*, and here they set up inflammatory action, becoming surrounded by a capsule or shell. It is supposed that there are several productions of young trichinæ in the alimentary canal, with subsequent migrations.

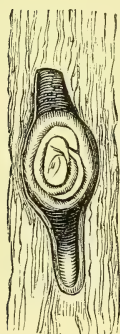
**ANATOMICAL CHARACTERS.**—In man trichinæ at first excite gastro-enteric catarrh, often attended with enlargement of the mesenteric glands. After about the fifth or sixth week the muscles, when examined with a lens, are seen to present fine striæ or minute dots, of a greyish-white and opaque aspect, which are collections of the parasite, contained in capsules or cysts produced by their irritation. These become more abundant as the case advances, and they are chiefly observed in the muscles of the loins, the diaphragm, intercostals, muscles of the neck, eye, larynx, and tongue. In the limbs they are mainly found in those nearest the trunk, being most numerous near their tendinous attachments. The affected parts feel unusually firm and resistant. On micro-



scopic examination the muscular fibres are found to be more or less destroyed, and the interstitial connective tissue increased. Each little cyst (Fig. 36) is somewhat ovoid in shape, being at first transparent, but soon becoming thicker and more opaque, and ultimately calcifying. The *trichina* (Fig. 37) is coiled up in its interior, and is very minute, the female being larger than the male. The head is finely pointed, unarmed, with a minute mouth in the centre. In fatal cases of trichinosis extensive bronchitis, pulmonary congestion or inflammation, venous thrombosis, and parenchymatous degeneration of various organs are frequently observed.

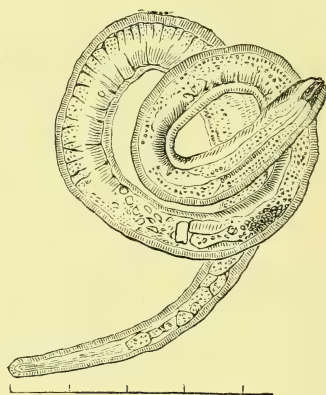
**SYMPTOMS.**—Trichinosis generally begins with symptoms of more or less gastro-enteric disorder, such as a sense of pressure and fulness in the epigastrium, impaired appetite, discomfort after eating, nausea or

FIG. 36.



Slightly magnified cyst of *Trichina Spiralis*.—(Virchow.)

FIG. 37.



100th of an inch  $\times$  300.

The *Trichina Spiralis* removed from its cyst.  
—(Virchow.)

vomiting, eructations, colicky pains, and diarrhoea, with a feeling of much languor and depression; in some cases the onset is characterized by violent sickness and purging, simulating cholera or irritant poisoning. Occasionally the disease sets in quite insidiously, with merely a feeling of lassitude and depression, wandering pains, and stiffness in the limbs. The subsequent characteristic symptoms are those dependent upon the condition of the muscles. Those of the limbs which are affected become painful, tender, swollen, hard, and rigid; there is much stiffness, movement being greatly impaired, and the joints are fixed in a state of more or less flexion, any attempt to extend them causing severe pain. From implication of various muscles there may result attacks of severe dyspnoea, aphonia, trismus, dysphagia, impaired movement of the tongue, and other symptoms. A peculiar oedema is also observed, affecting the face and eyelids, and extending in the limbs from the upper part towards the hands and feet.

Symptomatic pyrexia accompanies this condition, often severe, the temperature sometimes rising to  $106^{\circ}$ , and the pulse to 120 or 140; abundant clammy perspirations may be observed, and occasionally sudamina appear. In cases tending towards a fatal issue low typhoid symptoms set in, frequently accompanied with signs of bronchitis, pneu-

monia, and other inflammatory affections. Should recovery ensue, the muscular symptoms subside, as well as the pyrexia, but convalescence is usually protracted, marked debility, anæmia, and œdema remaining for a considerable time.

DIAGNOSIS.—Trichinosis may in severe cases be mistaken at first for cholera or irritant poisoning. It may also simulate typhoid fever in the early stage. After a time the symptoms referable to the muscles are quite characteristic.

TREATMENT.—To *prevent* trichinosis, meat containing the parasites must be avoided, and microscopic examination of pig's flesh is practised in some parts of the continent, before it is allowed to be sold. In order to be quite safe, the best plan is never to eat any pig's flesh which has not been thoroughly cooked. In the treatment of the actual disease, a matter of the first importance is to get rid of the trichinæ from the alimentary canal, by means of castor oil or some other aperient, which may be given even though diarrhœa should be present. Benzine, carbolic acid, and other drugs have been administered with the view of destroying the parasites, but it is doubtful whether they succeed in this object. The *general* treatment must be of a supporting character, quinine and *stimulants* being also administered. Hot and anodyne fomentations, or warm baths, most effectually relieve the symptoms connected with the muscles. Other symptoms and complications must be treated as they arise.

---

## CHAPTER XLIII.

### DISEASES OF THE LIVER AND ITS APPENDAGES.

#### CLINICAL CHARACTERS.

THE liver is an organ which performs several functions highly important in the animal economy. The most obvious of these is the formation of bile, which is not only useful for purposes connected with digestion, but is also undoubtedly eliminatory. Consequently biliary derangements have always occupied a prominent place, both with the profession and the public, in accounting for the causation of a large number of ailments. The liver has also a glycogenic function, and the disorders to which this function is supposed to be liable have already been discussed in connection with diabetes. Moreover, it appears that albuminous substances, especially fibrin, are reduced in the liver to various simpler compounds, such as leucine and tyrosine, and some physiologists even affirm that urea is formed in this organ. As the result of functional derangements of the liver a number and variety of morbid conditions and symptoms have been supposed to originate, not only associated with the alimentary canal, but also with remote organs, and with the general system, including the gouty state or lithæmia. For a full discussion of this subject reference may be made to the work on Diseases of the Liver by the late Dr. Murchison. In my opinion the view which attributes all these disorders solely to the liver is too exclusive; at any rate they cannot be discussed here, and in the following summary of the clinical phenomena connected with hepatic diseases, only those will

be referred to which are obviously associated with the liver, and which are met with more or less when this organ is the seat of organic diseases, or when its functions are interfered with in various ways.

1. **Morbid sensations** connected with the liver are referred mainly to the right hypochondrium, but may extend across the epigastrium to the opposite side, or they may shoot in various directions. They include different kinds of pain, with or without tenderness; or merely a sense of uneasiness, fulness, weight, and heaviness. Sympathetic pains in the right shoulder are supposed to be very common in hepatic diseases.

2. Some important symptoms result from **disturbance of the biliary functions**, the chief being those associated with *jaundice*, which will be specially discussed. Bile may also be secreted *in excess*, thus acting as an irritant, and causing bilious diarrhoea and vomiting. It may further be *deficient in quantity* or of *improper quality*, hence originating signs of deranged digestion in the intestinal canal.

3. **Obstruction of the portal circulation** leads to mechanical congestion of its tributary veins. The obvious clinical phenomena which may result therefrom are those indicating gastro-intestinal catarrh and its consequences; hæmorrhage into the alimentary canal; ascites; enlargement of the spleen; distension of the superficial abdominal veins; and hæmorrhoids. After death the veins within the abdomen are often found much enlarged and varicose; while the spleen and pancreas present the usual morbid changes which follow long-continued venous congestion.

4. Important symptoms arise from disturbance of the **glycogenic functions** of the liver. These have already been sufficiently discussed under *diabetes*, and they are not noticed in connection with mere local diseases of this organ.

5. If the liver is enlarged, it sometimes originates symptoms by **pressing upon neighbouring structures**, such as the diaphragm, inferior vena cava, or duodenum.

6. **Physical examination** of the liver may demonstrate either displacement; alteration in shape; enlargement; contraction; or alteration in the characters which the organ presents on palpation. The general characters of *hepatic enlargement* are as follows:—(i.) Its site corresponds to that of the liver, or there is a history of its having grown from this direction; it does not descend into the pelvis, but can be traced within the margin of the thorax, and appears to be superficial; sometimes it is distinctly visible, or even bulges out the lower part of the chest. (ii.) Though the dimensions may be very great, yet as a rule the normal general outline of the liver can be traced more or less distinctly; while the sensations on palpation are often sufficiently characteristic. (iii.) The organ is somewhat movable on manipulation, but not to any marked extent. (iv.) On percussion there is absolute dullness, with considerable sense of resistance generally; the dullness can be traced upwards towards the chest, and may have the curved outline regarded as characteristic of the liver; it is, however, influenced by different degrees of distension of the stomach and bowels. (v.) The movements of the diaphragm are often interfered with, especially on the right side; but the liver is generally a little altered in position by deep breathing. (vi.) Posture may also affect the organ, it being more prominent and lower in the abdomen in the standing posture.



7. Occasionally the **gall-bladder** presents an enlargement, which has the following general characters:—(i.) It usually occupies the right hypochondrium, and can be felt coming from underneath the margin of the liver, appearing to be superficial; occasionally, however, it is so much enlarged as to extend down to the crest of the ilium. (ii.) As a rule the shape is pyriform, with the base towards the abdominal wall. (iii.) The surface is generally smooth, and the enlarged organ has in the great majority of cases an elastic or fluctuating feel. (iv.) Almost always the tumour is very movable from side to side, turning on a fixed point, which lies under the liver; even a change of posture may alter its position considerably. Now and then it is fixed by adhesions.

---

## CHAPTER XLIV.

### ON SYMPTOMS CONNECTED WITH THE LIVER.

#### I. HEPATALGIA.

THE occurrence of intermittent attacks of severe pain deep in the region of the liver has been attributed, especially by the late Dr. Anstie and by Dr. Spender, to a simple neuralgia in some instances. Dr. Allbutt also recognizes hepatalgia, but regards it “as a pain aroused by the coincidence of an impressionable or neurotic habit, with the presence of gall-stones at rest in the bladder.” This affection is but a part of a general nervous condition, attended with similar pains in other parts, as well as with deep mental depression. The attacks are not accompanied with vomiting, but it is said that there may be jaundice. The main difficulty in diagnosis lies in separating the pain of hepatalgia from that due to the passage of a gall-stone.

#### II. JAUNDICE—ICTERUS.

*Jaundice* is one of those symptoms which has been dignified by being described as a special disease. Essentially it merely signifies the peculiar discoloration of the skin and other structures which is observed when the bile-pigments accumulate in the blood.

ÆTIOLOGY AND PATHOLOGY.—Cases of jaundice have long been divided into:—1. Those in which there is a mechanical obstruction preventing the escape of the bile through the ducts. 2. Those in which no such obstruction exists.

1. **Jaundice from Obstruction.**—This may be due to:—(i.) *Impaction of some foreign material* in the hepatic or common bile-duct, namely, a gall-stone; thickened or gritty bile; mucus; rarely parasites, either formed in the liver or its duct (*distoma hepaticum*\* and *hydatids*), or having entered from the intestines (*round-worm*); very rarely fruit-

\* The *distoma hepaticum* or liver-fluke is a small trematode worm, often found in sheep, very rarely in the human being, occupying either the gall-bladder or bile-ducts. It is of a flattened, elongated, oval form; soft; and brownish or yellowish in colour.

stones or other bodies which have passed into the duct from the bowel. (ii.) *Catarrh of the mucous membrane* of the duct, or of the duodenum about the orifice, causing narrowing of its canal. (iii.) *Organic changes* in the walls of the duct or at the orifice, leading to more or less *stricture*, or even to complete *obliteration*, namely, congenital constriction or closure; thickening of the walls from inflammatory changes; perihepatitis; or cicatrization of an ulcer, either in the duct or the duodenum. (iv.) *Pressure* upon the duct, *invasion of its canal*, or *closure of its opening*, by tumours or growths, especially by projecting growths from the liver; enlarged glands in the portal fissure; and pancreatic disease implicating the duodenum: rarely by a pyloric tumour; growths in or behind the peritoneum; hepatic aneurism; faecal accumulation in the colon; uterine and ovarian enlargements, including pregnancy; or a renal tumour or displaced kidney. (v.) *Spasm or paralysis* of the muscular coat of the duct (?).

Physiologists differ in their views as to the mode in which the bile is secreted, and this influences the opinions held as to the pathology of *obstructive jaundice*. It is generally maintained that both the bile-acids and bile-pigments are formed in the liver; some physiologists believe, however, that the latter are generated either partly or entirely in the blood, and merely separated by the liver. Hence arise the two theories;—1. That the discoloration of jaundice is due to *excessive absorption* of the bile by the veins and lymphatics after its formation. 2. That it results from *suppression of its secretion*, and the consequent retention of the pigment in the blood. The former is probably the correct view, and the intensity of the jaundice will be in proportion to the rapidity with which the secretion of bile is going on, and to the slowness of its decomposition in the blood. Absorption of biliary secretion is always proceeding in the normal state, but the bile thus taken up is speedily changed in the process of nutrition.

**2. Jaundice without Obstruction.**—The conditions under which this variety is supposed to occur are:—(i.) In certain *specific fevers*, namely, yellow, remittent, intermittent, and relapsing fevers; very rarely in typhus, typhoid, or scarlatina. (ii.) When certain *poisons* are present in the blood, especially in connection with pyæmia; [snake-bites; poisoning by phosphorus, mercury, copper, or antimony; and after the inhalation of chloroform or ether. (iii.) In *acute or chronic atrophy* of the liver; or after *destruction of its tissue* from any cause. (iv.) In *congestion* of the liver. (v.) From *disturbed innervation*, especially after sudden intense mental emotion. (vi.) When the blood is *insufficiently aerated*, as in cases of pneumonia, in new-born infants, or as the result of overcrowding and bad ventilation. (vii.) Where bile is formed in *great excess*. (viii.) In cases of *habitual or long-continued constipation*. (ix.) In certain states of the *portal system of veins*, such as when they contain abundant pigment granules; or when they are unusually empty after profuse hæmorrhage from the alimentary canal. (x.) As an *epidemic* (?).

Before mentioning the various explanations which have been given of the occurrence of jaundice under the circumstances just enumerated, I cannot but express my agreement with those who consider that in many of these cases it results from obstruction, at all events to some degree, which obstruction may arise from pressure upon the smaller ducts within the liver; from catarrh of the main ducts; or from the formation of plugs of mucus. The views suggested by different writers

as to the pathology of the various forms of *non-obstructive jaundice* are that it is dependent upon:—1. *Suppression of secretion*. 2. *Increased absorption*, so that more bile enters the blood than can undergo decomposition, either from excessive secretion; undue retention of bile in the intestines, owing to constipation; or diminution of pressure in the portal vessels. 3. *Impaired and delayed metamorphosis* of the bile-elements in the blood, some being of opinion that the bile-acids are converted in this fluid into bile-pigments, owing to imperfect oxidation. 4. *Conversion of the hæmatine of the blood into bile-pigments*. With regard to the influence of nervous disturbance in producing jaundice, it is presumed that this may affect the activity of the secretion; the state of the portal veins; or the rapidity of the changes in the blood.

Jaundice is by no means a necessary accompaniment of even grave organic disease of the liver itself, and, in such cases, when it is marked, it is generally owing to some projection from this organ interfering with the main duct; or to the glands in the portal fissure being involved. It may, however, be due to destruction of the hepatic tissue; or to the ducts or the portal branches in the substance of the liver being interfered with.

**ANATOMICAL CHARACTERS.**—In marked jaundice not only are the skin and conjunctivæ more or less stained with bile-pigment, but likewise most of the tissues, organs, and fluids of the body, as well as morbid exudations and effusions. In the skin the pigment accumulates chiefly in the rete mucosum, at the same time involving the sweat-glands considerably. The nerve-tissues are but slightly affected; and the mucous membranes generally, with their secretion, still less. The bile-pigments are found in the clot and serum of the blood, but not the bile-acids; in prolonged cases coagulation is imperfect, and the corpuscles are altered in their characters; while not uncommonly extravasations of blood are found. In cases of obstructive jaundice the liver itself becomes at first enlarged uniformly, without any alteration in shape, and mottled of a more or less deep yellow tint, or in some cases being olive-green; its ducts are distended; and in time numerous particles of pigment collect in the hepatic cells. Should the obstruction implicate the common duct, the gall-bladder will be distended. If the obstruction is persistent, the liver undergoes degeneration, becoming atrophied, very dark or sometimes almost black, and softened, many of its cells being destroyed, leaving only a granular detritus, which is visible under the microscope. The kidneys also are much changed in prolonged cases, being deeply coloured, their tubules containing a black or brown deposit, and their secreting cells presenting granules of pigment, or ultimately breaking down and undergoing destruction.

**SYMPTOMS.**—The most obvious clinical phenomena associated with jaundice are those derived from the *external discoloration*; the characters of the *urine*; and the consequences of *absence of bile from the alimentary canal*. Usually the earliest signs are afforded by the urine; next by the conjunctivæ; and lastly by the skin. The conjunctivæ are more or less deeply tinged of a yellow colour. The skin may present a variety of tints, from a faint yellow to a brownish- or blackish-green. The colour is deepest where the epidermis is thin, and varies with age, complexion, amount of fat, and other circumstances. If the lips or gums are pressed, so as to expel the blood, a yellowish hue is often observed. The urine exhibits a colour ranging from a light saffron-yellow to one resembling mahogany or porter; on standing it usually becomes



greenish. Its froth is yellow, and it will tinge white linen or blotting-paper dipped into it, often staining the under-clothing. Chemical examination is most important, as indicating the presence of *bile-pigments*; and, as many believe, of *bile-acids*. The former are tested for by nitric acid; the latter by sulphuric acid and sugar. (See EXAMINATION OF URINE.) Dr. George Harley and others affirm that the bile-acids are only present in the urine in cases of *obstructive* jaundice, and not in that due to *suppression*; also that they may disappear in prolonged cases of the former, owing to destruction of the liver-tissue. Another important character presented by the urine is that it often contains *leucine* and *tyrosine*, crystals of which may be seen under the microscope, after careful evaporation of some of the excretion to a syrupy consistence. In some cases the quantity of urine is below the normal at first; the reaction is acid; the proportion of urea and uric acid varies, and these constituents may be in excess. In prolonged cases sugar sometimes appears. Renal epithelium or casts tinged with biliary colouring matters are occasionally observed.

When the bile does not reach the intestines, the consequences are constipation, with unhealthy stools, these being deficient in colouring matters, often pale drab or clay-coloured, dry, and offensive, also containing excess of fat; and the formation of much foul gas from decomposition of the intestinal contents, with consequent flatulence and the passage of foetid flatus. Occasionally diarrhœa is observed from time to time; or dysenteric symptoms set in. There is usually a disinclination for food, especially for fatty matters; and eructations are common, which may have a bitter taste.

Evidence is often afforded of the presence of bile in the sweat, milk, saliva, and tears. From the accumulation of bile-acids in the blood result not uncommonly cutaneous itching, which may be very distressing; slowness of the heart's action and pulse, which may fall to 50, 40, 30, or even 20 per minute; and a feeling of languor, depression, debility, lowness of spirits, incapacity for exertion, irritability, and drowsiness. The symptoms last-mentioned are also partly due to the wasting and impaired nutrition which usually soon become apparent, in prolonged cases being very marked. In some instances urticaria, lichen, boils, carbuncles, or petechiæ are observed. Yellow vision (*xanthops*y) is an extremely rare phenomenon, and its cause is very uncertain.

Jaundice, especially the non-obstructive variety, may be accompanied with typhoid symptoms; with low nervous phenomena; or with dangerous hæmorrhages, particularly from the stomach and bowels, ending speedily in death. It is important to observe that these phenomena are often quite out of proportion to the intensity of the jaundice. They have been attributed to the accumulation in the blood of bile-acids, of cholesterin, of products resulting from the decomposition of bile-acids or their formative elements, or of some noxious substance formed in the cells of the liver; or to the metamorphosis of materials in the process of preparation for excretion by the urine being checked or modified, owing to a deficiency of bile, which is required for these changes, so that, instead of urea and such compounds, intermediate products are formed, which collect in the blood and act as poisons. Might it not be that, in some of these cases at all events, the above symptoms are independent of the jaundice, and result either from some general morbid state, or from some condition of the kidneys which leads to blood-poisoning?

*Physical examination* will probably in cases of obstructive jaundice reveal in a short time enlargement of the liver, not great, and quite regular in outline. If the common duct is obstructed, the gall-bladder may also present a fluctuating enlargement. In prolonged cases the liver may ultimately afford the physical signs characteristic of atrophy.

The *course, duration, and intensity* of jaundice vary greatly according to its cause. It may be merely a slight temporary disturbance; or permanent and extreme in degree.

**DIAGNOSIS.**—The first matter in diagnosis is to be certain that jaundice is present. The discoloration of the skin might possibly be simulated by a chlorotic tint; by the cachexia of chronic lead-poisoning, malaria, or cancer; by the colour associated with supra-renal disease; or by bronzing from exposure to the sun. The conjunctivæ and urine should always be particularly examined. It must be remembered, however, that the yellowness due to the collection of fat under the conjunctiva may be mistaken for that of jaundice. Also pigments sometimes form in the urine, which render it very dark; and now and then malingerers purposely stain the skin, and add colouring matters to the urine.

It may be difficult to determine whether jaundice is of the *obstructive* or *non-obstructive* variety, but the latter, as well as its particular cause, may be generally recognized:—1. By the circumstances under which it occurs; and the collateral symptoms. 2. By the discoloration being less marked. 3. By the presence of more or less bile in the stools. 4. By examination of the urine, which, according to Dr. G. Harley, gives indications of the presence of bile-acids only in the obstructive form of jaundice, though many observers deny this; and which in the non-obstructive variety yields leucine and tyrosine.

The precise cause of *obstructive* jaundice is determined by:—1. The age, sex, habits, and general past history of the patient. 2. The preceding and accompanying symptoms, both local and general. 3. The rapidity with which the jaundice has set in; and its intensity. 4. Careful *physical examination* of the abdomen. 5. The course and progress of the case; and the effects of treatment. Intelligent attention to these points will generally lead to a correct opinion. The more rare causes can only be made out by exclusion, and are often merely guessed at. The fact of the gall-bladder being enlarged or not will as a rule show whether any obstruction involves the hepatic or the common bile-duct.

**PROGNOSIS.**—In most cases the prognosis of jaundice depends rather upon the morbid condition with which it is associated than upon this particular symptom. As a rule, therefore, it may be stated that *non-obstructive* jaundice is much the more grave. Typhoid and low nervous symptoms are highly dangerous; as are also hæmorrhages, and signs of interference with the renal secretion. In *obstructive* cases not only will the prognosis vary with the cause of the jaundice, but likewise with the rapidity with which it comes on; its intensity; and its mode of progress. In every case a cautious prognosis should be given, as it is never certain how matters may turn out, and this is particularly true when jaundice sets in rapidly, and becomes speedily intense. Catarrhal jaundice generally soon disappears. Of course when this symptom is due to obstructive organic disease, especially cancer, there is but little hope of its removal; but it is astonishing to what an extreme degree the discoloration may attain in some instances, without any proportionate general disturbance to support the idea that bile

acts as a poison. Jaundice in connection with pregnancy is considered highly dangerous.

TREATMENT.—In general terms the management of cases of jaundice may be summed up thus:—1. To treat the condition upon which it depends; and to remove any obstruction to the flow of bile, if practicable. 2. To promote secretion of bile, if necessary, by remedies to be hereafter considered; or, on the other hand, to limit its formation. 3. To attend carefully to the diet, especially avoiding fatty and oily substances, as well as much starch, sugar, or alcoholic stimulants. 4. To treat the symptoms due to the absence of bile from the alimentary canal, especially constipation and flatulence; or to supply a substitute for this secretion, in the way of artificially prepared inspissated ox-gall, gr. v-x, given two or three hours after meals. 5. To promote the renal and cutaneous excretions. 6. To attend to the general condition, giving quinine, iron, and other *tonics*, as well as adopting hygienic measures for improving the health in chronic cases; treating adynamic symptoms by *stimulants*; low nervous symptoms by encouraging free elimination by the bowels, kidneys, and skin; and checking hæmorrhages by *astringents*. In cases of permanent obstruction it has been proposed to make an artificial fistula into the gall-bladder, having first excited adhesion with the abdominal wall by means of escharotics. The irritation of the skin may demand measures for its relief; alkalies with opiates or morphia internally, or the latter administered hypodermically, and warm or alkaline baths are most serviceable for this purpose. It must not be forgotten that the colour of jaundice remains for a time after any cause of obstruction has been removed; and if this has been effected it is not necessary to continue further active measures. The removal of the bile from the system may be promoted by occasional *alkaline baths*, *aperients*, Cheltenham and other *mineral waters*; while convalescence is hastened by attention to hygienic and other measures which tend to improve the general health.

---

## CHAPTER XLV.

### CONGESTION OR HYPERÆMIA OF THE LIVER.

ÆTIOLOGY.—*Active* congestion of the liver occurs to some degree during every period of digestion. As a morbid condition it is met with:—1. In consequence of *errors in diet*, particularly in those that take but little exercise, especially habitual excess in eating, or indulgence in too rich articles of food; and abuse of alcohol or hot condiments. 2. As the result of continued *exposure to excessive heat* in tropical climates; or of a *sudden chill* while heated. 3. In connection with malarial, yellow, relapsing, and other *fevers*. 4. *Vicarious* of menstruation; or, it is said, of habitual discharges, such as bleeding from piles. 5. As the result of *local injury*. 6. Associated with *morbid deposits* in the liver. 7. In the early stage of *inflammation*.

*Mechanical* congestion affecting the liver is generally due to some condition of the heart or lungs which interferes with the general venous



circulation; very rarely to local obstruction of the hepatic vein or inferior vena cava.

*Passive* congestion is said to follow habitual constipation; or to result from a torpid state of the portal system.

**ANATOMICAL CHARACTERS.**—It is only the *mechanical* form of congestion of the liver that is generally seen after death. The organ is enlarged more or less, quite uniformly, its surface being smooth, and its capsule stretched. It often feels unusually firm. On section an excessive quantity of blood flows; the colour of the surface is unusually dark, sometimes even purple; and the vessels appear abnormally filled, becoming in time permanently dilated. The dark colour is rarely uniform, but is evident chiefly in connection with the infralobular branches of the hepatic veins, constituting the so-called *hepatic* congestion. *Portal* congestion is the term employed when the vessels at the circumference of, and between the lobules, are most distended, but this is rarely seen. The ultimate effects of long-continued mechanical congestion will be described in a subsequent chapter, but allusion may be made here to what is termed the *nutmeg liver*. This morbid condition is thus named because a section of the organ presents a variegated appearance, resembling that of a nutmeg, there being a mixture of red, white, and yellow tints. It is observed after congestion from cardiac obstruction has lasted for some time, and depends essentially on the following pathological changes. The branches of the hepatic vein are distended and over-loaded, of a deep red colour, and well-defined; the circumference of the lobules corresponding to the portal branches is anæmic, and has undergone degenerative fatty changes, being consequently pale and opaque; while the bile is stagnant in many of the smaller bile-ducts, which accounts for the yellow tint.

**SYMPTOMS.**—Locally hepatic congestion tends to produce a sense of uncomfortable tension, fulness, and weight over the liver, especially after meals, and when the patient lies on the left side; sometimes there is slight tenderness. There may be pain in the right shoulder. Slight jaundice is often present, but the stools contain bile. The spleen becomes enlarged in course of time in cases of mechanical congestion. Commonly the alimentary canal is deranged, as evidenced by impaired appetite, thickly furred tongue, constipation or diarrhœa, and flatulence; but these symptoms are often the result of the same cause which originates the hepatic congestion, though they may be partly due to deficiency or unhealthy quality of the bile. Some degree of general disturbance often accompanies congestion of the liver. The urine is frequently deficient and concentrated, depositing abundant urates; it also commonly contains biliary pigments.

The *physical signs* of congested liver are moderate enlargement, liable to some variation; regularity and uniformity in shape, as well as over the surface and margins; with frequently somewhat increased firmness of the organ.

**TREATMENT.**—For *active hepatic congestion* the measures to be adopted are to remove its exciting cause, an *emetic* being useful if it is due to irritating articles of food; to restrict the diet to small quantities of beef-tea, milk, and such articles; to apply warm poultices, fomentations, or sinapisms over the hepatic region, or to dry-cup freely, or in some cases even to remove a little blood by leeching or cupping, or by applying a few leeches around the anus; and to give a dose of calomel or blue pill, followed by a *saline aperient*, such as citrate of magnesia, sulphate with

carbonate of magnesia, sulphate of soda, or cream of tartar. After the acute symptoms have subsided alkalies with bitter infusions are useful, as well as *alkaline* and *saline mineral waters*; subsequently the principles of treatment must be similar to those to be described hereafter as applicable to chronic hepatic diseases. These must also be followed in the management of cases of *mechanical congestion*.

---

## CHAPTER XLVI.

### ACUTE INFLAMMATORY DISEASES OF THE LIVER.

#### I. CIRCUMSCRIBED OR SUPPURATIVE INFLAMMATION—HEPATIC ABSCESS.

**ÆTIOLOGY AND PATHOLOGY.**—The usual form of acute inflammation of the liver-tissue is that which ends in suppuration, and even this is rare, except in tropical climates. The cases of hepatic abscess met with in this country occur principally among sailors and others who have come from these regions. Murchison distinguished two forms of hepatic abscess, the *tropical* and the *pyæmic*, the latter occurring in temperate climates.

The causes of acute hepatic inflammation may be stated generally as follows:—1. Occasionally *direct injury* to the liver, or over the hepatic region. 2. *Convection of septic matters* from various parts of the body, either internal or external, the products of wounds, operations, abscesses, ulceration, or gangrene. The deleterious substances may come from any part, but hepatic abscess is especially liable to occur after ulceration or gangrene of the stomach or bowels; as the result of operations affecting the alimentary canal; and in connection with ulceration or suppurative inflammation associated with the bile-ducts or gall-bladder; because under such circumstances the materials are immediately taken up by the portal system of vessels. Some authorities are of opinion that in these cases the disease originates in phlebitis, extending along the portal vessels to the liver. 3. Occasionally the softening and breaking-down of an *embolus* or *thrombus* in the *portal vein*—*suppurative pyle-phlebitis*—the particles being conveyed into the liver, and originating an abscess there. 4. Rarely some *direct irritation* in the substance of the liver, for example, a suppurating hydatid-cyst; gall-stones; round-worms which have entered through the bile-duct; or foreign bodies. 5. The ætiology of *tropical abscess* requires special comment. It has been regarded by Budd and others as in all cases essentially *pyæmic*, resulting from previous dysentery. In some instances it is highly probable that this is the true pathology, but by no means in all, for often no sign whatever of dysentery can be discovered. There are two views as to the *exciting causes* of the hepatic inflammation in such cases:—*a.* That it is the direct consequence of *continued intense heat*, combined with *malarial influence*. *b.* That in addition to these influences, which induce a *predisposing* depraved condition of the system, there must be a *sudden chill* acting upon the body. Intemperance, excessive eating, and indolent and luxurious habits generally, act as powerful *predisposing causes* of hepatic abscess.

**ANATOMICAL CHARACTERS.**—The *post-mortem* examination in cases of acute hepatitis generally reveals that suppuration has been established. The disease is supposed to commence with active hyperæmia; followed by effusion of lymph and degeneration of the hepatic cells, causing the affected part to become swollen or prominent, paler, yellowish, and softened; then suppuration begins in points in the centre of the lobules, which gradually coalesce, forming abscesses of various sizes. The pus-cells are probably partly leucocytes; partly the products of endogenous multiplication of the liver-cells. The situation, number, size, and exact shape of the hepatic abscesses vary widely; as well as the nature and amount of their contents; and the condition of the surrounding tissue. The *right* lobe is much more frequently affected than the left. Important differences as to number and size are stated to characterize *tropical* and *pyæmic* abscesses respectively. In the former variety there is generally one large abscess, and rarely does the number exceed three; in the latter the separate accumulations of pus are very numerous and small, not often exceeding a hen's egg in size. My own limited experience of hepatic abscess would lead me to the conclusion that there are at least exceptions to this rule.

Originally hepatic abscesses are more or less rounded, but by coalescence and extension they often become very irregular. The contents generally resemble healthy pus at first, but in time they may become sanguineous, or altered by admixture of bile, or more or less foetid and decomposed. At first the walls consist of liver-tissue, usually congested or infiltrated, softened, and ragged; subsequently the boundary may become converted into a smooth firm capsule.

The progress and termination of these abscesses are also variable. When large, and especially when of the tropical variety, they advance towards the surface of the liver, finally bursting, either externally, or into the peritoneum, intestines, stomach, gall-bladder, hepatic duct, hepatic or portal vein, inferior vena cava, or pelvis of the right kidney; or, in rare instances, opening through the diaphragm into the pleura, lung, or pericardium. After the discharge of the pus cicatrization may take place, causing contraction and depression of the surface of the liver. In some cases an abscess remains dormant for a considerable period, and then rapidly extends. It is also believed that the fluid portion of the pus may be absorbed, the contents becoming first caseous, then putty-like, and finally calcareous, the tissue around forming a dense cicatrix.

The gall-bladder is sometimes inflamed in cases of hepatic abscess. The bile contained in it is frequently unhealthy, but presents no special characters. The consequences of the rupture of an abscess into various structures are described in other parts of this work.

**SYMPTOMS.**—As a rule the symptoms of hepatic inflammation are very pronounced. They are both *local* and *general*.

*Local.*—Pain and tenderness are generally complained of over some part of the hepatic region, often preceded by mere uneasiness. The pain differs much in its severity and characters; in most cases it is at first dull, aching, and tense, but usually increases when suppuration sets in, and may then assume a throbbing character; it is more marked when the inflammation is near the surface. Sympathetic pains about the right shoulder and scapula are occasionally present, but, it is affirmed, only when the upper surface of the right lobe is affected. In this event also a deep breath or cough aggravates the pain; while the breathing is hurried, short, and chiefly upper costal, there being like-



wise some feeling of dyspnoea, with in many cases a short dry cough. Jaundice is very uncommon in connection with tropical abscess, but some degree of it is frequently observed in pyæmic cases. Ascites is extremely rare, unless the inflammation depends on pyle-phlebitis, when signs of great obstruction of the portal vein are prominent, and this is an important matter in diagnosis. More or less disturbance of the alimentary canal is almost always observed, such as loss of appetite, furred and irritable tongue, thirst, nausea or vomiting, constipation or diarrhoea. The urine is at first very markedly febrile; after suppuration has been established it often becomes pale, copious, and deficient in urea.

*Physical characters.*—The liver is at first uniformly and moderately enlarged. Should the abscesses formed be small and deeply seated, nothing further can be observed; but if one or more of them become large and superficial, the following characters are presented:—1. The general enlargement increases considerably, and in addition a *bulging prominence* presents in some direction, or occasionally more than one. This is generally observed in the epigastrium or right hypochondrium; sometimes it causes distension of the lower part of the chest, with flattening of the spaces. 2. The general surface and margins of the liver usually feel *smooth* and *regular*, but occasionally, from the projection of several small abscesses, or on account of peri-hepatitis, they are *undulated* and *irregular*. 3. The local bulging soon yields a sensation of *elasticity*, and afterwards of *fluctuation*, gradually extending and becoming more perceptible, surrounded often by a ring of inflammatory induration. No *hydatid-fremitus* can be felt. 4. The *hepatic dulness* is altered in outline as well as increased in *area*, and when the abscess tends towards the thorax, this is often one of the chief signs noticed. 5. *Auscultation* may reveal *friction-sound* over an abscess, due to peritonitis. It may also indicate encroachment upon the limits of the chest by the liver; and interference with the expansion of the right lung. 6. By means of the *aspirateur* pus may be obtained, and this is highly important as a means of diagnosis in doubtful cases. I may mention that marked *pulsation* may be observed in connection with an hepatic abscess presenting in the epigastrium, conducted from the aorta, and simulating an aneurism. The spleen may be enlarged, but this is chiefly observed in pyæmic cases, and is not usually the direct result of the hepatic disease.

*General.*—Chills or rigors often usher in an attack of acute hepatitis, followed by more or less pyrexia, with considerable constitutional disturbance. Suppuration is usually indicated by repeated rigors; fever of a hectic type, not uncommonly remittent or intermittent, attended with abundant sweats; and much prostration and wasting. Ultimately typhoid symptoms are very liable to arise, ending in low nervous disturbance and death. The constitutional symptoms are as a rule more severe in pyæmic than in tropical cases.

*COURSE AND TERMINATIONS.*—The ultimate course of events in hepatic abscess will depend upon the progress of the disease. The symptoms may subside, and the abscess undergo retrograde changes, ending in a cure. Almost always, however, it tends to open in one or other of the directions already mentioned, and the corresponding symptoms may be readily gathered from a little consideration. When it approaches the surface of the body the abscess causes redness, œdema, and the other signs of superficial suppuration before it bursts or is opened. Most cases of hepatic abscess prove fatal and are rapid in their progress, but

tropical cases may last for six months or more ; the pyæmic form is much the more fatal and speedy in its termination. Some cases go on for a long period, and ultimately recover, the abscess discharging its contents and cicatrizing.

## II. PERI-HEPATITIS.

This term is applied to inflammation of the *covering of the liver* and of *Glisson's capsule*, which is not uncommon as an acute affection, associated with peritonitis or organic diseases of the liver ; or resulting from injury, or from extension of inflammation from neighbouring parts. It is also said to arise from a chill. It leads to exudation, with consequent thickening, opacity, and adhesions ; while occasionally pus is formed.

**SYMPTOMS.**—The symptoms are pain over the liver, sometimes sharp, increased by cough and deep breathing ; with superficial tenderness ; but no particular derangement of the hepatic functions, or alterations in the physical characters of the liver. There is usually more or less pyrexia. If the affection is chronic, or if repeated attacks occur, as not uncommonly happens in connection with syphilis or chronic heart diseases, signs of obstruction of the portal vein or bile-duct, and of atrophy of the liver may be established.

## III. INFLAMMATION OF THE BILE-DUCTS.

*Catarrh of the bile-ducts* is by no means an uncommon affection, being especially met with in children, and in old gouty persons. Its chief causes are extension of catarrh from the duodenum ; hepatic congestion ; irritation of the mucous membrane by gall-stones, parasites, foreign bodies, and perhaps by unhealthy bile, which may cause considerable inflammation ; and blood-poisoning in connection with fevers and other affections. The morbid appearances are similar to those of other forms of catarrh. Occasionally croupous or diphtheritic inflammation is observed in the bile-ducts.

**SYMPTOMS.**—The symptoms of simple catarrh merely indicate more or less obstruction of the bile-duct, with consequent jaundice and enlargement of the liver and gall-bladder, this being generally preceded by signs of gastro-duodenal catarrh. There is often local pain and tenderness, with some degree of pyrexia. The duration and course of these cases vary, but generally they soon recover.

## IV. GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. **DIAGNOSIS.**—The occurrence of acute local symptoms connected with the liver, accompanied with constitutional disturbance, should lead to the suspicion of inflammation of this organ, especially in tropical climates, or if there is any obvious cause of pyæmia. At first there may be considerable difficulty in distinguishing inflammation from mere active congestion ; and also in separating the different kinds of inflammation from each other, especially *suppurative hepatitis* and *peri-hepatitis*. When an abscess forms, this is generally revealed by evident *physical signs* ; as well as by increased constitutional disturbance. Commonly, however, distinct objective indications of pyæmic abscesses are wanting. The differences between *pyæmic* and *tropical* abscess have already been

alluded to. The chief conditions which may be mistaken for abscess in the liver are inflammation and suppuration of the gall-bladder; a suppurating hydatid-cyst; and abscess in the abdominal parietes. Local peritonitis may simulate hepatitis.

2. PROGNOSIS.—In the milder forms of hepatic inflammation the prognosis is generally favourable, but when suppuration occurs it is very serious. It will then depend mainly on the size and probable number of the abscesses; the direction in which they open, Maclean stating as his experience that the largest number of recoveries follows bursting into the lung, and then into the intestine, and that the prognosis is much more favourable when the abscess points at the ensiform cartilage than in an intercostal space; the general condition of the patient; and whether the liver-affection is or is not associated with some other morbid state, such as dysentery. *Pyæmic* abscesses are very fatal.

3. TREATMENT.—The slighter forms of hepatitis may be treated in the same manner as active congestion. Much difference of opinion is held as to the management of *tropical abscess* in its early stage. The usual measures recommended are venesection, or local bleeding by leeches or cupping; constant poulticing or fomentations; the use of *saline purgatives*; and the administration of calomel. Dr. Maclean, who strongly opposes bleeding and mercury, advocates the free employment of ipecacuanha, as in dysentery. Tartar emetic and tincture of aconite have also been used. With regard to *pyæmic abscess*, there can be no question but that the severe lowering measures mentioned above are most injurious in this form of the disease. When suppuration occurs, poultices and fomentations must be assiduously applied. The question of *opening abscesses* connected with the liver is one which is also much discussed. Most authorities seem to be in favour of operating; some prefer leaving the abscess to take its own course, on account of the dangers of peritonitis, decomposition from entrance of air, hæmorrhage, or gangrene. If there is satisfactory evidence of the existence of a single abscess it appears to me certainly advisable to evacuate the pus, and even in doubtful cases the aspirateur may be advantageously employed. When there are several collections of pus, as in pyæmia, operative interference is contra-indicated. The different modes of evacuation advocated are by means of the *aspirateur* or a *small trochar and canula*; by *free incision*; or by the application of *caustic potash* so as to produce a slough, this being also used to excite adhesions to the abdominal wall. The air should be as carefully excluded as possible, and carbolic acid freely used. In the case of a moderate-sized abscess, it seems best to let out all the pus at once, and to leave a canula or drainage-tube in; when very large, it may be emptied gradually by successive operations. Large poultices should be afterwards applied, being very frequently changed, and *antiseptics* must be freely used, the patient lying as much as possible in that position most favourable for the escape of the pus. It is useful in some cases to wash out the abscess with weak carbolic acid. In the early period of the disease the diet should consist of milk, beef-tea, and such articles; when suppuration is set up it should be as nourishing as possible, while *stimulants* are called for at this time, as well as quinine, mineral acids, or tincture of steel. *Hypnotics* are often required; and various symptoms demand attention in many cases. The general treatment applicable to pyæmia is indicated in pyæmic cases.



## CHAPTER XLVII.

## ACUTE YELLOW ATROPHY.

**ÆTIOLOGY AND PATHOLOGY.**—The causation of this rare hepatic disease is very uncertain. Most cases occur in connection with pregnancy, but the complaint has also been attributed to severe nervous disturbance from depressing emotions; to blood-poisoning in cases of typhus, scarlatina, and other fevers; to malarial influence; or to the production within the body of some special poison, the product of faulty digestion or assimilation. Micrococci have been described by Dreschfeld as present in the early stage of acute yellow atrophy. The chief *pre-disposing causes* mentioned are age, the disease being almost always observed before 40, but never in childhood; the female sex; intemperance; venereal excesses; and syphilis.

As regards its *pathology*, most authorities consider acute atrophy as being the consequence of *diffuse parenchymatous inflammation of the liver*, excited by the action of some morbid poison. It has also been attributed to obstruction of the smaller bile-ducts; or to excessive formation of bile within them, whereby pressure is exercised on the surrounding structures.

**ANATOMICAL CHARACTERS.**—The obvious characters presented by the liver in acute atrophy are marked diminution in its size and weight; relaxation and softening of tissue; change in colour to a dull yellow; and disappearance of all traces of lobular divisions. The organ may be reduced to half its ordinary bulk, or even less, being especially diminished in thickness, and it lies out of sight at the back of the abdomen, shrunken and flaccid, while the peritoneum covering it is lax, and is often thrown into folds. In parts where the disease is less advanced, hyperæmia and a greyish exudation have been described. Microscopic examination reveals fatty degeneration and destruction of the gland-cells, until ultimately nothing remains but a granular detritus, oil-globules, and pigment. There is only a little mucus in the gall-bladder and bile-ducts as a rule. Extravasations of blood in the alimentary canal and other parts, with ecchymoses, are not uncommon. The spleen is generally enlarged. The kidneys exhibit degeneration of, and deposits of pigment in, the epithelium-cells. Leucine and tyrosine are found in the blood; as well as in the tissues of the liver, spleen, and kidneys.

**SYMPTOMS.**—There may or may not be *premonitory* symptoms indicative of gastro-enteric catarrh, or general uneasiness and painful sensations may be experienced, but there is nothing characteristic about these phenomena. Slight jaundice is usually soon observed, and afterwards increases, but seldom becomes intense, and it may be limited to the upper part of the body. It has been attributed to blocking-up of the smaller ducts by the *débris* of the cells. Among the ordinary symptoms of acute atrophy of the liver are pain and tenderness over the epigastrium and right hypochondrium, vomiting, and constipation. There is not much pyrexia, but the pulse is often hurried, and is liable to much

variation, while the temperature is considerably raised in some cases towards the close.

The most striking clinical phenomena in this disease, however, are :—  
1. Those significant of the *typhoid state*, with prominent nervous symptoms. 2. Great diminution or complete disappearance of the hepatic dulness. 3. Generally enlargement of the spleen. 4. Peculiar changes in the urine. 5. Hæmorrhages into various parts. The nervous symptoms consist at first of headache, great depression, languor, irritability, and restlessness; speedily followed by low delirium, stupor, coma, twitchings, and convulsions, with involuntary discharge of fæces and urine. At the same time the tongue becomes brown and dry, with sordes on the teeth. The urine yields considerable quantities of leucine and tyrosine; while urea, uric acid, and salts are much diminished, being sometimes almost entirely absent; some bile-pigment is usually present, and often a little albumin or blood. Hæmorrhage most frequently takes place into the stomach and bowels; cutaneous petechiæ and vibices are not uncommon; and in rare instances uterine hæmorrhage or epistaxis occurs. The *course* of acute atrophy of the liver is generally very rapid; and the *termination* almost invariably fatal. When the disease arises in the course of pregnancy it leads to miscarriage or abortion.

DIAGNOSIS.—At first it is difficult or impossible to diagnose acute atrophy of the liver, but once the symptoms are fully developed, and the physical signs indicative of diminution in the size of the organ become evident, the nature of the disease is plainly revealed.

PROGNOSIS is very grave, the disease almost always ending fatally.

TREATMENT.—Free purgation; promotion of the action of the skin by hot-air or vapour baths; the administration of *diuretics*; blistering and leeching the head; and the use of cold douches, have been the chief measures recommended in the treatment of acute atrophy of the liver, but they are of little service when the disease is established. Hæmorrhages and other symptoms must be treated as they arise.

---

## CHAPTER XLVIII.

### CHRONIC DISEASES OF THE LIVER.

#### I. HYPERTROPHY AND ATROPHY.

A SIMPLE *hypertrophy* of the hepatic tissue is said to be observed in some cases of leucocythæmia; very rarely in diabetes; and as the result of prolonged residence in hot climates. Clinically it is indicated by a slow, moderate, and uniform enlargement of the liver; without any evident symptoms, either local or general.

On the other hand, *atrophy* generally occurs in old age; or it may result from starvation; or from pressure upon the surface of the organ by tight stays, peritoneal adhesions, and other conditions.

## II. FATTY LIVER—HEPAR ADIPOSUM.

**ÆTIOLOGY.**—This affection belongs to the *fatty infiltrations*, the secreting cells of the liver becoming filled with oil. The conditions under which it is usually met with are:—1. In connection with phthisis and other *wasting diseases*, such as cancer, gastric ulcer, or chronic dysentery. 2. In *chronic lung and heart affections*, which lead to imperfect aëration of blood. 3. As the result of *over-feeding*, especially excessive consumption of hydro-carbonaceous substances; and *abuse of alcohol*, particularly in the form of ardent spirits. Deficient exercise and indolent habits aid greatly in the development of the disease under these circumstances. Some individuals are much more predisposed to fatty liver than others. *Fatty degeneration* of the hepatic cells may be set up in connection with other morbid conditions of the liver, such as albuminoid disease or cirrhosis.

**ANATOMICAL CHARACTERS.**—In well-marked fatty liver the morbid characters include enlargement and increase in weight, though the specific gravity is diminished, the margins of the organ being thickened and rounded, and the surface quite smooth; a more or less yellow colour, with opacity, both externally and on section, this being generally mottled with red; softening of the tissue, which has a doughy, inelastic feel, pits on pressure, and readily breaks down or tears; anæmia, but little blood escaping from the cut surface; loss of distinctness of outline of the lobules; and evidence of the presence of much fat, obtained either by the knife, by blotting-paper, or by ether. The liver may yield as much as from 43 to 45 per cent. of oily matters, which consist of olein and margarin, with traces of cholesterin. Microscopic examination shows enlargement of the cells, which also become spherical, and are more or less loaded with fat. In the less advanced cases the change is only revealed by the aid of the microscope. It is found that the morbid process extends from the circumference of the lobules towards their centre.

**SYMPTOMS.**—As a rule there are no evident symptoms connected with the liver in fatty disease. Dyspeptic disturbances are common. *Physical examination* is the only positive means of diagnosing fatty liver:—1. There is enlargement in a downward direction, slow in its progress, and usually moderate in degree, the organ never attaining any great size. 2. The shape is quite normal; and the surface and margins are smooth and regular, the latter feeling rounded. 3. Palpation often reveals a soft, doughy consistence of the liver. The *general* symptoms are frequently those associated with fatty changes, namely, want of tone; inaptitude for exertion; pallor and pastiness of the skin. Signs of fatty changes in other organs and tissues, such as the heart, vessels, and kidneys, may be observed.

## III. ALBUMINOID, LARDACEOUS, OR WAXY LIVER.

The *ætiology* and *pathology* of this morbid condition have already been considered. (See page 74.) The liver is one of the most common seats of albuminoid disease.

**ANATOMICAL CHARACTERS.**—Commonly the size and weight of the liver are considerably increased, as well as its specific gravity. The



shape is scarcely altered, but the organ is somewhat flattened, with rounded edges. The surface and margins are quite smooth; the peritoneum is stretched; and the tissue feels very firm and resistant. On section the usual pale, anæmic, dry, greyish, and glistening aspect characteristic of lardaceous disease is observed; often the surface is quite homogeneous, without any trace of lobules, or these may appear to be enlarged. The ordinary chemical tests characteristic of the albuminoid material are yielded; and microscopic examination reveals its presence in connection with the vessels and cells. It is first observed in the middle zone of the lobules, where the branches of the hepatic artery are distributed. The exact appearances may be modified by the association of other morbid conditions with the albuminoid disease, such as fatty degeneration, cirrhosis, or syphilitic cicatrices. Commonly other organs are involved at the same time.

**SYMPTOMS.**—As a rule hepatic symptoms are not prominent. Local sensations rarely amount to more than a feeling of weight, tension, and discomfort. Jaundice and signs of obstruction of the portal circulation are also very uncommon, and when present are due either to pressure by enlarged glands in the portal fissure, or by thickenings in connection with local inflammatory changes; or ascites may result from chronic peritonitis, or possibly from constitutional debility and anæmia. The *physical signs* of albuminoid disease are:—1. Enlargement, chiefly in a downward direction; gradual in its progress, the liver frequently attaining great dimensions at last, so that it presents a visible prominent tumour. 2. No perceptible alteration in form, the surface being smooth and uniform, with rounding of the margin. 3. Consistence dense and resistant, often extremely hard. There are the usual *general* symptoms indicative of albuminoid disease: with, in most cases, signs of implication of other organs; as well as of the existence of some constitutional condition with which the disease is associated.

#### IV. HYDATID TUMOUR OF THE LIVER—ECHINOCOCCUS HOMINIS— ACEPHALOCYST.\*

**ÆTIOLOGY AND PATHOLOGY.**—The best illustration of the morbid conditions resulting from the development of the embryo of a tape-worm in the human body is afforded by the complaint now under consideration; and though the parasite may be met with in almost every organ and tissue in the body, yet the liver is by far its most frequent seat, so that the subject may be discussed once for all in the present chapter. A *hydatid tumour* is derived from the development of embryos of the *tænia echinococcus*, each of which produces a *scolex*, named *echinococcus hominis*; and these scolices become enclosed in cysts. This variety of tape-worm infests dogs and wolves, and it is supposed that fragments are evacuated in their excreta, the ova of which are subsequently set free, become mixed with water or food, and are thus introduced into the alimentary canal of a human being. When the embryos are liberated, they bore the walls of the stomach with their hooks, and then migrate, usually settling in the liver, and there developing into scolices. The *echinococcus* also infests sheep, and it is in consequence of eating their

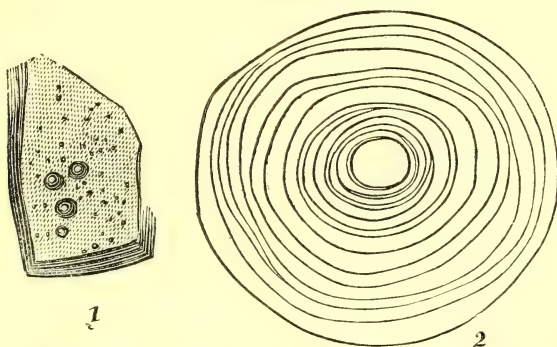
\* For a detailed account of the most important and interesting facts relating to hydatids, the reader is referred to the recent work on "Hydatid Disease," by Dr. I. Davies Thomas of Adelaide.

organs which are the seat of this parasite that dogs become the subjects of tape-worm.

Iceland is the country in which hydatid disease is especially prevalent. It is also very common in some parts of Australia. In this part of the world it is only very exceptionally met with, and usually in persons who have been abroad. Most cases occur during middle life; and among the poorer classes.

**ANATOMICAL CHARACTERS.**—In the first place it will be well to describe the various structures which ordinarily enter into the formation of a typical *hydatid tumour*:—1. Externally there is in the great majority of cases a firm, whitish or yellowish, fibrous, vascular capsule, derived from the connective tissue of the organ which harbours the hydatid, and due to irritation, which is adherent to the surrounding structures. This covering is not, however, an essential part of a hydatid. 2. Within it, moulded as it were to its interior, but easily separated, is a cyst or bladder, *mother-cyst, sac, or vesicle*, of very variable thickness, which increases with advancing age. This consists of two distinct structures.

FIG. 38.



Hydatid found in man. 1. A fragment of the natural size; at its edge are shown the layers of which it is composed; on the external surface are several hydatid germs of different periods of development. 2. One of the germs flattened and magnified forty times, showing the stratified layers.

(a) The outer portion (*ectocyst* of Huxley) is tough, elastic, greyish, semi-transparent or gelatinous in aspect, and compared to boiled white of egg. Under the microscope it is seen to consist of several concentric layers, a section presenting a characteristic laminated appearance; under the highest powers it presents a nearly hyaline, or at most a faintly granular appearance. (Fig. 38.) The most internal layer (*germinal* or *embryonic membrane, endocyst* of Huxley, *parenchymatous layer* of Leuckart), is extremely delicate; and has a cellular structure, with coarsely-granular, highly-refractive bodies, and calcareous corpuscles. The inner surface is said by Naunyn to be provided with cilia. 3. A quantity of fluid is contained within the cyst, usually completely filling it, perfectly colourless, transparent, and watery as a rule, occasionally slightly opalescent; of low specific gravity—1007 to 1015; generally neutral in reaction, but occasionally faintly alkaline or acid; and consisting mainly of a strong solution of chloride of sodium, without any, or only traces of albumin or other organic substance, but said to contain succinate of soda. 4. Floating in this fluid, or attached to the inner surface of the mother-cyst when small, are numerous *secondary* or

*daughter-cysts*; in some instances these amount to hundreds or thousands, and completely fill the space, so that there is little or no fluid, and they become flattened by mutual pressure; each daughter-cyst has precisely the same structure as the mother-sac. Within the larger of them there may be a third generation, and, rarely, a fourth is observed, —*grand-daughter-cysts*. 5. When the walls of the sacs are examined carefully, little whitish opaque spots are visible on the inner surface, which are the scolices of the *echinococcus* in various stages of development, usually arranged in groups or clusters, but occasionally single. These may also be free in the fluid, rendering it somewhat opaque. These echinococci are produced within delicate sacs, named *brood-capsules*, and they may be found enclosed in these capsules or free. Each scolex is very minute, measuring from  $\frac{1}{50}$  to  $\frac{1}{6}$  of a line in length, but the length and form vary, according as the head is retracted into the body or

FIG. 39.

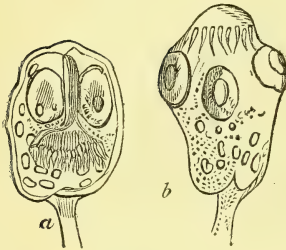


FIG. 40.

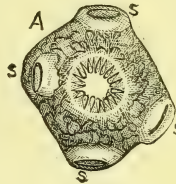


FIG. 41.

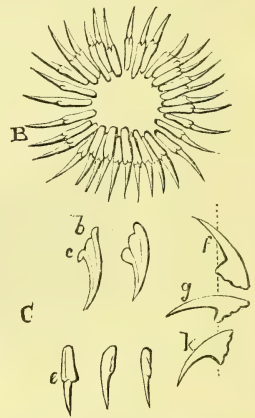


FIG. 39.—Two *Echinococci* from a hydatid tumour. The one (a) has the head retracted within vesicle; the other (b) has the head extruded.

FIG. 40.—(A.) An *Echinococcus* viewed transversely, the head being directed towards the observer s, s, suckorial discs.

FIG. 41.—(B) The circle of hooklets seen upon its under surface; thirty-four in number, seventeen long and seventeen short. (C) b, c, Lateral views of the separate hooklets—b, the base; c, the central extremity or bifid process of the base; e, hooklets viewed upon the concave or inferior border; f, g, k, a diagram illustrating the movements and position of the hooklets.

extruded. (Fig. 39.) The head (Fig. 40) presents a proboscis, four suckers, with a double circle of characteristic curved hooklets, which are movable and of unequal length (Fig. 41); a constriction separates it from the body, the latter being striated longitudinally and transversely, and presenting posteriorly a depression with a pedicle, by which the animal is fixed to the sac in its early condition. Numerous round and oval calcareous particles are imbedded in the tissue.

In most cases there is but a single tumour as described, but sometimes two or more are found, though one generally predominates over the others. The size varies extremely, and a hydatid growth may attain such dimensions as completely to fill the abdomen, and even to encroach upon the chest. The daughter-cysts usually range from a millet-seed to an egg in size, but subsequent generations are very minute. Originally the shape tends to be spherical. The right lobe of the liver is the most frequent seat of hydatid tumour, but it may occupy any part of the organ, being either deep in its substance or superficial.



If the hydatids are numerous, large, or superficial, they necessarily alter more or less the dimensions and form of the liver, giving rise to prominences. The surrounding hepatic tissue is often atrophied and compressed; sometimes the healthy portion becomes hypertrophied. Peritonitis may be excited over the tumour, giving rise to thickening and adhesions.

The events which are liable to happen in the course of hydatid disease are important, and may be summed up as follows:—1. The tumour enlarges, displacing adjoining structures and interfering with their functions, until it finally bursts in some direction, or is ruptured by violence, or in some other way. The opening may take place externally through the abdominal or lower thoracic wall; into either pleura or lung, especially the right, which is the most common direction; the pericardium rarely; the peritoneum; the stomach or intestines; the gall-bladder or the bile-ducts; the hepatic vein or inferior vena cava. 2. Inflammation and suppuration sometimes occur, either spontaneously from rapid growth; from injury or operation; or from the entrance of bile. 3. If the hydatid is slow in its progress, it not uncommonly undergoes degenerative processes as it advances in age, and these may ultimately bring about a spontaneous cure. The entrance of bile is supposed sometimes to induce this result. The outer capsule then becomes much thickened, firm, irregular, opaque, and ultimately calcified partially or completely. This impedes further growth, and the contained hydatids compress each other, shrivel and dry up, and finally die. The fluid also thickens and becomes opaque; and, in short, fatty and calcareous degeneration take place throughout, until there only remains a putty-like *débris*, in which shreds of the vesicles and hooklets of the echinococci are imbedded, revealing the nature of the mass. Hæmatoidine crystals are often found in it, as well as usually much cholesterin. A cicatrix-like depression may finally be left. 4. Occasionally cysts are found in which there are no echinococci. The name *acephalocyst* has been applied to this condition, and it has been regarded as an abortive or sterile form of the parasite, in which development is arrested; or as an earlier stage of its growth.

Allusion may be briefly made here to a very rare form of this disease, named *multilocular hydatid-cyst*. The liver is found occupied by a mass, in some cases as large as a child's head or even larger, consisting of a stroma of cellular tissue, usually altered considerably by fatty degeneration, in which are imbedded cells or alveoli of various sizes, enclosing a gelatinous substance, in which microscopic examination reveals fragments of the laminated membrane of hydatids, hooklets, or occasionally even perfect scolices, as well as abundant calcareous particles. The centre of this mass is very liable to undergo suppuration, thus altering its characters considerably. This arrangement of the tumour has been attributed to the embryos having been deposited in the lymphatics, blood-vessels, or ducts of the liver; or to the absence or early rupture of the external fibrous cyst, so that the parasites are able to grow and migrate in various directions, and may thus enter the different vessels.

Other organs and tissues are not uncommonly the seat of hydatid tumour along with the liver.

**SYMPTOMS.**—In general terms the ordinary clinical history of hydatid tumour of the liver may be summed up in the absence of morbid

sensations referable to this organ, of any interference with its functions, or of constitutional disturbance; while the liver presents a peculiar form of enlargement. The disease may be latent from first to last. Should the growth attain a great size, a sense of local fulness and tension is often felt; and in rare cases jaundice or signs of portal obstruction arise, in consequence of pressure upon the bile-ducts or portal vein, or because these vessels become blocked up by hydatids. Surrounding structures may also be interfered with, especially the diaphragm and respiratory organs. Should the cyst rupture, the consequent symptoms depend on the direction in which this lesion takes place, being in many instances very grave. If the opening is external or into the lungs, characteristic structures may be discharged. The occurrence of suppuration is indicated by the ordinary local and constitutional signs of hepatic abscess.

The *physical signs* of hydatid tumour demand particular attention.

1. The liver is increased in size, and this is generally the first thing which attracts notice. The growth is, as a rule, very chronic and imperceptible in its progress, but finally the tumour may attain enormous dimensions, so as to give rise to a general enlargement of the abdomen, or it may encroach upon the chest, causing the right side to bulge.
2. The form of the liver is altered, as evidenced by palpation and percussion; while there is often an evident tumour in some part, especially the epigastrium or right hypochondrium. Smaller prominences are sometimes felt along the margins or surface of the organ, causing lobulation and irregularity.
3. Any prominent hydatid tumour generally feels quite smooth, and more or less elastic or fluctuating.
4. Hydatid-fremitus is often elicited very clearly.
5. In any doubtful case it is justifiable to make an exploratory puncture with the aspirateur, and thus to remove some of the fluid, the physical and chemical characters of which are quite characteristic. Perhaps some of the microscopic structures might come away at the same time.

It must be remarked that the signs above described are modified considerably by the degenerative and other changes which are liable to occur in a hydatid-cyst. The outer wall may feel hard and bony. If a case only comes under observation when the abdomen presents a general enlargement, it is by no means easy in many instances, except by the history, to make out where the growth originated.

The *multilocular cyst* is said to be distinguished by being nodulated, hard, and tender; by jaundice, ascites, and enlargement of the spleen being usually present; and by the tendency in the tumour to inflame and suppurate. This variety may run a very rapid course.

## V. CANCEROUS AND OTHER GROWTHS.

**ÆTIOLOGY.**—The liver is one of the most frequent seats of internal cancer, which may be either *primary* or *secondary*, the latter especially occurring after cancer of the stomach. The disease may originate by extension from neighbouring structures. It has sometimes been attributed to injury. Most cases are met with between 50 and 70 years of age, the affection being extremely rare before adult life. In some patients there is a hereditary taint. Dr. Walshe states that hepatic cancer is more common among males, and such has been my own experience.

ANATOMICAL CHARACTERS.—Ordinarily hepatic cancer assumes the form of distinct *nodules* or *tuberous masses*, having characters intermediate between those of scirrhus and encephaloid, approaching more towards one variety or the other in different instances. There is a wide difference as to size and number, the nodules being small at first, and gradually enlarging until they may ultimately reach the dimensions of a child's head, or even attain a larger size than this. Commonly several are found, unequal in size, and by their coalescence considerable tracts of the organ are sometimes involved. Originally the shape is spherical, but when the masses reach the surface they become flattened or even depressed in the centre, so as to present shallow concavities or *umbilications*. As a rule they are not separated from the surrounding tissue by any definite structure, but occasionally a distinct cyst exists around a cancerous mass. Generally the consistence is moderately firm, but it may range from that of a soft, brain-like, semi-fluctuating substance to that of a hard, cartilaginous tissue, and the amount of cancer-juice which can be expressed will vary accordingly. The colour of a section is in most cases white or yellowish-white, but more or less dotted and streaked with red, on account of the vessels present; it may, however, be extremely vascular and dark-red, resembling "*fungus hæmatodes*." The proportion of cancer-cells and fibrous stroma in any mass, as observed under the microscope, depends on the variety to which it belongs.

The liver is usually enlarged in proportion to the number and size of the growths, being often extremely large and heavy, as well as irregular in shape. Its tissues are more or less destroyed and compressed; the vessels and ducts are encroached upon or obliterated; and as a consequence jaundice and signs of obstructed circulation are often present. Sometimes thrombosis occurs in the portal branches or trunk. New vessels are developed, originating in the hepatic artery. Some observers describe the cancer as beginning in the centre of the lobules; others in the interlobular tissue. When a mass reaches the surface it excites localized peritonitis, with consequent thickening and adhesions. Neighbouring tissues may be involved by extension; and the lymphatic glands in the portal fissure are often implicated.

The growth of hepatic cancer is sometimes extremely rapid, especially when it is of the soft variety. Certain changes are liable to occur. The vessels of encephaloid cancer often give way, leading to extravasations of blood into its interior, which afterwards undergo changes, thus giving rise to unusual appearances. Very soft accumulations have been known to burst into the peritoneum in rare instances. Degenerative changes frequently arise in the less rapid forms, in the way of caseation; or of atrophy with contraction, induration, and the formation of a firm cicatrix. A section frequently presents a reticulated appearance, owing to fatty degeneration having taken place.

In exceptional cases hepatic cancer is *infiltrated*, and the organ may be smaller than usual, as well as diminished in weight.

Melanosis, cystic cancer, epithelioma, and colloid have been extremely rarely met with in the liver; but cylindrical-celled epithelioma is said to be more common. More recent and careful observation has shown that some of the morbid growths generally regarded as being of a cancerous nature, are composed of spindle-celled sarcoma; other forms of sarcoma may also occur, as well as myxoma, lymphadenoma, and other growths. Cysts derived from obstructed bile-ducts, and erectile tumours have also been described in the liver.



**SYMPTOMS.**—Hepatic cancer is in the great majority of cases characterized by marked local disturbances, but the disease may be latent. At first merely a sense of discomfort and weight is experienced, soon, however, increasing to distinct pain and tenderness, which local sensations frequently become very severe, especially if the growth of the cancer is rapid, or if peritonitis is excited. The pain is often lancinating, shooting either towards the back or shoulders, or over the abdomen. Jaundice and ascites are also common symptoms, being usually the result of obstruction of the main ducts and vessels, in consequence of pressure exerted upon them by projections from the liver, or by glands in the portal fissure; ascites may, moreover, be associated with chronic peritonitis. Once jaundice sets in, it is usually persistent, and often becomes intense, but it may be temporary from catarrh of the ducts. The spleen is but rarely enlarged. The superficial abdominal veins are sometimes distended. Sir William Jenner has drawn attention to the association of cancerous nodules about the umbilicus with hepatic cancer.

The *physical characters* of the liver indicative of cancer are :—1. Enlargement, frequently very great, rapid in its progress, and chiefly increasing in a downward direction. 2. Alteration in shape and irregularity of outline, nodules or larger masses being felt or sometimes even seen along the surface and margins, which nodules are not uncommonly umbilicated. 3. As a rule considerable firmness and resistance of the projections, though they occasionally have a soft elastic feel, or even yield a sensation of obscure fluctuation. 4. Occasionally friction-fremitus and sound during breathing, these signs being chiefly due to peritonitis.

Digestive derangements are present in most cases of hepatic cancer, and they frequently first attract attention. The cancerous cachexia is usually well-marked, being accompanied with rapid wasting, debility, and anæmia. There may be pyrexia from time to time, which is sometimes considerable when the progress of the disease is rapid. The urine may contain excess of indican. Cancer is frequently present in other organs, either as a primary or secondary formation, especially in connection with the alimentary canal.

The *progress* of hepatic cancer is generally very rapid, and the disease is rarely prolonged beyond a year.

Other forms of tumour in the liver generally give rise to no definite symptoms, but they might possibly be detected on physical examination, or might give rise to local symptoms due to pressure.

## VI. CIRRHOSIS OF THE LIVER.

**ÆTIOLOGY AND PATHOLOGY.**—Undoubtedly several distinct morbid conditions of the liver have been included under the term *cirrhosis*, which have totally different modes of origin. Moreover, two distinct forms of true cirrhosis are now recognized, namely, the *atrophic* and the *hypertrophic* varieties. *Atrophic cirrhosis*, which is the common form, is usually considered as resulting from a *chronic interstitial inflammation*, extending into the minutest portal canals, and leading to proliferation of cellular tissue between the lobules; or, as some pathologists describe, to the formation of an exudation, which undergoes organization and then contracts, with consequent pressure upon and obliteration of the vessels, and atrophy of the secreting elements. Some authorities have attributed

cirrhosis to a *constitutional diathesis*, characterized by the formation of a fibroid tissue in different organs and structures of the body, of which the morbid change in the liver constitutes but a local development; while still others have regarded the disease as commencing in *degeneration and destruction of the secreting cells*, the ducts, vessels, and areolar tissue remaining, this process being followed or not by proliferation of the last-mentioned tissue. The important *exciting cause* of cirrhosis is *abuse of alcohol*, and especially indulgence in ardent spirits on an empty stomach—hence the common name *gin-drinker's liver*. The alcohol being absorbed, and afterwards circulating through the liver, is believed either to set up inflammation, or to lead to degeneration of the cells, according to the particular view held as to the pathology of the disease. Cirrhosis, however, is certainly occasionally met with where there is no history of intemperance, and it has then been attributed to the influence of malaria or prolonged heat; to the abuse of hot condiments and various articles of diet; to the circulation of products of faulty digestion; or to the extension of a localized peritoneal inflammation. Cases have come under my notice which could not be traced to any definite cause, and it has appeared to me that occasionally there may be some hereditary influence at work. The complaint is chiefly met with between 30 and 50 years of age, being rare in youth, and not common in advanced life. There is no doubt, however, but that cirrhosis of the liver may be met with even in young children, of which a striking example has been brought before the Pathological Society by Dr. Griffiths of Swansea. Males suffer more than females; and also those persons who, from their occupation or in any other way, are more exposed to the ordinary exciting cause of the disease.

With regard to *hypertrophic cirrhosis*, it is affirmed that there is no distinct ætiological relation between this complaint and abuse of alcohol. The cases which have come under my notice, however, have been clearly traceable to this cause.

**ANATOMICAL CHARACTERS.**—In the advanced stage of *atrophic cirrhosis* the morbid appearances are very characteristic. The liver is greatly contracted, wasted, and diminished in weight, being sometimes reduced even to  $\frac{2}{3}$  or  $\frac{1}{2}$  the normal, especially the left lobe and edge, the latter being often merely a thin fibrous rim. The general form is frequently somewhat rounded. The surface is very pale, and is covered more or less with roundish prominences, varying in size from minute granules to projections or knobs measuring  $\frac{1}{4}$  to  $\frac{1}{2}$  an inch or even more in diameter, like hob-nails—hence the names *granular* and *hobnailed liver*. They may be tolerably uniform in size, but are more commonly unequal. Local puckering or depressions are also often observed. The capsule is thickened, opaque, and inseparable; while local peritoneal adhesions to the diaphragm and other structures, as well as thickenings, are almost constant. The consistence is remarkably dense, firm, tough, and leathery as a rule, which is best realized on making a section. This exhibits the same granular appearance as the surface, and sometimes in a much more marked degree. The colour is generally a mixture of dirty-white or greyish and yellow; the former being arranged in lines or bands of different widths, sometimes extending over considerable tracts; the latter, which varies in exact tint, being in some specimens bright yellow, in others almost brownish, corresponding to the granulations. The name *cirrhosis* is derived from this yellow appearance. In extreme cases, however, but little of this colour is evident.

The intimate changes in structure, and the microscopic appearances in cirrhosis of the liver must now be considered. The *white tissue* is generally supposed to be made up mainly either of fully-developed fibrous tissue; or of young connective-tissue elements or embryonic tissue in process of development, and chiefly resulting from proliferation. It has, however, been described as consisting in some instances of the remains of the vessels, ducts, and other tissues which have not undergone destruction. The fibrous tissue infiltrates the liver, occupying chiefly the capsule of Glisson surrounding the smaller branches of the portal vein, and the vaginal veins, and extending thence more or less into the interlobular spaces. Generally this material presents numerous new vessels running through it, which are stated by Frerichs to be derived from the hepatic artery; and although they have some communication with the portal vein, they seem to be chiefly intended for the nutrition of the liver and the secretion of bile. The *yellow nodules* correspond to lobules or groups of lobules which have not yet completely disappeared. The colour is chiefly due to stasis of bile, owing to pressure upon the minute ducts; it partly results, however, from fatty degeneration of the cells. A large proportion of these cells have become wholly destroyed, and most of those which remain are greatly altered, appearing shrunken or fatty, or containing pigment-granules. The degeneration begins at the circumference of the lobules, and extends towards their interior. The fibroid change rarely involves the hepatic lobules themselves, but the changes in them are due to the gradual compression to which they are exposed by the contracting tissue. The *vessels* present important changes. Many of the smaller branches of the portal vein are compressed or obliterated, and its capillaries are destroyed, so that injection of them from the main trunk is impossible. This trunk and the larger branches are often dilated, and may be occupied by thrombi. Sometimes a considerable branch of the vein is compressed. The hepatic artery is also commonly dilated, and new capillaries form in the fibrous tissue; frequently black pigment is found in its branches. The chief divisions of the hepatic vein are not altered, but many of its capillary tributaries are obliterated, and the communications between this vessel and the portal system are more or less destroyed. The remaining capillaries are commonly in a state of fatty degeneration.

The degree to which the changes just described are observed necessarily differs considerably according to the stage of the disease. In the earliest condition there can be no doubt but that the liver is enlarged, which is proved rather by clinical observation than by *post-mortem* examination. At this time the granular appearance is absent or but slightly marked; while the entire organ is congested, and is described as being occupied by a succulent, vascular, greyish material, consisting of young connective-tissue elements. In exceptional instances a cirrhotic liver is enlarged, owing to the co-existence of fatty or lardaceous disease.

*Hypertrophic cirrhosis* presents different characters from those of the atrophic form. The liver is enlarged throughout, and may attain to twice or three times its natural size, being also much increased in weight. It retains its normal shape and outline, and is tolerably smooth, but may be more or less irregular or somewhat hobnailed. The organ is very dense. On section it is seen that either embryonic tissue or fibroid tissue infiltrates it in a diffused manner. In some parts only dense fibrous tissue may be seen; in others remnants of the hepatic parenchyma are visible, sometimes considerable, scattered irregularly,



and of a colour varying from orange-yellow to green. Hypertrophic cirrhosis is said to differ from the atrophic form in that the change begins, not in the blood-vessels, but in connection with the interlobular branches of the bile-ducts, and the branches occupying the peripheral parts of the lobules, from which it extends, and only involves the portal vessels at a late period; it also tends to implicate the lobules themselves. The bile-ducts become greatly dilated, and their epithelium increases to an extent sufficient to block up the smaller ducts. The hepatic cells become atrophied, fatty, and more or less filled with bile-pigment.

The effects of cirrhosis outside the liver are highly important, and are visible on *post-mortem* examination, being mainly those already mentioned as resulting from obstruction of the portal circulation. Considerable anastomoses form between the hæmorrhoidal veins; and also between the superficial branches of the portal vein in the liver and the veins of the diaphragm and abdominal walls, through the peritoneal adhesions, and along the suspensory ligament.

Changes similar to those observed in cirrhosis of the liver are not infrequently met with in other organs and tissues at the same time.

A brief account will now be given of certain other forms of *chronic atrophy* of the liver:—

1. As the result of long-continued *mechanical congestion from heart-disease*, the liver contracts and presents characters much resembling those of true cirrhosis, but there is an important difference, and the atrophy is rarely so marked as in the latter disease. It results from pressure exerted by the distended tributaries of the hepatic vein upon the contiguous cells, causing their degeneration; hence the *centre* of the lobules becomes first wasted and depressed, while the circumference remains and forms granulations. Ultimately extensive depressions are produced, and more or less proliferation of connective-tissue occurs. Attacks of *chronic peri-hepatitis* are also common, which increase the tendency towards atrophy of the organ.

2. Dr. Murchison described a form of *granular atrophy*, generally independent of intemperance, in which the fibrous tissue is not increased, and the liver is softer than in health.

3. Atrophy may result from *adhesive pyle-phlebitis*, in consequence of which the trunk or some of the branches of the portal vein are obliterated. Cicatricial retractions are observed on the surface, with corresponding indurations.

4. Another form of atrophy of the liver is that due to chronic or repeated attacks of *peri-hepatitis*, which induces thickening of the capsule, or causes pressure upon the vessels, while fibrous bands pass into the interior, but there is no granular appearance.

5. Syphilis may lead to atrophy of the liver, either by exciting *peri-hepatitis* or simple *interstitial hepatitis*; or as the result of changes in *gummatous deposits*.

6. The last variety is named *red atrophy* by Rokitansky, or *chronic atrophy* by Frerichs. It may be associated with the deposit of pigment in the minute vessels of the liver, especially after prolonged or repeated attacks of malarial fever; or it sometimes follows ulceration affecting the alimentary canal. The entire organ is wasted, but the condition differs from true cirrhosis in the absence of any granulations on the surface; in a section being dark-brown or bluish-red and homogeneous, there being little or no indication of lobules; and in the consistence being less firm. The hepatic cells are often diminished in size, and

filled with brown pigment-granules. The ramifications of the portal vein are destroyed, its branches ending in cæcal club-shaped extremities.

**SYMPTOMS.**—In all the different forms of contracted liver just described, the chief diagnostic clinical indications are derived from the evidences of interference with the portal circulation; and from the signs afforded on *physical examination*. There are, however, additional symptoms resulting from derangement of the secreting functions of the liver; and others evidencing more or less marked constitutional disturbance.

In the *early stage of atrophic cirrhosis* it is customary to describe a train of symptoms which set in insidiously as a rule, but in reality they are merely those of congestion of the liver with gastro-enteric disorder, such as a sense of local discomfort or uneasiness; dyspeptic disturbances; or an inclination to sickness or retching. Though it might be suspected that cirrhosis was being set up, should such symptoms arise in association with abuse of alcohol, there is nothing characteristic about them. Occasionally the disease begins with severe local symptoms, indicating acute hepatic congestion, catarrh of the bile-ducts, and gastro-enteritis, accompanied with pyrexia. For a time there are *physical signs* of enlargement of the liver. As the case advances more or less of the consequences of *portal obstruction* are observed, namely, ascites, often extreme in amount; enlargement of the superficial veins of the upper part of the abdomen, especially on the right side; gastro-enteric congestion and catarrh; occasionally hæmorrhage from the stomach or intestines; hæmorrhoids; and enlargement of the spleen. Digestive disturbances are usually prominent, being due both to the state of the alimentary canal, and to deficiency or unhealthy quality of the bile. Although painful sensations over the hepatic region are sometimes present in the early stages, when the disease is advanced there is rarely much uneasiness, if any; there may be local tenderness, which is chiefly due to peritonitis or peri-hepatitis. Jaundice also is but seldom a prominent symptom, being often entirely absent, but more or less yellow discoloration is observed in many cases from time to time, especially at the early period, mainly due to hepatic congestion, catarrh of the ducts, or pressure of enlarged glands in the portal fissure upon the main duct. Extreme jaundice occasionally results from peri-hepatitis; or it appears towards the termination of a case of cirrhosis, independently of any obstruction. The stools almost always contain bile.

*Hypertrophic cirrhosis* is said to differ clinically from the atrophic form in that jaundice is an early and marked symptom, often becoming very intense, and being persistent, though variable; while the signs of portal obstruction are either absent, or only come on at a late period, and even then the ascites is slight. Cerebral and other toxæmic symptoms are also prominent. The spleen is generally enlarged. In my experience of cases of hypertrophic cirrhosis the distinctions just mentioned have been by no means constant.

*Physical signs.*—The signs of *atrophic cirrhosis* are :—1. Diminution in area of hepatic dullness, in proportion to the degree of contraction. 2. Granulation or nodulation of the surface of the liver, with a feeling of hardness; in short, the tactile characters described under the morbid anatomy. Sometimes the edge of the liver can be grasped between the thumb and fingers, and the changes thus readily realized. 3. Occasionally friction-sound. Ascites often obscures the examination, but under such circumstances the organ can frequently be easily felt after

paracentesis; or even by suddenly pressing down upon it. It must be remembered that in some instances the liver is not materially altered in dimensions, or that there may even be enlargement of the organ, but the nodulated surface can then generally be readily recognized. In the *hypertrophic* form of cirrhosis the liver is found by palpation and percussion to be more or less enlarged, sometimes enormously; regular in form, with a sharp anterior margin; and very hard.

The *constitutional* symptoms are frequently very marked in advanced cases of cirrhosis, there being considerable emaciation and weakness; a peculiar sallow, earthy complexion; a dry harsh skin; and flabbiness of tissues. Purpuric spots and blotches on the skin are sometimes visible, and there may be extensive ecchymoses, or hæmorrhages from mucous surfaces may take place. The urine often yields sediments of uric acid and urates, and uroerythrin is present in some cases.

COURSE AND TERMINATIONS.—The *progress* of cirrhosis is generally very chronic, but it may run a tolerably rapid course from the first appearance of distinctive symptoms. Sometimes, even after serious symptoms have appeared, great improvement may take place, so that the patient may feel as if almost or quite restored to health, and may live for many years if he exercises proper care. In most cases, however, cirrhosis leads to a fatal issue. The chief *modes of death* are from gradual asthenia and exhaustion; jaundice with typhoid symptoms; lung-complications; acute peritonitis; or hæmorrhage from the alimentary canal. It must be remembered that the more serious of these events are liable to occur at any time.

The other forms of contracted liver only differ clinically from atrophic cirrhosis in the circumstances under which they arise; and in the *physical characters* presented by the liver on palpation, if the organ can be felt. In the variety due to *peri-hepatitis*, considerable pain and tenderness are generally complained of from time to time.

## VII. SYPHILITIC DISEASE.

The morbid conditions of the liver which may result from syphilis are:—1. *Albuminoid disease*. 2. *Peri-hepatitis* and its consequences. 3. *Simple interstitial hepatitis*, leading to general atrophy and induration. 4. *Gummatous hepatitis*, in which syphilitic gummata are formed more or less extensively, undergoing degenerative changes, and becoming surrounded by a dense fibroid tissue, from which processes extend toward the surface of the organ in various directions. They are rounded or irregular; usually numerous, and often grouped in clusters; varying in size from a pin's head to a chestnut; opaque and buff-coloured; and very tough. They may soften in the centre, or become calcified or absorbed. The liver-tissue becomes destroyed; and deep cicatricial depressions or furrows are seen on the surface of the organ, giving rise to a lobulated appearance. During life the characters of the liver may often be determined by *physical examination*, in the gummatous form the organ becoming enlarged slowly, and it may ultimately reach an enormous size, at the same time being irregular in form. Pain is frequently experienced, with tenderness; and sometimes signs of obstruction of the bile-ducts and portal vein appear. There may be such marked wasting and cachectic symptoms, that, taken with a much enlarged and nodular liver, cancer may be simulated. The progress is usually very chronic.



## VIII. TUBERCULOSIS.

Tubercle is usually observed in the liver only in connection with general *acute miliary tuberculosis*. Occasionally it is secondary to chronic tubercular disease in other parts. The tubercle may break down and form small cavities. Clinically the disease cannot be recognized with any certainty. The liver is usually enlarged.

## IX. RICKETY LIVER.

It will be sufficient to mention this form of diseased liver, which has been specially recognized by some authorities, as it has been already discussed under rickets.

## CHAPTER XLIX.

## AFFECTIONS OF THE GALL-BLADDER.

THE morbid conditions to which the gall-bladder is liable need only be briefly indicated. Most of them cause enlargement of the organ, and it is important to be able to recognize the distinctive clinical characters of each form of enlargement.

1. DISTENSION WITH BILE.—When anything obstructs the common bile-duct, such as a gall-stone, the gall-bladder becomes filled with bile, and may attain enormous dimensions. There will then be the usual signs of obstructive jaundice, with enlargement of the liver; while the gall-bladder is perceptible as a fluctuating tumour, sometimes reaching nearly to the iliac crest, and being generally somewhat tender.

2. ACUTE INFLAMMATION AND SUPURATION.—The mucous membrane of the gall-bladder is liable to simple catarrh, or to croupous or diphtheritic inflammation, like other mucous surfaces; but the most important form of *acute* inflammation is that which is attended with the formation of pus in its interior, which particularly results from irritation of its mucous membrane by gall-stones, or from obstruction of the cystic duct by these bodies. The condition is clinically indicated by a very painful and tender fluctuating enlargement of the gall-bladder, which may ultimately assume the characters of an abscess, or may even burst; accompanied with marked rigors and pyrexia, the latter tending to become of a hectic type. The inflammation is often preceded by signs of gall-stones; but there is neither jaundice nor hepatic enlargement as a rule.

3. CHRONIC INFLAMMATION. HYDROPS VESICÆ FELLEÆ. DROPSY OF THE GALL-BLADDER.—If the cystic duct is obstructed for a long period, the gall-bladder may become gradually dilated, owing to the accumulation of a clear, serous or synovial-like fluid, the product of unhealthy secretion from the mucous surface, probably partly the result of chronic catarrh; while its walls become much thinned and atrophied. The organ is more or less distended, and often attains an extreme size; but there is little or no pain or fever; while jaundice is absent; and the

liver is not enlarged. Occasionally the course of events is different. The liquid portion of the contents of the gall-bladder becomes absorbed, leaving an inspissated substance, in which calcareous salts are deposited; the walls undergo thickening and contraction from chronic inflammation; and ultimately a firm puckered mass is left, enclosing a chalky pulp.

4. ACCUMULATION OF GALL-STONES.—Gall-stones are often present in the gall-bladder, without affording any clinical evidence of their existence. In some instances, however, and especially when they are very numerous and large, they cause local uneasy or painful sensations, which are increased after food, or after much exertion or jolting; as well as reflex disturbance of the stomach and other parts; and sometimes much constitutional discomfort and depression. Occasionally also they give rise to severe symptoms from time to time, by attempting to enter the cystic duct, and subsequently falling back into the gall-bladder. They may further excite inflammation or ulceration of the mucous surface, the latter ending sometimes in perforation, or giving rise to pyæmia. In rare instances such a number of calculi collect, that they form a tumour, even of considerable size, having the general characters of an enlarged gall-bladder as regards position, shape, and mobility, but presenting the following distinctive characters:—1. The tumour feels hard and sometimes nodulated. 2. On palpation a peculiar sensation is experienced, owing to the rubbing together of the calculi, compared to that produced by grasping nuts or pebbles. 3. A corresponding sound may be heard on auscultation; and occasionally loud rattling is perceptible on shaking or moving the patient. Now and then local peritonitis is excited by this enlargement, so that it becomes adherent and fixed. When such a tumour exists, there are necessarily more marked subjective sensations, such as weight and uneasiness, especially on moving from side to side. The progress of these cases, as well as the growth of the enlargement, is very slow and gradual.

5. CANCER.—The signs of this rare disease are:—1. Lancinating pains, with much tenderness, in the region of the gall-bladder. 2. A tumour, having more or less of the characters of enlarged gall-bladder, but usually feeling firm, resistant, irregular and nodulated, without the peculiar sensation of gall-stones; being adherent and fixed; and growing rapidly. There are always evidences of cancer in other parts; with well-marked cancerous cachexia. A fistulous communication with the intestines is often established. Gall-stones are usually present in the gall-bladder. Jaundice and vomiting are common symptoms.

---

## CHAPTER L.

### GALL-STONES—BILIARY CALCULI—CHOLELITHIASIS.

ÆTIOLOGY AND PATHOLOGY.—There is considerable uncertainty as to the mode of origin of gall-stones. The chief views may be thus stated:—1. That they are merely the result of *inspissation and concentration of bile*. 2. That they depend upon certain *biliary ingredients being in excess*, especially cholesterin and colouring matters. 3. That the bile has some *abnormal chemical composition*, either when first formed, or as the

consequence of subsequent changes, which prevents it from holding certain elements in solution, and hence they are deposited. Thus calculi have been attributed to deficiency of soda, with excessive acidity of the bile; excess of lime causing a separation of pigments; decomposition of the salts of soda with the biliary acids; or decomposition of the biliary acids themselves, with consequent precipitation of cholesterin and pigment. 4. That they originate in *plugs of mucus, epithelium, or foreign bodies*, upon which the ingredients of the bile are afterwards deposited as a nucleus. It is highly probable that each of these views is correct in different cases, and when once the formation of a gall-stone has commenced, its increase may be due to some other cause than that which originated it in the first instance. There can be no doubt but that a catarrhal state of the gall-bladder and ducts favours the production of calculi, either by inducing stagnation of bile; or, as some believe, by the mucus then formed favouring decomposition of this secretion, or impregnating it with carbonate of lime. This decomposition has also been attributed by Thudichum to the absorption of some ferment from the intestines.

There are some important *predisposing causes* of gall-stones, namely, advanced age; the female sex; sedentary habits; habitual constipation; over-indulgence in animal food and in stimulants; and organic diseases of the liver, gall-bladder, or bile-ducts, interfering with the escape of bile. Biliary calculi have also been attributed to drinking water containing excess of lime, but on no adequate grounds.

**ANATOMICAL CHARACTERS.**—By far the most frequent *original seat* of biliary calculi is the *gall-bladder*, but they may also be formed in any portion of the bile-ducts, or even in the liver itself. The number varies from one to hundreds or thousands; usually several are found. There is also a wide range as to size, this being in an inverse ratio to the number; several are sometimes cemented together, so as to form a large concretion. Originally most of the calculi are round or oval, but when numerous, owing to mutual friction they become worn and angular, presenting flat or concave facets, or occasionally actual articulations. When formed in the ducts they exhibit curious shapes, being branched or coral-like. As a rule gall-stones have a brownish or greenish-yellow colour, and are opaque, but they present an endless variety of tints, ranging from white to black, blue, green, red, and other colours, according to their composition; occasionally they are somewhat translucent. They frequently have a greasy or saponaceous feel, with a waxy, brittle consistence, being readily cut or crushed; sometimes they are very firm. Most of them sink in water when recent, but some float, and most gall-stones will do so after having been dried. The structure is rarely homogeneous and uniform. In the majority of cases, after a calculus has existed for some time a section reveals distinctly three parts, named from within out—the *nucleus*, of which there may be more than one; the *body*, which is often made up of concentric layers, or presents a radiated appearance; and the *cortex or crust*, this being usually smooth externally, but occasionally wrinkled, rough, or even tuberculated and warty. As a rule the layers become lighter in colour from the centre towards the circumference, but not always. Sometimes a fractured calculus presents a crystalline aspect. The *chemical composition* is very variable, but the most common ingredients are cholesterin and bile-pigments, with a little lime or magnesia. To these may be added biliary and fatty acids, generally combined with lime; modified bile-pigments; phosphates; car-



bonates; salts of soda or potash in small proportions; and metals (iron, copper, and manganese). The nucleus is often made up of mucus and epithelium, and the former material may also unite the different parts. The appearances differ according to the composition, which is not necessarily uniform even in the same layer. It is quite impossible to describe the characters corresponding to the various ingredients, but it may be stated generally that in proportion to the amount of cholesterin which a calculus contains it is whiter, more transparent, crystalline or radiated and lamellar, and of lighter specific gravity.

*Biliary sand or gravel* is not uncommonly met with, consisting either of cholesterin, bile-pigment, or black pigmentary matter.

The morbid conditions which are liable to be set up by gall-stones may be stated as follows:—1. Irritation, inflammation, suppuration, or ulceration, with consequent pyæmia or perforation, affecting either the gall-bladder or ducts, perforation taking place in different directions, but especially into the stomach, duodenum, or peritoneum, or externally through the abdominal wall; rarely into the colon, portal vein, pleura, pelvis of the right kidney, or vagina. Permanent fistulæ may be left. 2. Inflammation and abscesses in the liver, if lodged there; or the formation of a cyst around the calculi. 3. Obstruction of some of the ducts in the liver, or of the hepatic, cystic, or common bile-duct, with the usual consequences. 4. Obstruction of the intestines by a large calculus, this having probably entered through a fistulous communication from the gall-bladder. 5. Inflammation, ulceration, or gangrene of the bowel, with consequent perforation.

**SYMPTOMS.**—It is only needful here to describe those symptoms which indicate the passage of a gall-stone along the bile-duct to the intestine—*biliary or hepatic colic*—these being usually severe, but not always. An attack of hepatic colic begins with a sudden intense pain in the right hypochondrium, in some cases most excruciating, often coming on just after a meal or after effort; it is described as constricting, griping, tearing, burning, or boring, and shoots over the abdomen, round the side to the back, or towards the right shoulder. The patient is doubled up and rolls about just as in ordinary colic, groaning or screaming, and pressing upon the abdomen, which gives some relief, there being generally no tenderness at first. The pain may subside, leaving a dull aching, but urgent paroxysms recur at intervals. The attacks are accompanied with much exhaustion; signs of collapse, which may be extremely grave and accompanied with rigors; a distressed and anxious expression of countenance; faintness, which may end in actual syncope; and cramps of the abdominal muscles. There is no pyrexia usually. Sympathetic vomiting is frequently present; and sometimes hiccup is a distressing symptom. Among occasional symptoms are observed spasmodic tremors or actual convulsions, and marked rigors. In the course of a day or two, should the gall-stone reach the common duct, as a rule the usual signs of obstructive jaundice are developed, which may become intense, the duration of the jaundice depending upon that of the obstruction. When the calculus reaches the duodenum the suffering generally subsides suddenly, with a feeling of intense relief, and then the jaundice gradually disappears. In the great majority of cases biliary calculi pass along the intestinal canal, and are discharged in the fæces, sometimes in great numbers, without producing any further mischief, and they may be detected by washing the stools through a sieve or through muslin. Very rarely they pass into the stomach, and are vomited.

There are a few points of practical import which require notice. The intensity of the pain is by no means necessarily in proportion to the size of a gall-stone, but rather depends upon its angular shape. It usually diminishes when the concretion reaches the common duct, because this is larger than the cystic duct, but it increases again as the orifice into the duodenum is approached. Jaundice is not a necessary accompaniment, or it may be but slight, because when the calculus is angular in form it leaves room for the bile to flow by, or its passage is sometimes too rapid to allow of the appearance of jaundice; on the other hand, this symptom may become persistent and extreme, owing to the permanent impaction of a gall-stone. It is very important to look for biliary calculi in the stools, as by their shape, number, and size an opinion can often be arrived at as to whether any remain behind in the gall-bladder, while at the same time their characters are recognized. After one large gall-stone has escaped, other smaller ones often follow without causing any particular disturbance. Sometimes the pain subsides, but no calculus is passed, because it returns to the gall-bladder. Pain and soreness may remain after the escape of a concretion into the duodenum, owing to nervous irritability on the part of the patient, or to local irritation of the nerves; or inflammation may be excited, indicated by pain and tenderness, with fever. The symptoms of hepatic colic are occasionally merely due to the passage of grit or inspissated bile. An attack may end fatally, from the mere intensity of the pain and collapse, quite irrespective of the serious morbid changes which a gall-stone is liable to set up, any one of which may cause death.

---

## CHAPTER LI.

### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT OF CHRONIC HEPATIC AFFECTIONS.

#### I. DIAGNOSIS.

THE main elements which assist in the diagnosis of chronic hepatic diseases, both from other affections and from each other, are as follows:—1. The *general history* of the patient may reveal some known cause of certain liver-complaints, especially abuse of alcohol; over-eating, with deficient exercise and general luxurious habits; prolonged residence in tropical climates or in malarial districts; the previous occurrence of dysentery or ague; or syphilitic infection. *Family history* may afford some aid in diagnosis, especially if indicating a cancerous taint; while in some cases the *age* and *sex* of the patient also deserve consideration. 2. The *constitutional condition* is highly important. Thus there may be some disease with which lardaceous or fatty liver is likely to be associated; or signs of the cancerous cachexia, of syphilis, or of cirrhosis may be evident. On the other hand, the absence of any constitutional disturbance is sometimes serviceable in diagnosis. 3. The presence or absence of *symptoms referable to the liver*, as well as their nature, intensity, and the history of their progress, deserve careful attention, especially as regards pain and tenderness; jaundice; and ascites or other evidences of portal obstruction. 4. *Physical examination* is of course of essential value. This will be presently more fully

alluded to. 5. The *state of other organs* may afford much aid in diagnosis, especially by revealing local manifestations of some constitutional disease, for instance, cancer of the stomach, or waxy kidney; or of some morbid condition with which hepatic derangement is likely to be associated, especially ulceration in some part of the alimentary canal, gastro-enteric catarrh, or disease of the heart obstructing the circulation. 6. The *rapidity of the progress* of a case up to the time when it is first seen; its subsequent *course*; and the results of *treatment*, are to be taken into account in doubtful cases.

*Physical examination* demands special attention, particularly in detecting and making out the characters of enlargements or contractions of the liver; and of enlargements of the gall-bladder. For differential diagnosis of *hepatic enlargements* the following points must be noted, and in the description of each individual disease an endeavour has been made to arrange the characters in the same order:—*a.* The extent, direction, and rapidity of growth. *b.* Whether the liver is normal in shape and outline; or if it presents outgrowths or irregularities. *c.* The conditions of the surface and margins, as to smoothness, nodulation, &c. *d.* The degree of resistance and other sensations afforded by the liver generally, as well as by any special prominences, including fluctuation and hydatid-fremitus. *e.* Whether there is any evidence of local peritonitis, indicated by friction-fremitus or sound, or by adhesions of the liver with the abdominal wall. *f.* Now and then it is requisite to use the aspirateur for diagnostic purposes. The characters to be observed in connection with *contractions* of the liver, and abnormal conditions in the *gall-bladder*, have been sufficiently indicated in their respective descriptions. It is necessary to add with regard to the gall-bladder, that it should always be noticed whether this is altered alone or along with the liver, and *vice versâ*.

It may be useful to enumerate here the causes of *enlarged liver*. The ordinary forms are due to:—1. Congestion, especially mechanical. 2. Accumulation of bile from any obstruction in the ducts. 3. Albuminoid disease. 4. Fatty infiltration. 5. Hydatid disease. 6. Cancerous and other growths. 7. Acute hepatitis, especially when ending in suppuration. 8. Cirrhosis in its early stage; and the hypertrophic form. As rare causes may be mentioned—9. Simple hypertrophy. 10. Syphilitic gummous hepatitis. 11. Tubercle. 12. Lymphatic growths. 13. A peculiar enlargement associated with vitiligoidea. 14. Rickets. It must be remembered that the liver may be enlarged by a combination of certain of the conditions just mentioned.

The chief practical difficulties in making out a diagnosis which have come under my notice are as follows:—1. Hepatic enlargement, and sometimes even changes in shape and other characters of the liver, may be simulated by the normally large size of the organ in children; congenital malformation; pressure by a rickety or otherwise deformed thorax, or as the result of tight-lacing; depression by various morbid conditions within the chest, especially pleuritic effusion and tumours or other causes of downward displacement, which may be acute; or elevation towards the chest by abnormal conditions within the abdomen. On the other hand, morbid changes affecting the liver may be obscured by distension of the colon with gas, which may even give rise to signs simulating atrophy of the organ. The liver is also occasionally displaced in such a way that its anterior edge is directed forwards, so that atrophy is simulated. 2. Morbid states of other structures often give



rise to signs of hepatic derangement; or, on the contrary they may put these in the background. Thus, enlargement of the liver may be simulated by a rigid and contracted state of the right rectus abdominis muscle; inflammation and suppuration in the abdominal walls; accumulation of fæces in the colon; or by a tumour in connection with the right kidney, supra-renal capsule, or peritoneum, especially the great omentum. Again, a neighbouring disease, particularly scirrhus of the head of the pancreas, often interferes with the escape of bile from the liver, and thus leads to its enlargement, accompanied with jaundice. The co-existence of ascites, or of chronic peritonitis with effusion, frequently renders physical examination unsatisfactory. The use of the aspirateur is then most serviceable, in order to remove the fluid; and also the plan of making sudden pressure over the liver. Not uncommonly disease of the liver is obscured by symptoms referable to a morbid state of some other organ, for example, cancer of the stomach; and it often itself gives rise to serious disturbance of the alimentary canal. Occasionally the liver is affected along with other organs and structures in the abdomen, particularly in cases of cancer, and then it is frequently impossible to make out distinctly what parts are actually involved. It is sometimes difficult to distinguish between enlarged liver and pleuritic effusion or a tumour in the chest; and these conditions may exist together. 3. Sometimes the liver becomes so enormous, especially as the result of hydatid disease, that it fills the abdomen, and hence it becomes impossible to determine exactly where enlargement commenced. In such cases the history of its growth, as regards the region from which it started; and perhaps the greater prominence of the enlargement over the hepatic region, may clear up the difficulty. 4. In some instances the liver is the seat of two or more distinct morbid conditions, the signs being modified accordingly, such as cirrhosis with fatty or albuminoid disease. 5. With regard to individual diseases, it may be mentioned that *hydatid tumour* is liable to be confounded with distended gall-bladder; soft cancer; right pleuritic effusion; hepatic abscess which has become somewhat chronic; aneurism; cystic disease of the kidney; hydatids outside the liver; or peritoneal effusion limited by adhesions. *Cancer* may be simulated by syphilitic disease; waxy liver, especially if combined with cirrhosis, or if some parts of the liver are more affected than others, so as to give rise to local projections; other forms of cirrhosis attended with enlargement; or multilocular hydatid disease.

It is requisite to make a few remarks relative to *pain* referred to the hepatic organs. This may be simulated by painful affections of the superficial structures, either muscular or neuralgic; gastric and duodenal disorders, either functional or organic; intestinal colic; accumulation of fæces in the colon; aneurismal, pancreatic and other tumours pressing on the nerves; the passage of a renal calculus; pleurisy; the pain met with in hypochondriasis; or local peritonitis. The attacks due to the passage of a *gall-stone* are generally clearly indicated by the individuals in whom they occur; the past history; and the symptoms present, especially when these are followed by jaundice, and by the escape of calculi in the stools. It must be borne in mind that gall-stones are not uncommonly associated with organic disease of the liver or gall-bladder. Simple *hepatalgia* is difficult to make out positively. Its characters have already been sufficiently indicated.

## II. PROGNOSIS.

The prognosis in the case of a chronic hepatic disease depends mainly upon the nature of the complaint; the degree to which the functions of the liver are interfered with, the escape of its secretion prevented, or its circulation impeded; the constitutional condition; the state of other organs; the possibility of removing any causes which may be keeping up the disease; and the results of treatment. *Fatty* and *lardaceous diseases* are very slow in their progress, and in many cases do not seem to hasten the fatal issue materially, though they are but little amenable to treatment. *Cancer* is necessarily fatal, and is frequently very rapid in its course, especially when of the softer kind. *Hydatid disease* is markedly chronic, and usually unattended with danger; while it may be cured in many cases by appropriate treatment. It occasionally proves dangerous in consequence of the cyst rupturing, or becoming inflamed and suppurating; or through some of its contents being discharged into the bile-ducts, blocking them up. *Syphilitic liver* can frequently be much improved by early and suitable treatment. The different forms of *contracted liver* are generally serious as regards their ultimate prognosis, though usually slow in their progress. I desire, however, again to draw attention to the fact, that in cases of cirrhosis, if the ascites can be permanently got rid of, a result which may not uncommonly be attained, the patient may be restored to comparatively good health, and may live for many years, engaged in the ordinary avocations of daily life, even in cases which appear to be almost hopeless. It must be remembered that serious and sometimes rapidly fatal hæmorrhage from the alimentary canal is liable to occur in cirrhosis. From the account given of the clinical history of *gall-stones*, it will be evident that there are many dangers attending them. Examination of any calculi passed, as to size, number, and shape, will aid in determining whether any remain in the gall-bladder; and whether the attacks of hepatic colic are likely to recur.

## III. TREATMENT.

The management of cases of chronic hepatic disease should be conducted according to very simple and obvious principles.

1. The **diet** needs careful supervision. It often has to be adapted to some constitutional condition, and therefore of a nutritious character, containing abundant protein elements; but it should always be as simple and easily digestible as possible, and particular caution is requisite in the use of alcohol or hot condiments, of fatty, amylaceous, and saccharine substances, and of rich articles of diet generally. In certain conditions it is important to limit the consumption of nitrogenous elements. In many cases it is highly important to forbid all stimulants, or only to allow light wines, and if spirits are ever needed, they should be given much diluted, and in restricted quantities. Any one who is accustomed to indulge in excess of alcohol, and particularly in ardent spirits, must be impressed with the absolute necessity of relinquishing this habit. It is desirable to recommend the patient to take an abundance of salt with food.

2. **Hygienic management** is of much consequence in some cases of hepatic disease. In addition to the ordinary measures for improving the general health, the points which claim special notice are removal from a tropical climate, or from a malarial district; cessation of sedentary and luxurious habits generally, a sufficient amount of exercise in the open air being taken daily; and the maintenance of free excretion of the skin, by the aid of baths.

3. Treatment directed against some **constitutional condition** often proves highly serviceable, and it may have a direct effect upon the liver, which applies particularly to *fatty*, *lardaceous*, and *syphilitic* disease. General *tonic* treatment, as well as remedies for improving the quality of the blood, are beneficial in many cases, such as the various preparations of iron, strychnine or nux vomica, or mineral acids with bitters. Tincture of iodine well-diluted; iodide of potassium or of iron; and carbonate of ammonia or chloride of ammonium, have been found by different observers to influence the size of albuminoid liver. Chloride of ammonium has been much recommended in various forms of chronic hepatic disease. Of course mercury and iodide of potassium are the remedies for syphilitic disease.

4. There is a class of therapeutic agents which act more or less directly upon the liver, influencing its secretory functions, and hence named **cholagogues**. Our knowledge respecting these agents has been much extended and rendered more certain by the experiments conducted at the Edinburgh University under the direction of Prof. Rutherford. The term *cholagogue* is applied to any drug which increases the flow of bile, and Rutherford divides the agents belonging to this general group into (*a*) *hepatic stimulants*, or those which influence the bile-secreting mechanism; and (*b*) *bile-expellents*, which increase the expulsion of bile, by stimulating muscular contraction of the gall-bladder and bile-ducts. He found that there is no relation between the action of medicines as intestinal stimulants and hepatic stimulants, many agents which exert a powerful effect upon the intestines having no influence upon the liver, and *vice versâ*. The chief medicines which have usually been credited with a special action upon the liver are mercurial preparations, especially blue-pill, calomel, and grey powder; podophyllum and podophyllin; nitro-muriatic acid; and taraxacum. Rutherford's experiments seem to prove that calomel has no influence as a bile-secreting agent, and taraxacum has only a very feeble action of this kind. With regard to recognized drugs, he found the following to be more or less powerful hepatic stimulants, namely, podophyllin, provided the dose is not too large; aloes, jalap, and colocynth; dilute nitro-muriatic acid; corrosive sublimate, either alone or combined with calomel; sulphate, phosphate, benzoate, and salicylate of sodium; sulphate of potassium; phosphate and benzoate of ammonia; and ipecacuanha. Rhubarb is a certain, though not a powerful, hepatic stimulant. Rutherford has experimented upon other drugs, and has discovered several agents which seem to have an important influence upon the hepatic secretion, including substances named euonymin, sanguinarin, baptisin, hydrastin, juglandin, inulin, and iridin.

The experiments just alluded to refer to the action of medicines upon the liver in health. These must not be relied upon too implicitly in the treatment of diseased conditions, for clinical observation affords evidence that in certain morbid states, when the bile is deficient, mercurial preparations decidedly increase the quantity of this secretion. It may be



that they act by aiding in the removal of some impediment to the formation of bile; or by promoting its discharge. Murchison remarked respecting these agents, that they probably irritate the upper part of the small intestines, so that the bile is propelled onwards, instead of being reabsorbed. The best plan of administering mercury, in the case of an adult, is to give occasionally a tolerably full dose of calomel or blue-pill, either alone, or combined with rhubarb or colocynth pill and extract of henbane. A combination of calomel and extract of conium has also been found very useful. For children grey powder answers best. It is certainly injurious to fall into the habit of constantly taking these medicines. A dose of podophyllin now and then is frequently very serviceable. A combination of nitro-muriatic acid with extract of taraxacum enjoys considerable repute, especially in the treatment of congestion of the liver, and of the earlier stages of cirrhosis, but probably the taraxacum acts mainly through its direct action upon the alimentary mucous membrane. Sir Ranald Martin recommended the nitro-muriatic acid bath (ʒi of strong nitric, and ʒij of hydrochloric acid to a gallon of water at 90° to 98° F.), in which the feet are placed, and then the inside of the upper and lower extremities, as well as the abdomen, are sponged over freely. This bath seems to be of much benefit to those who come from tropical climates, suffering from disordered liver. Rutherford has found euonymin and iridin very efficient remedies in the treatment of "biliousness" and functional hepatic derangement. He gives of the former gr. ij, of the latter gr. iv, at night, in the form of a pill, followed in the morning by a mild saline aperient.

5. Symptoms referable to the **alimentary canal** commonly call for treatment in connection with liver-diseases, such as those due to gastric or enteric catarrh, constipation, flatulence, or hæmorrhage; or there may be co-existing organic disease affecting the stomach or intestines, such as cancer. These conditions must be treated by the usual remedies, especially by alkalies and their carbonates, citrates, tartrates, and other vegetable salts; different bitter infusions or tinctures; *saline aperients*; and *saline mineral waters*, either English or Continental. It is very desirable to keep the bowels acting as regularly as possible, though the frequent use of strong purgatives must be avoided. Should the patient be suffering from hæmorrhoids, confection of senna or of sulphur are valuable aperients.

6. The two prominent symptoms so frequently calling for treatment in liver-affections, namely, **jaundice** and **ascites**, have already been fully considered. I cannot, however, refrain from again insisting upon the importance of having recourse to the early and repeated removal of fluid by paracentesis, in cases of ascites associated with cirrhosis.

7. **Local applications** are frequently of service in hepatic affections, especially to relieve pain and congestion. They include chiefly dry heat; poultices and fomentations, to which *anodynes* may be added; sinapisms; anodyne plasters; dry-cupping; or the removal of a little blood by leeches or cupping.

8. It is desirable to look to the condition of **other organs**, and treat them if required, particularly the heart, a diseased state of which may be the immediate cause of hepatic symptoms. The kidneys also demand due attention.

9. The treatment of **hydatid tumour** requires separate consideration. For the cure of this complaint operative interference is needed, no known

drug having any influence upon the parasite, and a spontaneous cure being extremely rare. It is only, however, when the growth attains some size, and becomes a source of trouble, that this course of treatment should be adopted, though it should not be delayed for too long a period. There is much difference of opinion as to the most efficient plan of operation. The principal methods advocated are:—1. Puncture with the aspirateur, or by means of a trochar and canula, and evacuation of the fluid. 2. Puncture and subsequent injection of the cyst with some irritating liquid, such as bile or tincture of iodine, with the view of exciting inflammation. 3. Removal of the contents through a large incision. 4. Gradual opening of the cyst, by the repeated application of caustic potash to the abdomen, over the most prominent part of the tumour. This plan has been adopted with the view of causing adhesions to form, and thus preventing the escape of fluid into the peritoneum; and it has also been had recourse to for the same object previous to puncture with the trochar. 5. Puncture of the tumour with needles, and transmission of electric shocks through it. Some authorities assert that all that is necessary is to evacuate the fluid, and that then the parasite will die. Others consider that it is necessary to excite inflammation. The balance of evidence seems to be certainly in favour of the more simple methods. Some recommend the employment of a very small trochar, others of a large one; again, there is a difference of opinion as to whether it is requisite to remove the whole of the fluid or not, some even using an exhausting syringe to draw this off. Murchison advocated the employment of a very fine trochar, and advised that the canula should be removed before the whole of the fluid has been drawn off, or as soon as it ceases to flow in a full stream, first passing a wire through the tube to ascertain that it is not stopped up by a hydatid vesicle. The object of this plan is to prevent the entrance of air, which is one of the main dangers, as it tends to set up suppuration. Another danger is the escape of fluid into the peritoneum; and in order to prevent this mishap, pressure should be made over the punctured portion of the abdomen during the removal of the canula. The opening should be made over the most prominent part of the tumour. The administration of chloroform is not advisable, but local anæsthesia may be induced. After the operation the opening is to be closed with lint steeped in collodion, over which a compress and bandage should be applied. Absolute rest is necessary for two or three days; and an opiate should be given at once, and repeated if necessary. The fluid may collect again, and it may be requisite to repeat the operation. Murchison, however, cautions against doing this too soon, as the enlargement may be due to inflammatory effusion. In cases which are ultimately successful, a considerable degree of fulness may remain for some months. Should the tumour be very large, its walls are likely to be thicker and less elastic, and then it appears desirable to use a large trochar. A free incision is only admissible when suppuration has taken place; the whole of the contents of the hydatid tumour being then evacuated. It is also recommended to use a large trochar under such circumstances, and to leave an elastic tube in, the cyst being washed out with carbolic acid solution. The different events which may happen in connection with hydatid tumour must be treated on ordinary principles. In those countries where hydatid disease is prevalent, *prophylactic* measures are necessary, namely, to prevent dogs from feeding on the offal of sheep; to exclude them from slaughter-houses; to give them meat thoroughly boiled; to destroy

their excreta which contain tape-worms : and to physic them periodically (Murchison.)

10. The treatment of **gall-stones** also calls for a few special remarks. During the passage of a gall-stone the chief measures to be carried out are:—*a.* To administer *narcotics* and *anodynes*, especially opium or morphia in full doses, subcutaneous injection of the latter being very valuable; belladonna; hyoscyamus; or chloroform and ether, either internally or by inhalation. *b.* To treat certain symptoms, especially vomiting and collapse. *c.* To apply dry heat, hot fomentations, poultices, or anodyne applications constantly over the hepatic region; or to put the patient in a warm bath. Antimony and other *emetics*, which were formerly much employed, partly with the view of mechanically expelling the calculus, as well as strong *purgatives*, ought certainly to be avoided, in my opinion. Much good is effected in some cases by the treatment introduced by Dr. Prout, of making the patient drink a considerable quantity of a warm solution of bicarbonate of soda (3i or 3ij to Oj). Large warm enemata may also prove beneficial. The application of a few leeches over the hepatic region seems to be useful in prolonged cases, especially if there is much tenderness. For the *prevention* of gall-stones attention to diet and hygiene is most essential; and the use of remedies which improve the condition of the alimentary canal, or of those which act upon the liver, is also often of much service. It has been supposed that hepatic calculi can be dissolved after their formation, by the administration of a mixture of turpentine and ether, chloroform, alkalies, or alkaline mineral waters, especially Carlsbad. It is very doubtful whether either of these agents has any such effect, but alkalies and mineral waters often do a great deal of good in other ways in cases of gall-stone. The various consequences which may result from gall-stones must be treated as they arise. Inflammation involving the *gall-bladder*, from whatever cause, requires the application of poultices and fomentations. If pus forms, or if in chronic cases much fluid collects, it is sometimes requisite to puncture the gall-bladder, and allow the fluid to escape, either leaving an external fistula or closing up the wound. Cholecystotomy has also been performed for the removal of gall-stones retained in the gall-bladder, or impacted in the cystic duct.

---

## CHAPTER LII.

### DISEASES OF THE SPLEEN.

#### A. CLINICAL CHARACTERS.

1. The spleen is often diseased without giving rise to any local **morbid sensations**. When it becomes much enlarged it may cause a sense of fulness and tension, which is chiefly felt about the left hypochondrium. Occasionally more or less pain and tenderness are complained of in connection with splenic diseases.

2. A most important indication of splenic disease is derived from the **constitutional condition**. In prolonged chronic cases a state known as



*splenic cachexia* is induced. This is characterized by extreme anæmia, the mucous membranes being pale and bloodless, and the face presenting a waxy, or sometimes an earthy and sallow aspect; great debility; wasting, but not usually rapid; a sense of prostration and dullness; shortness of breath on any exertion, with hurried breathing, chiefly due to the anæmia; tendency to hæmorrhages, especially in the form of epistaxis, bleeding from the gums, and petechiæ under the skin; and œdema of the legs and eyelids, or even general dropsy.

3. Symptoms may arise from **pressure** by an enlarged spleen on surrounding parts, especially on the diaphragm, dyspnœa being thus increased, or even pulmonary congestion and catarrh induced. Vomiting may also be excited by pressure on the stomach.

4. The chief aid in the diagnosis of morbid conditions of the spleen is derived from **physical examination**. The characters of *splenic enlargement* or *tumour* are as follows:—*a.* In *position* it is extra-pelvic, and occupies mainly the left hypochondrium, being felt to come from beneath the margin of the thorax on that side. In its growth an enlarged spleen tends towards the front of the abdomen, as well as downwards and to the right, so that ultimately it extends into other regions and comes to be superficial, while it can generally be separated posteriorly from the mass of the dorsal muscles. Percussion often shows increase in area of *splenic dullness* upwards towards the thorax, or backwards, but it rarely reaches above the fifth rib, and does not extend as far back as the spine. There is also an undue sense of resistance on percussion; with deficient elasticity of the ribs. *b.* The *form* is usually very characteristic, being more or less that of the spleen exaggerated. The anterior border can be felt to be directed obliquely downwards and towards the right, being sharp and thin, and often presenting one or more notches or shallow excavations. The posterior edge and lower end are rounded. The outline of the spleen may occasionally actually be visible. *c.* As regards *consistence*, a splenic tumour generally feels firm and solid; now and then it gives a sensation of elasticity, but fluctuation is extremely rare. The surface is almost always smooth, but may be irregular. *d.* Another important character of a splenic tumour is its great *mobility*. As a rule it can be readily moved in all directions by manipulation; and it is more influenced by the act of respiration than any other tumour, being often felt below the ribs after a deep inspiration, when previously imperceptible. Posture also affects it markedly. *e.* Occasionally a *splenic murmur* is heard.

Some of the difficulties which are met with in recognizing enlargement of the spleen require notice. 1. The organ is often not sufficiently large to come below the margin of the thorax, and can then only be made out by percussion. 2. Even when of some size, it may be kept up by the costo-colic fold of peritoneum, or by adhesions at its upper end. 3. Adhesions may also prevent any mobility, and cause the tumour to become fixed. 4. The enlargement is sometimes so great as to obscure altogether the outline of the spleen, and the characters of its anterior margin, the latter then assuming a vertical direction. 5. Enlargement of other organs may conceal a splenic tumour. 6. Accumulation of flatus in the colon may interfere with its detection. 7. The principal morbid conditions for which enlarged spleen is likely to be mistaken, or *vice-versâ*, are cancer about the cardiac end of the stomach; enlarged left lobe of the liver; a tumour of the omentum; or an enlargement in connection with the left kidney or supra-renal capsule.

## B. SPECIAL DISEASES OF THE SPLEEN.

## I. CONGESTION OR HYPERÆMIA.

**ÆTIOLOGY.**—The spleen readily becomes congested, on account of its great vascularity, and the yielding nature of its capsule. After every meal it is more or less overloaded with blood. *Active* hyperæmia is commonly observed in acute febrile diseases, especially in typhoid, relapsing, and intermittent fevers, and to a less degree in typhus, erysipelas, pyæmia, puerperal fever, and acute tuberculosis. The condition is said to be occasionally vicarious of menstruation. Injury or morbid deposits may also cause it. *Mechanical* congestion of the spleen follows any obstruction affecting the portal circulation, either direct or secondary to chronic heart and lung-affections.

**ANATOMICAL CHARACTERS.**—The morbid characters presented by a recently congested spleen are enlargement, often considerable, the capsule being stretched and smooth; increase in weight; intense redness, of a dark hue; and diminution in consistence, the substance of the organ in some instances being quite pulpy or almost liquid. The amount of blood is much increased; red blood-cells are extremely abundant; and the splenic tissue appears to be augmented in some cases. After long-continued or repeated hyperæmia the spleen becomes permanently enlarged, hardened, and hypertrophied.

**SYMPTOMS.**—The only clinical sign of a congested spleen usually observed is that the organ is enlarged, but not as a rule to any great degree, and the size is liable to vary considerably. Occasionally it feels soft, but is generally tolerably firm. There is no spontaneous pain in most cases, but tenderness is common, and may be marked in acute congestion. Temporary general anæmia has been stated to be associated with extreme splenic congestion.

## II. HÆMORRHAGIC INFARCTION—SPLENITIS.

**ÆTIOLOGY AND PATHOLOGY.**—The spleen is one of the organs in which emboli most frequently lodge, giving rise to *hæmorrhagic infarctions*. Some pathologists are of opinion that these infarctions may also arise from the formation of local thrombi within the vessels of the organ. Occasionally considerable inflammatory action is excited, especially when the emboli have septic properties, as in cases of typhus fever or pyæmia, and this is the most frequent cause of *splenitis*. In rare cases inflammation of the spleen results from injury; and it has also been stated to arise from malaria, especially in certain tropical climates; or as an idiopathic affection.

**ANATOMICAL CHARACTERS.**—Infarctions in the spleen, as seen on section of the organ, are usually in the form of wedge-shaped masses with their bases towards the surface, often projecting somewhat; when situated deeper in the organ they are more or less rounded. They vary considerably in number and size. Originally each infarction is dark and firm, and is surrounded by a zone of congestion; in time, however, the ordinary changes take place, the colouring matter becoming altered and removed, until the mass assumes a yellowish-white colour. Fre-

quently caseous degeneration with ultimate absorption follows, a depressed cicatrix remaining; or calcification may take place. In pyæmia and allied affections the infarctions rapidly break down into a purulent fluid, at the same time the spleen being more or less inflamed and congested throughout. *Idiopathic* inflammation cannot at first be distinguished from mere congestion, the spleen being enlarged, very dark, and softened. One or more abscesses may form, which sometimes finally involve the entire organ, this being converted into a mere bag of pus. An abscess occasionally bursts externally; or into the peritoneum, stomach, or thorax. Rarely it becomes encapsuled, and undergoes curative changes, its fluid portion being absorbed, so that finally only a caseous material remains, which may calcify. The peritoneum corresponding to the affected part is often inflamed.

**SYMPTOMS.**—Very rarely can splenic embolism and its consequences be recognized during life, but it may be suspected if, along with some source of embolism, there should be rigors and pyrexia, with *local* signs indicating inflammation of the spleen, namely, pain and tenderness in the left hypochondrium; enlargement of the organ; and vomiting in many cases. A splenic abscess is scarcely ever diagnosed; it may possibly give rise to a fluctuating enlargement, or even burst externally. The process of suppuration is attended with hectic fever and rapid wasting. Should the abscess rupture internally, the usual signs of such an event will be observed.

### III. HYPERTROPHY.

**ÆTIOLOGY AND PATHOLOGY.**—By far the most important form of enlarged spleen is that which is due to *hypertrophy* of its tissue. This condition occurs mainly in three classes of cases, namely:—1. Those of *malarial* origin, where the hypertrophy results from long-continued or repeated active congestion, in connection with ague, or even after mere exposure to malarial influence. 2. Those in which it follows *chronic portal obstruction*, with consequent mechanical congestion of the spleen. 3. Those in which it is part of the special disease termed *leucocythæmia* or *leukæmia*. Sometimes the cause cannot be definitely made out. When splenic hypertrophy follows congestion it is supposed to be chiefly due to interference with the escape of the corpuscles out of the organ. Leucocythæmia will need to be separately discussed.

**ANATOMICAL CHARACTERS.**—In hypertrophy from hyperæmia the spleen is increased in size and weight, sometimes to a great degree, but retains its normal form; its consistence is increased; and a section appears pale and dry, sometimes grey, or presents black spots or patches due to pigment. The tissue is quite normal, but is increased in amount and condensed, the trabeculæ being also thickened and firm, appearing as white traversing lines.

**SYMPTOMS.**—Hypertrophy of the spleen may exist for a long time, and to a marked degree, without producing any evident disturbance, either local or general. In many instances, indeed, it can only be recognized by *physical examination*, which usually distinctly reveals the enlarged organ, and it may thus be accidentally discovered, the patient being unaware of its presence. Pressure-symptoms may be produced if the spleen attains a large size. In advanced cases, or in those of malarial origin, signs of more or less splenic cachexia are observed, and these may become very prominent, or the condition may end in true leucocythæmia.



## IV. LEUCOCYTHÆMIA—LEUKÆMIA.

**ÆTIOLOGY AND PATHOLOGY.**—Although it is convenient to discuss leucocythæmia in connection with affections of the spleen, it must not be regarded as merely a disease of this organ, although it is usually seriously implicated. One of the most prominent features of this complaint consists in certain changes in the blood, especially the presence of great excess of white corpuscles or leucocytes—hence the term *leucocythæmia*. Moreover, the lymphatic glands are not uncommonly affected; lymphatic deposits are sometimes found in various organs, as well as in connection with serous and mucous membranes; and the more recent researches have shown that morbid changes are often observed in the marrow of bones. Different forms of leucocythæmia are accordingly described, namely:—1. *Splenic leucocythæmia*, in which the spleen is primarily affected, but the absorbent glands and other structures may become secondarily involved. 2. *Lymphatic leucocythæmia*, where there is a primary enlargement of the glands, this being the complaint known as *lymphadenoma* or *Hodgkin's disease*, under which heading it will be subsequently described. 3. *Lymphatico-splenic leucocythæmia*, where the glands become implicated at an early period after the spleen, these cases being regarded as of a composite nature. 4. *Myelogenic leucocythæmia*, in which the marrow of bones is primarily involved, a rare but probable variety of the disease.

But little can be said definitely as to the *causation* of leucocythæmia. It has been regarded as the result of a special diathesis. Dr. Gowers found that in one-fourth of the cases analyzed by him there was a history either of ague or of residence in an ague district. The interval between the malarial affection and the disease varied from a few months to thirty years. This observer also states that in women the sexual processes appear to have a distinct influence. The disease is most frequent in them during the climacteric decade (40 to 50), and practically ceases when the menstrual epoch is over. In some cases it has commenced during pregnancy; in a larger number it has succeeded pregnancy. Among other causes to which leucocythæmia has been attributed in individual cases may be mentioned injury to the spleen; depressing influences, such as want of food, over-exertion, and especially depressing mental emotion; and previous diseases, particularly small-pox, typhoid fever, acute rheumatism, pneumonia, and syphilis. With regard to *predisposing causes*, age and sex seem alone important. Leucocythæmia may occur at any age, but is most common between 30 and 40. The disease is twice as frequent in men as in women (Gowers). Hereditary influence has only been traced in one or two instances.

The *pathology* of leucocythæmia is very uncertain, and at present little or nothing positive can be stated on the subject. It has been regarded as a primary disease of the blood, attended with the formation of excess of white blood-corpuscles, which afterwards accumulate in the spleen; but this theory is not borne out by facts. It seems probable that in most cases there is a primary change in the spleen; but it is believed that in rare instances the morbid condition may begin in the marrow of bones. According to recent researches, it appears that the red blood-corpuscles are the products of the development of the smaller lymphoid cells (globulins, hæmatoblasts). These are probably partly produced in the splenic pulp and in the marrow, from

pre-existing cells, and from the protoplasmic trabeculæ of the tissues; partly from the lymphatic glands and other true lymphatic structures. It is further supposed that the transformation of the lymphoid cells takes place largely in the splenic pulp and in the medulla of bones. In ordinary *splenic leucocythæmia* it is believed that, owing to a diseased condition of the pulp, this transformation does not take place, but the hæmatoblasts are changed into white corpuscles or leucocytes, which in part accumulate in the spleen, helping to enlarge it and to further alter its structure, in part pass into the circulation. The Malpighian follicles in the spleen are said not to be affected at the outset, but may become involved secondarily, along with the lymphatic glands, and with other organs in which lymphatic tissue is developed. This secondary change is partly attributed to the accumulation of leucocytes. The marrow is usually only affected secondarily, if at all, and is very rarely primarily involved. In *lymphatic leucocythæmia* the morbid change begins in the glands, and may also implicate the Malpighian follicles of the spleen from an early period. In *lymphatico-splenic leucocythæmia* both the splenic pulp and the Malpighian follicles are involved. Part of the increase of white corpuscles in the blood has been attributed to their proliferation, or to a new formation by the walls of the vessels.

**ANATOMICAL CHARACTERS.**—The *spleen* in leucocythæmia is more or less enlarged; ultimately it may attain enormous dimensions, and may weigh as much as 15 pounds. The organ generally retains its normal outline, the enlargement being uniform, but if it is very great, the shape tends to become more or less altered. As a rule it is abnormally firm, but not invariably, and it may be softened. Usually the capsule of the spleen is thickened, yellowish, and opaque in patches, due to local peritonitis; adhesions also frequently form with the diaphragm and other neighbouring structures. A section of the organ is smooth, and yields comparatively little blood. The appearance of the cut surface varies according to the variety of the disease. It is usually brownish-red or brownish-yellow, but presenting whitish lines, due to thickened trabeculæ; the Malpighian follicles are not then conspicuous, and they may not even be discoverable with the microscope. In other cases these structures are enlarged, and may form distinct growths, of some size, which rarely become softened. Hæmorrhagic infarctions or their remains are not uncommonly evident. Microscopically it is found that there is an increase in the splenic pulp, the trabeculæ being augmented, as well as the retiform tissue of nucleated fibres and cells, among which the lymphoid corpuscles lie. The Malpighian follicles may present signs of fatty or albuminoid degeneration. Minute octohedral crystals are found in abundance after death in many cases, of unknown nature. Chemically the spleen yields gluten, glycoll, hypoxanthus, xanthin, leucin, and tyrosin.

When the *lymphatic glands* are affected they become more or less enlarged, in some cases forming considerable tumours by their aggregation. As a rule, however, in cases of primary splenic leucocythæmia, the enlargement of individual glands is not great, these rarely exceeding a walnut in size. The affection of the glands is only exceptionally general, but different groups are involved in different cases, the mesenteric and cervical being the most frequent. They resemble in appearance and structure normal absorbent glands, being of a soft consistence, and presenting on section a smooth uniform surface, from which

a turbid fluid can be expressed. The colour is grey or reddish-white. Rarely caseating or suppurating spots are seen, or extravasations of blood. The cortical portion of the glands is much thickened ; and a microscopic examination only reveals that the normal elements of the gland-tissues are in excess.

With regard to *other organs and structures*, the most important changes associated with leucocythæmia are the presence of an adenoid or lymphoid growth, and distension of the capillaries with leucocytes. These changes have been found in connection with the liver, alimentary canal, kidneys, lungs and air-passages, heart, thymus and thyroid glands, tonsils, suprarenal capsules, skin, serous membranes, as the peritoneum, pleura, and cerebral membranes, and the retina. The liver is enlarged in the majority of cases, and may attain a very great size. It may be merely congested or fatty, but often presents disseminated lymphoid growths, greyish-white, generally interlobular, often surrounding branches of the portal vein. The kidneys may be the seat of granular or marked fatty degeneration, and similar changes may be observed in the heart. In connection with the alimentary canal, the gums are sometimes swollen or ulcerated ; but the chief seats of the lymphoid growth are the tonsils and the follicles of the tongue, with Peyer's and the solitary glands in the intestines, which may ulcerate. In the lungs adenoid collections occasionally break down and form cavities ; hæmorrhagic infarcts may also form. Effusions into the pleuræ, pericardium, and peritoneum are not uncommon in leucocythæmia. Hæmorrhages may also occur, as into the brain, retina, or other structures.

The changes in the *blood* in leucocythæmia are highly important. When seen in bulk it is paler than normal, and may become pink or greyish-red. In advanced cases it coagulates imperfectly, forming a grumous chocolate-coloured mass ; or it may separate into three layers, an irregular lymphatic layer forming between the red clot and the buffy coat. The specific gravity is much lowered, owing to an increase in the water, the average being 1042. The most important microscopic change in the blood, as determined by the *hemacytometer*, is a marked and persistent increase in the number of white corpuscles or leucocytes. A lesser degree and transient form of this increase is named *leucocytosis*, and is said by Virchow to accompany all forms of lymphatic excitement. In leucocythæmia the increase is progressive, and ultimately the number of white corpuscles may equal, or even exceed, that of the red. It has been proposed to regard as leucocythæmia only those cases in which the proportion of white to red corpuscles exceeds 1 to 20, but this rule cannot hold good. Smaller cells are also often visible, regarded as globulins or hæmatoblasts, especially when the lymphatic glands are involved, and Virchow distinguishes the splenic and lymphatic forms of leucocythæmia by the predominance respectively of white corpuscles or small uninucleated corpuscles, but Cornil and Ranvier, as well as other observers, maintain that such a distinction is not real. The white corpuscles are either of natural size, or some of them are considerably enlarged ; they appear granular, but on the addition of water they swell up, and exhibit from one to four nuclei. Some of them have undergone fatty degeneration. The red corpuscles are diminished in number, and hence the number of white corpuscles may appear greater than it really is. The total number of corpuscles is always below the normal, sometimes very considerably. Nucleated red corpuscles have been described by Klebs and Erb, which they consider as intermediate elements between white



and red corpuscles, but Cornil and Ranvier have failed to find them. These observers state that many white corpuscles, particularly the largest, contain very small spherical granules of an amber yellow colour, grouped round the nuclei; this appearance they explain by destruction of the red corpuscles, and absorption of the particles by the white corpuscles. It is said that the proportion of white corpuscles differs in blood taken from different parts of the body, being highest in that of the splenic vein. Usually the red corpuscles are normal in appearance, but sometimes they are pale. The proportion of iron in the blood is necessarily diminished. Other changes noticed are increase in fat and fibrin, the latter sometimes presenting a peculiar granular appearance; and in some instances the presence of abnormal ingredients, namely, albulin, mucin, hypoxanthin, lactic, formic, and acetic acids. After death in cases of leucocythæmia soft yellow clots are often found in the heart and great vessels, sometimes presenting an almost puriform appearance.

The changes in the *bones* which have been described in cases of leucocythæmia demand notice. Lymphatic growths may be found in these structures, even forming considerable masses. The medulla is grey or reddish-grey, and presents blood-corpuscles, lymphoid cells, or sometimes cells intermediate between white and red corpuscles. The vessels are diminished in number. All the bones may be thus affected, but especially those which have most spongy tissue, as the ribs or vertebræ. They may be normal in size, or enlarged. The compact osseous substance is sometimes thinned, or may even be perforated.

**SYMPTOMS.**—The essential clinical phenomena in leucocythæmia may be summed up as:—1. More or less intense *splenic cachexia*, which often attains a high grade. 2. In the majority of cases the *physical signs of enlarged spleen*, in some instances the organ being hypertrophied to such a degree as to lead to general enlargement of the abdomen. 3. In a certain proportion of cases *enlarged lymphatic glands*, either externally, within the cavities of the body, or in both situations; and occasionally signs of *enlarged liver*. 4. Sometimes evidences of *pressure* by the spleen on surrounding structures, especially the diaphragm, heart, and stomach. 5. Peculiar changes in the *blood*. It is quite sufficient to prick the finger so as to get just a drop of this fluid, and examine it microscopically, in order to observe the principal changes already described. The patient becomes weak and pale, presenting the usual symptoms of *anæmia* in a marked degree, and *œdema* may supervene. *Hæmorrhages* from various parts are common, *epistaxis* being often an early symptom, and bleeding may also occur from the alimentary canal, lungs, or other parts; while slight operations or injuries are liable to be followed by grave or fatal hæmorrhage. Among other prominent symptoms may be mentioned *palpitation*, *dyspnœa* of various kinds, *cough*, and different nervous phenomena, with disorders of the special senses. There may be signs of serous effusions. Changes in the gums may be noticed, and a "*leukæmic stomatitis*" has been described. *Jaundice* is rare, but a yellowish tint of the skin is not uncommon, or it may be pigmented. As a rule no subjective sensations are complained of in the abdomen, except a sense of weight and fulness, but tenderness or transitory pains may be felt. Digestive derangements are of frequent occurrence; and vomiting and diarrhœa are often prominent symptoms. Usually *pyrexia* is absent in the earlier stages of leucocythæmia, but there may be some irregular febrile disturbance, and later on the temperature is often raised persistently. As a rule it is highest in cases which run a rapid

course. The temperature may reach from  $101^{\circ}$  to  $104^{\circ}$  in the evening, and there is generally a greater or less morning fall. Sometimes considerable pyrexia occurs at irregular periods. Excessive sweating is common. The urine is usually strongly acid, and of high specific gravity; uric acid is in excess; and hypoxanthin, formic, and lactic acids have been found in the urine. Albuminuria is rarely present, except with renal disease. Menstruation is generally stopped. The ophthalmoscope may reveal changes in the retina, especially hæmorrhages; and yellowish or white spots, due to collections of lymphoid cells. Leucocythæmia is generally a chronic disease, but its duration is said to range from six months to seven years. Death may take place gradually from asthenia and exhaustion, frequently preceded by delirium, stupor, and coma, or by syncope; or more speedily as the result of hæmorrhage, diarrhœa, or complications. The most frequent form of fatal hæmorrhage is epistaxis. Internal hæmorrhages may also cause death, especially cerebral. The most important complications are pleural or pericardial effusions; pneumonia or bronchitis; venous thrombosis, which, it is said, may take place in the penis, and lead to persistent erections; renal disease; erysipelas; and boils.

## V. OTHER MORBID CONDITIONS.

1. **Albuminoid Disease.**—For the *ætiology, morbid anatomy, and constitutional symptoms* of this condition reference must be made to the general account already given. All that need be said here is, that the albuminoid deposit in the spleen is in some cases limited to the Malpighian corpuscles, producing the appearance known as the *sago-spleen*, in which translucent granules are observed, resembling boiled sago. *Clinically* enlargement of the spleen from albuminoid disease is recognized by its very hard and dense consistence; and by its steady growth, the organ finally reaching extreme dimensions in some cases. Other organs are always involved; while there is some constitutional condition present with which albuminoid disease is associated.

2. **Syphilitic Disease.**—In rare instances of congenital syphilis the spleen is much enlarged and firm. In one case which came under my notice the organ reached nearly to the crest of the ilium, presented a firm and sharp margin, and was freely movable.

3. **Cancer** of the spleen is almost a curiosity. It occurs in the form of nodules or masses of encephaloid, and is always secondary. During life the enlargement is recognized by its irregular form, and nodular character. Usually pain and tenderness are complained of. Other organs are always implicated.

4. **Hydatid tumour** has been in rare instances met with in the spleen, the liver being affected at the same time. It may give rise to a prominent tumour, having the usual semi-globular shape and fluctuating sensation of a hydatid-cyst.

5. **Tubercle** in the spleen is chiefly met with as a part of acute miliary tuberculosis. In rare instances it has been observed in cases of chronic phthisis. This condition cannot be recognized clinically.

6. **Rickets** is believed by some to produce special changes in the spleen, as described under that disease.

7. The spleen is often **shrunk** and **atrophied**, but this leads to no obvious ill effects.

## C. GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. DIAGNOSIS.—Practically it is only by *physical examination* that diseases of the spleen can usually be positively recognized. The chief difficulties in the diagnosis of splenic enlargement have already been indicated. The *general symptoms* aid the diagnosis materially in advanced cases. The *previous history* also helps in some instances, especially if it reveals exposure to malarial influence, or the previous occurrence of attacks of ague. Should there be persistent portal obstruction, more or less enlargement of the spleen is a necessary consequence.

Examination of the blood is of essential importance in the diagnosis of leucocythæmia, and must be repeated again and again in doubtful cases. This examination will also help to determine whether a case is one of true leucocythæmia or of Hodgkin's disease, when enlargement of the glands occurs at an early period.

2. PROGNOSIS.—*Acute* affections of the spleen are rarely dangerous in themselves. *Chronic* affections are usually slow in their progress, except in the case of malignant disease, and if there is mere hypertrophy the health is often not disturbed for a long time. Treatment has usually but little effect in reducing this hypertrophy. *Leucocythæmia* has been considered an incurable disease, but more recent experience has proved that it may be greatly benefited if a case comes under treatment at an early period. The prognosis is worse if there are signs of marked organic changes in the blood-forming organs; and also in proportion to the increase of white corpuscles, and the diminution of red corpuscles in the blood.

3. TREATMENT.—No special interference is needed in *acute* forms of splenic disease, unless an abscess should form and be recognized, which must be treated in the usual way. A case has been recorded in which  $8\frac{1}{2}$  ounces of a dark grumous fluid were removed by aspiration from a spleen which was the seat of softening from acute inflammation. Quinine has a marked influence in reducing *malarial congestion* and its consequences, even after it has existed for some time, and such a condition ought to receive as early attention as possible, and be treated persistently, so as to prevent future ill-effects. When there is *mechanical congestion*, any impediment in connection with the portal circulation must be removed, if possible; but if this is not practicable, *saline purgatives* act beneficially, by relieving the vessels. In *hypertrophy* one of the chief objects of treatment is to improve the general health, and the condition of the blood, by the administration of iron, mineral acids, quinine, arsenic, and similar remedies; as well as by a nutritious diet, change of air, and attention to hygienic measures. Iodides and bromides, as well as mercurials, have been supposed to reduce the size of the spleen, but in my experience they have been of no use whatever. Cases of *leucocythæmia* have been decidedly benefited by the use of phosphorus. In a case under my own care the administration of this drug seemed to have a marked effect in reducing the size of the spleen and of enlarged glands, and in diminishing the number of white corpuscles in the blood. It tends, however, to produce fatty changes in organs, and other untoward effects. Cod-liver oil is sometimes useful. Friction over the corresponding region with iodide of mercury ointment has been recommended with the view of reducing the size of the spleen, but is of no avail. The use of voltaic electricity has been found to have a marked



influence in this direction, and is strongly recommended by Dr. Gowers, who affirms that it contracts the spleen, expels retained leucocytes, and perhaps stimulates directly its functional action. Injection of ergotine has also been employed, it is said, with advantage. As a last resource, extirpation of the spleen has been practised. This may be permissible in cases of simple enlarged spleen, but in leucocythæmia is invariably fatal, on account of hæmorrhage. Various symptoms must be treated as they arise.

### CHAPTER LIII.

## DISEASES OF THE PANCREAS.

### A. CLINICAL CHARACTERS.

1. **PANCREATIC** affections are frequently attended with **painful sensations**, which are described as lying deep in the abdomen, a little below the epigastrium. The pain may shoot in various directions, and occasionally comes on in violent paroxysms, resembling those of hepatic colic. In some cases there is deep tenderness.

2. Important symptoms are believed to arise from changes in the quantity or quality of the **pancreatic secretion**. When formed *in excess*, being at the same time usually of an irritable quality, this secretion is supposed by some to be the cause of a form of pyrosis, attended with the discharge of a viscid, slimy fluid; as well as of diarrhœa, the stools containing a tenacious material, or presenting sometimes dysenteric characters. On the other hand, *deficiency* or *absence* of pancreatic juice from the alimentary canal, whether arising from changes in the gland-tissue, or obstruction in connection with the duct; and *abnormal quality* of the secretion, have been considered to give rise to a characteristic phenomenon, namely, the passage of a large amount of fatty or oily matter in the stools, which separates from the general mass of the fæces. Frequently there is constipation at the same time, the fæces being dry and hard. Other digestive derangements are common, and may be partly due to the absence of pancreatic juice from the bowels.

3. **Pressure** upon or **irritation** of neighbouring structures is often a cause of prominent symptoms in pancreatic disease, especially jaundice; vomiting, eructations, and other gastric disturbances; and aortic pulsation or murmur. The neighbouring veins are sometimes obstructed, especially the portal, with the usual consequences. Pain is also partly or entirely due to this cause in certain cases, resulting from pressure on the nerves in the vicinity, or on the vertebrae, the latter being occasionally eroded.

4. **Changes in the urine**.—It has been stated that in some cases of pancreatic disease fat has been present in the urine, either in the form of oil-globules, or of a greasy substance, which becomes like butter on cooling. Glycosuria has also been found associated with morbid conditions of the pancreas, but probably this depends on implication of the solar plexus.

5. **Physical examination** may reveal certain morbid conditions of the pancreas, but it requires to be performed very thoroughly, and in many cases repeatedly, the stomach and colon being empty, before a satisfactory conclusion can be arrived at. Only palpation can be said to be of any positive service. The healthy pancreas can now and then be felt on making deep pressure, in very thin persons with loose abdominal walls, especially if the spine is somewhat curved forwards; this is more frequently the case when the organ is enlarged and hardened. It is, however, in the detection of a *tumour of the head of the pancreas* that physical examination is of most value. This is distinguished by the following characters:—*a.* It is situated deeply at the back of the abdomen, in the region of the pancreas. *b.* The dimensions are always small; and the shape generally more or less rounded. *c.* The tumour is quite fixed. *d.* It feels dense and hard. It must be mentioned that marked pulsation and bruit may result from pressure of the pancreas upon the aorta. In rare instances *cysts* of the pancreas have reached such a size as to be obvious to physical examination as a deep-seated tumour, rounded and smooth, soft or indistinctly fluctuating.

6. Pancreatic disease is often attended with **general symptoms**, namely, extreme emaciation, anæmia, debility, and mental depression, due to interference with nutrition and other causes.

#### B. SPECIAL DISEASES.

The special diseases of the pancreas need but a brief consideration, and some of them only require enumeration.

1. **Acute Pancreatitis**.—Acute inflammation of the pancreas is very rare. It is said to be characterized anatomically by hyperæmia; swelling; induration or softening; and exudation into the cellular tissue and upon the surface, occasionally ending in purulent infiltration or the formation of abscesses. The last event is said to be not uncommon as the result of metastasis from the salivary glands and testis. Very rarely the inflammation ends in gangrene. The *symptoms* are described as dull, deep-seated pain in the region of the pancreas; nausea and vomiting of a viscid liquid; thirst; constipation; and some degree of pyrexia. Rupture of an abscess may give rise to serious symptoms.

2. The following morbid conditions may be mentioned together, namely, (i.) **Anæmia**, or **hyperæmia**. (ii.) **Hæmorrhage**. (iii.) So-called **Hypertrophy**, which generally involves the entire gland, and is the result of *chronic inflammation*, or of long-continued *mechanical congestion* from portal obstruction, the pancreas being enlarged and hardened, and the interstitial tissue being increased. (iv.) **Atrophy**, usually associated either with senile changes; some kind of cachexia; local disease of vessels; or pressure upon the organ as the result of surrounding disease. (v.) **Induration or softening**, with or without hypertrophy or atrophy. (vi.) **Fatty infiltration and degeneration**. If these conditions give rise to any symptoms at all, they are those indicative of deficiency or abnormal quality of the pancreatic secretion. A hypertrophied pancreas may be felt in some cases; and occasionally it gives rise to pressure-symptoms.

3. Not uncommonly **calculi** form in the pancreatic duct, which may be in large numbers and of some size. They interfere with the escape of the secretion. I am not aware that their passage causes any symptoms. The branches of the pancreatic duct are sometimes dilated into cysts,

which rarely attain a large size. This may also arise from pressure upon the duct from without, at or near its orifice; or from affections of the gland itself.

4. The most important disease of the pancreas is **scirrhus of the head** of this organ. There is a difference of opinion as to the nature of the morbid condition thus named, some pathologists regarding it as *scirrhus cancer*; others considering that it is merely due to *fibroid changes* resulting from *chronic inflammation*, and they affirm that the pancreas is peculiarly free from cancer, escaping often even when the disease involves all the surrounding structures. I have had the opportunity of observing several cases of this disease, and of making a *post-mortem* examination in four of these cases, in which certainly the affected portion of the pancreas presented well-marked general and microscopic characters indicative of scirrhus cancer. The mass varies in size, but does not attain large dimensions; it has an extremely hard and dense consistence, and a whitish section. It frequently becomes adherent to, or even involves the duodenum, which may be ulcerated and greatly narrowed. It may also form adhesions with other structures, to which the disease may subsequently extend. The pancreatic and common bile-ducts as a rule become obstructed. The latter is usually supposed to be closed, owing to pressure being exerted upon it by the enlarged pancreas, but this effect is probably more frequently due to contraction about the orifice or in the course of the duct, from changes in its own tissues, jaundice and its accompanying phenomena necessarily resulting. The body of the pancreas is usually enlarged; sometimes it is atrophied. Now and then serious disorganization of neighbouring parts is occasioned, leading to erosion of the vertebræ, perforation of the diaphragm, or the opening of a large vessel.

But little is known about the *ætiology* of this disease. Generally it occurs in elderly persons, but one of the most marked cases I have met with was in a young man aged twenty-three. In only one instance which has come under my notice was there any history of intemperance.

**SYMPTOMS.**—The clinical history of *scirrhus of the pancreas* is decidedly indefinite and uncertain. In general terms the clinical phenomena may be stated as deep pain in the region of the pancreas, aching, gnawing, or lancinating in character, or sometimes attended with a sense of burning or tightness, in some cases greatly increased paroxysmally, and also frequently intensified by food, coughing, deep breathing, movement, or the supine position; deep tenderness; nausea and vomiting, in some cases of a severe character; various digestive disturbances, the tongue, however, being often quite clean; jaundice, frequently intense; the passage of much fat in the stools, the bowels being usually constipated; the detection of a tumour having the characters already described; accompanied with great general wasting, anæmia, debility, and lowness of spirits. As showing the irregular and ill-defined clinical history of cases of this disease, I may state from my own observation, that there may be no pain or tenderness from first to last; that symptoms due to biliary obstruction may be the only prominent phenomena throughout; that it may be impossible to detect any tumour; and certainly that excess of fat in the stools is by no means always observed.

5. Among exceedingly rare morbid deposits which have been found in the pancreas are mentioned **encephaloid cancer, colloid, melanosis, sarcoma, tubercle, and syphilitic formations.**



DIAGNOSIS.—Only *scirrhus of the head of the pancreas* can be diagnosed with any approach to certainty, and in many cases it is exceedingly difficult to arrive at any positive conclusion, at all events for some time. The chief diseases for which this condition is liable to be mistaken are affections of the stomach, especially about the pylorus; of the duodenum; or of the liver. The paroxysms of pain may closely resemble those associated with the passage of a gall-stone. Occasionally, by pressing on the abdominal aorta, scirrhus of the pancreas gives rise to pulsation and bruit, simulating an aneurism. Whenever any of the symptoms above mentioned are complained of, and especially jaundice coming on without any obvious cause, pancreatic disease should always be borne in mind. I believe that it not uncommonly escapes recognition simply because it is never thought of. An important step towards a correct diagnosis consists in excluding as far as possible affections of all neighbouring structures. It must be remembered that the liver is liable to be enlarged, as the result of obstruction of its duct associated with pancreatic disease. *Physical examination* is of essential value in diagnosis, and in doubtful and obscure cases it should be thoroughly carried out again and again, by which means a satisfactory conclusion may in some instances be arrived at in course of time.

PROGNOSIS is necessarily serious in cases of scirrhus of the pancreas, the disease being fatal, and seldom of long duration.

TREATMENT must be entirely *symptomatic*, directed especially against pain, vomiting, jaundice, loss of flesh and strength, anæmia, and debility. The use of *digestants*, especially *liquor pancreaticus*, would probably be of much service.

## CHAPTER LIV.

### DISEASE OF THE SUPRA-RENAL CAPSULES— ADDISON'S DISEASE.

ÆTIOLOGY AND PATHOLOGY.—Dr. Addison first drew attention to a series of symptoms which he believed were associated with disease of the supra-renal bodies, one of the most prominent being a peculiar discoloration or *bronzing of the skin*. Since his time the subject has been investigated by many observers, especially by Dr. Greenhow, to whose writings the reader is referred for complete and detailed information.

The first question to be determined is, whether any relation exists between the phenomena of so-called *Addison's disease* and any morbid condition of the *supra-renal capsules*? Greenhow maintains that there is such a relationship, but others deny this. With regard to the *bronzing of the skin*, which, however, is by no means the most important or an essential symptom of Addison's disease, this has been described as being present in cases where there was no supra-renal mischief; but Greenhow affirms that the discoloration in these instances was different from true bronzing. On the other hand, supra-renal disease has been frequently noticed where there was no bronzing, which might be accounted

for in some instances by the fact that this symptom appears at a comparatively late period, and that the progress of the complaint may be so rapid as to terminate in death before the discoloration could be developed. But, further, this leads to the question of the *nature* of the lesion of the supra-renal capsules in Addison's disease. Some suppose that its phenomena may be due to any morbid condition of these organs; but Greenhow holds that they are only observed in connection with a *special* lesion, which will be presently described. As to the *mode* in which disease of the supra-renal capsules produces these effects, there is strong evidence to prove that it is not through any mere destruction of their tissues, and abolition of their functions, whatever these may be. These organs have a large supply of nerves, which are intimately connected with the trunk of the sympathetic in the abdomen, as well as with the phrenic and pneumogastric nerves, and through these with the cerebro-spinal centres. The morbid changes which affect the supra-renal capsules also tend to involve the nerves in their vicinity, and may even extend so far as to implicate the semilunar ganglia and solar plexus. It seems highly probable that the phenomena of Addison's disease are attributable to this implication of such important nerves, which are first irritated, and subsequently become atrophied and destroyed. This is borne out by the fact that bronzing of the skin has been found in connection with enlargement of the retro-peritoneal absorbent glands, which surrounded and compressed the solar plexus, the supra-renal capsules being perfectly healthy. The nerve-lesions have been considered by some observers as arising *primarily*, and as being altogether independent of supra-renal mischief.

With respect to the *exciting cause* of the special supra-renal lesion, Greenhow states that it is frequently due to the extension of inflammation from diseased or injured adjacent parts. It has also been referred in some instances to a severe strain, blow, or physical shock, usually in the back; over-exertion; nervous shock, grief, or anxiety; and intermittent fever.

There are some important *predisposing causes* of Addison's disease. It is much more frequent among males; is found almost exclusively in those employed in active manual labour, and especially in connection with those occupations which entail exposure to bodily injury from accident or over-exertion; and is almost confined to the laborious periods of life. There may be a predisposing *constitutional condition* in some instances.

ANATOMICAL CHARACTERS.—The supra-renal bodies may be the seat of the following morbid changes:—1. *Acute inflammation*, ending in supuration. 2. *Tubercle*. 3. *Cancer*, always secondary, and usually of the encephaloid variety. 4. *Albuminoid disease*. 5. *Fibroid degeneration*, with hardening. 6. *Fatty degeneration*. 7. *Atrophy*. 8. *Hæmorrhage*. 9. *Peculiar alterations* associated with *Addison's disease*. Only the last need be described, and the changes observed are supposed to be the result of a *chronic inflammatory* process, the organs becoming infiltrated with an exudation of a low type, which is converted into a firm fibrous material, and this undergoes degenerative changes, along with the tissues of the supra-renal capsules, which it invades and destroys. The affected organs are usually enlarged, firm, and nodulated; though in rare cases they are normal or diminished in size. In the early stage of the disease they are invaded by a softish, semi-translucent, greyish or greenish-grey, apparently homogeneous substance,

which on exposure to the air assumes a pinkish hue. This becomes firmer, and undergoes caseous degeneration, giving rise to yellowish, opaque, cheesy nodules; or, not uncommonly, forming a creamy or purulent-looking fluid, varying in thickness, and occupying either one large cavity in the centre of the capsule, or, more frequently, several small cavities. Sometimes calcification follows, cretaceous granules or small masses being formed, or a putty-like material, or finally a dry chalky mass. The grey material and the products of degeneration are always found associated together, though in very variable proportions, and gradations may be seen from one to the other. Under the microscope the former is observed to consist of a fibrillated stroma containing numerous lymphoid corpuscles; while the caseous masses are made up of altered cells, nuclei, granular matter, and fat. In many cases there is great thickening of the covering of the capsules, with extensive proliferation of the surrounding cellular tissue, and the formation of firm adhesions to adjacent organs; the nerves become thus invested in a dense indurated tissue, and their fibrous investment has also been found hypertrophied.

Other morbid appearances have been described in different cases of Addison's disease, including enlargement of the neighbouring absorbent glands, which are either normal in structure, or firm, glistening, pale, and in process of caseation; enlargement of the solitary glands of the small intestines, and sometimes of the large; mammillation, small ecchymoses, superficial erosions, or small ulcers in the stomach; atrophy of the mucous coat of the alimentary canal, with degeneration of its glands; and enlargement of the spleen, which may be considerable, the organ being usually dark-coloured and soft. Dr. Greenhow states that the composition of the blood does not undergo any important alteration in uncomplicated cases of this complaint.

**SYMPTOMS.**—One of the most prominent clinical phenomena of Addison's disease consists in a *peculiar cachexia*, which sets in gradually and indefinitely without any obvious cause, characterized by increasing muscular debility, languor, and indisposition for any bodily or mental effort, at last amounting to extreme prostration; an aspect of listlessness and depression; marked anæmia, the sclerotics being pearly-white; wasting, but not to any degree, nor is it always observed, while there is often a peculiar tendency to the formation of fat, the subcutaneous fat being in some cases very abundant; remarkable feebleness of the heart's action, the pulse becoming very soft, usually weak, and compressible, there being also a tendency to giddiness and faintness, sometimes amounting to prolonged attacks of syncope, and to palpitation on exertion, with breathlessness. Another characteristic feature is a gradual *discoloration of the skin*, which assumes the so-called *bronzed* appearance. This appears at very variable periods in different cases, and is due mainly to the presence of yellowish-brown pigment-granules in the *rete mucosum*, or occasionally of pigment-cells. Traces of pigment may also be found in the superficial layers of the epidermis, with pigment granules here and there in the cutis. The exact hue varies, and it becomes darker by degrees. It often resembles that of a mulatto, but may be simply dingy or smoky-brown, yellowish-brown, greenish-brown, greyish-black, or almost black. It extends all over the body, but is never uniform throughout, usually commencing and being most marked over exposed parts, such as the face and neck; on the upper extremities; in the axillæ; and about the penis, scrotum, and navel. It shades off



gradually, but where the skin has been injured or irritated, this part becomes much darker and presents defined margins. The palms and soles sometimes exhibit spots of pigment. The *mucous membranes* are also discoloured, the lips in some instances assuming a mulberry hue; or irregular and ill-defined spots and patches of pigment being observed upon them, as well as on the inside of the cheeks and on the gums, with dark streaks opposite the angles of the mouth. A peculiar pigment has also been described in the *structures of the eye*, as seen with the ophthalmoscope, but the conjunctivæ always remain normal. In addition to these symptoms there is usually more or less *pain in the epigastrium*, in some cases extremely severe; and *irritability of the stomach*, with nausea, retching, or vomiting, which may be urgent and irrepressible. Other digestive disorders are also common, appetite being lost, and obstinate diarrhœa sometimes sets in, though constipation is the rule. The tongue is usually red and moist. Pain in the loins is often complained of. Frequently one or both hypochondria feel tender; and Dr. Greenhow has noticed sometimes a rigidity of the abdominal muscles, as if they were instinctively contracted to protect deep-seated parts from pressure.

The *course* of Addison's disease is slow and chronic as a rule, but it is often subject to remarkable remissions, with improvement in the symptoms, and subsequent exacerbations. In exceptional instances the progress is acute and rapid; or the disease may be latent for a long time, and then run a very rapid course. Death generally results from gradual asthenia, there being towards the close frequent sighing or yawning, with persistent hiccup. The mind is in many cases clear to the last, but the patient may be drowsy or semi-comatose, or grave nervous phenomena may arise. The temperature is usually low throughout, but towards the close it falls considerably, the skin being cool or cold. The urine is often diminished; of low specific gravity; and deficient in solids.

DIAGNOSIS.—It is only necessary to mention that should symptoms of failing health and cachexia appear, without any evident organic mischief to account for this, Addison's disease ought to be remembered. When the bronzing appears, there should be no doubt respecting the nature of the complaint.

PROGNOSIS is very grave, the disease always ending fatally, but the duration may be very prolonged.

TREATMENT.—All that can be done is to promote general health and strength by means of a highly nutritious diet; by the administration of *tonics*, especially quinine, tincture of steel or syrup of the phosphate of iron, strychnia, and cod-liver oil; by attention to hygienic measures; and by maintaining the alimentary canal in good order. Rest, and the avoidance of all bodily and mental excitement, are also important elements in treatment. Symptoms must be attended to as they arise.

---

## CHAPTER LV.

## ABDOMINAL ANEURISM.

THE most important form of abdominal aneurism which comes under the notice of the physician is that which is connected with the aorta, but an aneurism may be found on the celiac axis or either of its branches, especially the hepatic; on either of the mesenteric or renal arteries; or on one of the iliac vessels. For the *pathology* and *anatomical characters* of this condition, reference must be made to THORACIC ANEURISM.

**SYMPTOMS AND SIGNS.**—In many cases the only clinical indications of abdominal aneurism consist in the detection of a *tumour*, presenting the usual *physical characters* of an aneurism. Often, however, there are signs of pressure on surrounding structures; local morbid sensations; and evidences of serious constitutional disturbance. In some instances such phenomena are alone present, there being no physical signs of an aneurism, or only such as are very obscure. The *physical characters* of an abdominal aneurismal tumour are as follows:—1. It is usually seated in some part of the course of the aorta, but frequently projects more to one side than the other, especially towards the left. Of course an aneurism may occupy other regions, corresponding to the particular vessel affected. 2. As a rule the shape is more or less rounded; the surface is smooth; and the tumour yields somewhat on being compressed. 3. Almost always the aneurism is quite fixed and immovable, being unaffected by respiratory movements, though if it is very large it may interfere with these movements. 4. One of the most important characters is the presence of more or less *pulsation*, synchronous with the cardiac systole usually, but sometimes also diastolic; distinctly expansile; tending laterally as well as forwards, and not uncommonly more towards one side than the other; and occasionally attended with a thrill. 5. Percussion reveals *dulness*, corresponding to the extent of the tumour; with a sense of much *resistance*. 6. In many cases there is a *systolic murmur*, which is sometimes very loud and harsh, but it is by no means always heard, or may be very slight, or more like an arterial sound; and occasionally a murmur is seated beyond the aneurism. There is never any diastolic bruit. The murmur is often influenced considerably by posture and pressure.

There are a few points of practical importance which demand notice. 1. The signs of an abdominal aneurism may be most evident in the back, and it is always essential to make careful examination from this aspect should the disease be suspected. Sometimes there is no obvious sign, except a murmur in this region. 2. There is no relation between the size of an aneurism and the degree of pulsation, or the loudness of a murmur. 3. Occasionally the tumour is movable; and not uncommonly both pulsation and murmur are considerably influenced by posture, it being on this account necessary to examine the patient in different positions. It is important, however, to observe that the impulse does not disappear when the patient is placed in a kneeling attitude, sup-

ported by the hands. 4. The physical signs may change considerably during the progress of any particular case of abdominal aneurism.

The *pressure-symptoms* will vary with the situation of the aneurism. Among the most common are neuralgic pains, sometimes extremely severe, and shooting in different directions, originating in pressure upon nerves, such pressure also occasionally causing permanent contraction of the flexors of the hip-joint; deep gnawing pain, from erosion of the vertebræ; and anasarca of one or both legs, with distension of the superficial veins, due to pressure on one of the iliac veins or on the vena cava. In some cases micturition is affected at times; and albuminuria may be induced as the result of pressure on the renal veins. Wasting of the testis has been noticed, in consequence of obliteration of the spermatic artery. Aneurism of the *hepatic artery* must be borne in mind as a possible cause of jaundice and ascites, in consequence of producing pressure upon the neighbouring duct and portal vein.

In some instances of abdominal aneurism a subjective feeling of uncomfortable pulsation is experienced. The alimentary canal is often out of order, and I have known obstinate constipation to be the one prominent symptom complained of in a case of aneurism of the abdominal aorta. Patients suffering from this disease frequently look well, and their general condition is often satisfactory; but sometimes they present a very peculiar aspect, indicating profound illness with anæmia, even when there are no distinct physical signs of the aneurism.

DIAGNOSIS.—The chief conditions which may simulate abdominal aneurism are:—1. Simple aortic pulsation. 2. The pancreas or a solid tumour transmitting an impulse from the aorta; or giving rise to a murmur by pressing upon the vessel. 3. A fluid accumulation, such as hepatic abscess or hydatid tumour, receiving and communicating an impulse from the aorta. The differential diagnosis must be founded on a careful consideration of the case in all its details, as regards history, symptoms, and physical signs; but it is important to notice that in all the conditions just mentioned which simulate aneurism, any pulsation observed is but rarely expansile, while both it and any murmur which may be audible generally disappear if the patient is placed in a kneeling attitude, supported on the hands. The diagnosis from mere *aortic pulsation* requires a few words of special comment. The chief facts in favour of this condition are as follows:—1. The pulsation is generally seated in the epigastrium. 2. It is observed most commonly either in highly nervous and anæmic persons, especially women; in very thin individuals; or in those who suffer much from chronic dyspepsia. 3. There are no signs of pressure; nor is there any pain or tenderness as a rule. 4. The impulse is scarcely ever expansile and lateral, but merely tends in a forward direction, and is never attended with a thrill; there is no increase in dulness, or any evident tumour; and if a murmur is present, it is soft and blowing or whiffing in quality, but never harsh or loud. Some cases are difficult to diagnose with certainty, and then their progress must be watched, and the effects of treatment observed.

It must not be forgotten that an abdominal aneurism may exist without giving rise to any physical signs. Occasionally also it exhibits the characters of a solid tumour, presenting neither pulsation nor bruit. If obscure abdominal symptoms are complained of, particularly deep pain near the spine; and especially if there are at the same time indications that the constitution is gravely disturbed, aneurism should be thought



of, and careful *physical examination* carried out repeatedly, over the back as well as in front of the abdomen.

**TREATMENT.**—In addition to what has been previously stated regarding the treatment of internal aneurism (*see* page 558), it is necessary to allude to a special method introduced for the cure of aneurism of the abdominal aorta, namely, the *rapid-pressure* treatment, first employed by Dr. William Murray, of Newcastle-on-Tyne. This plan consists in keeping the patient well under chloroform, applying a tourniquet over the aorta above the tumour, and maintaining steady and constant pressure until all pulsation has ceased in the aneurism on removing the tourniquet. The blood coagulates in the sac, and afterwards collateral circulation is set up. The results of this treatment have certainly been such as to commend its adoption in appropriate cases, if other measures do not appear to be followed by good effects. If the aneurism is situated high up, distal pressure may possibly be of service. Tuffnell's method has proved efficacious in the treatment of some cases of abdominal aneurism.

Pain is a symptom often calling for interference in cases of this disease, and it is best relieved by subcutaneous injection of morphia. Posture may influence considerably the severity of the pain. It is highly important to attend to the state of the digestive organs, and to keep the bowels acting regularly by means of mild *aperients*. A belladonna plaster may be worn constantly over the aneurism.

## CHAPTER LVI.

### DISEASES OF THE URINARY ORGANS.

#### A. CLINICAL CHARACTERS.

1. **THE morbid sensations** connected with the urinary organs may be referred to one or both lumbar regions; to the course of the ureters; to the hypogastrium; or to some part of the urethra. They chiefly include different kinds of pain, tenderness, uneasiness, a sense of fulness or tension, heat or burning along the urethra, and itching or tickling at the end of the penis. With respect to pain, it is important to ascertain whether it is increased by movement of the body, especially by sudden jolts, as after walking, jumping, riding, or driving; if it is affected by the act of micturition, being either relieved or intensified, or even only complained of during or after this act; and if it is influenced by any articles of food or drink. Not uncommonly a sympathetic pain is felt running along the spermatic cord to the testis; and this organ may be retracted.

2. **The act of micturition** is frequently disturbed. The principal deviations are a too frequent or almost constant desire to pass water, sometimes coming on suddenly, so that the patient cannot retain the urine for an instant, or, on the other hand, being combined with more or less dysuria or stranguary; simple *dysuria*, or difficulty of micturition, even to complete retention; and *incontinence*, the urine coming away

involuntarily, either constantly or only at times, and especially at night during sleep. The stream of urine may present abnormal characters. The amount passed may also vary greatly from the standard of health, being either diminished more or less to actual suppression—*oliguria*, and *anuria* or *ischuria*; or increased—*polyuria*.

3. Important symptoms which are frequently associated with certain urinary affections result from the **abnormal state of the blood** which they induce, of which the most prominent are *dropsy*, and the phenomena grouped under the term *uræmia*. The latter condition will be presently discussed in detail.

4. When the kidney is enlarged, it occasionally gives rise to symptoms by causing **pressure** upon adjoining structures.

5. **Rupture** of any portion of the urinary apparatus will occasion serious consequences, especially when this event is followed by extravasation of urine.

## B. SPECIAL EXAMINATION.

*Special examination* in connection with the urinary organs includes:—I. **Examination of the urine**. II. **Investigation for renal tumour**. III. **Examination directed to the bladder and urethra**, by external methods; by the use of the catheter, sound, or endoscope; and through the rectum or vagina. For an account of the special examination of the bladder and urethra reference must be made to surgical treatises, but it will be desirable to point out in this work the physical signs of a distended bladder. It should also be mentioned that in cases of renal disease it is of considerable importance to examine the **heart and arteries**, and to make use of the **ophthalmoscope**.

## I. EXAMINATION OF THE URINE.

*Examination of the urine* is a matter of the deepest importance, and is even at the present day but too much neglected in ordinary practice. It gives valuable information in other affections besides those directly associated with the urinary organs, and in the succeeding remarks it is proposed to give a concise outline of the mode in which the clinical investigation of this excretion must be conducted, much of which has been condensed from Dr. William Roberts's most valuable work. It need scarcely be remarked that a previous knowledge is requisite of the characters of the urine in health, as well as of its chemical composition, with the average proportion of its chief constituents, and the main physiological variations to which they are liable; the changes which the urine undergoes on standing after its discharge must also be remembered.

### 1. General Examination.

The first thing to be done with any specimen of urine is to observe its *physical characters*, including colour and general aspect; degree of clearness or turbidity; consistence; characters of the froth on shaking; odour; specific gravity; and presence or absence of any deposit. It is often of much importance to measure the quantity passed in the twenty-four hours; and when taking the specific gravity, or making quantitative analyses, a specimen from a mixture of the whole of this urine

should be employed. Then the *reaction* should be taken, this being done as soon as possible after the urine has been passed, by means of turmeric, and blue, green, or violet-tinted litmus papers. Should the urine be alkaline, it is requisite to determine whether this is due to fixed alkali or to ammonia, which is proved by drying the test-paper in the open air, when, if the alkalinity depends on ammonia, this evaporates and the paper is restored to its original colour. Further, it is very important, should the urine be ammoniacal, to ascertain whether it is discharged in this condition, or if the presence of ammonia results from subsequent decomposition, and how soon this substance is produced. The specific gravity is usually ascertained by means of the *urinometer*, care being taken that the instrument does not touch the sides or bottom of the glass containing the urine; and that the number on the stem which represents the density is read off by looking at it on a level with the surface of the liquid. Dr. Oliver employs marked *urinometer beads* to determine the specific gravity.

## 2. Chemical Examination.

This is carried out with a view of determining:—*a.* The presence and proportion of certain *normal constituents* of urine, especially urea, uric acid, creatinin, hippuric acid, chlorides, phosphates, sulphates, and colouring matters. *b.* The presence and quantity of *abnormal organic ingredients*, chiefly bile, albumin, sugar, pus, and fat. *c.* The nature of any deposit. *d.* The existence of various substances *introduced into the body from without*, such as lead or arsenic, or different vegetable agents. It is desirable to point out the tests employed for the most important of these materials.

1. UREA.—The **qualitative** test for urea consists in adding pure nitric acid to some urine carefully concentrated by evaporation in a water-bath, when a crystalline precipitate of nitrate of urea is thrown down, the crystals of which appear under the microscope as flat rhombic or hexagonal plates. When urea is present in large quantity, the mere addition of nitric acid to some urine in a test-tube will cause a crystalline precipitate to form. **Quantitative estimation.**—An approximate knowledge of the amount of urea excreted daily, sufficient for ordinary clinical purposes, is obtained by collecting the whole of the urine passed in the twenty-four hours, and taking the specific gravity of a mixed specimen, provided it does not contain sugar or albumin. A table has been drawn up by Professor Haughton showing the relations between the quantity of urine, its specific gravity, and the amount of urea. For accurate determination the *volumetric method* of Liebig is that generally employed. It depends upon the fact that urea forms with mercuric nitrate a precipitate of definite composition. For this process three solutions are required, namely, 1. One consisting of a volume of cold saturated solution of barytic nitrate with two volumes of saturated baryta-water. 2. A standard solution of mercuric nitrate. 3. A solution of carbonate of soda, about gr. xx to 3i. A measured quantity of the urine is first mixed with half its bulk of the baryta solution, in order to precipitate the sulphates and phosphates, which are then separated by filtration, a drop or two of the filtrate being further tested in order to see that these ingredients are entirely removed, and if they are not, more baryta solution must be added. A certain quantity of the filtrate is then taken, and the mercurial solution very cautiously dropped



into it from a graduated burette, until it begins to become turbid, the amount required to produce this effect being noted down. No precipitate falls until all the chloride of sodium present has been decomposed, and the quantity required for this purpose must be subtracted in the subsequent calculations from the total volume added. As soon as a precipitate forms, the mercurial solution is to be allowed to flow in freely at first, and afterwards again gradually, the mixture being stirred with a glass rod. In order to ascertain when the whole of the urea has been precipitated, a little of the carbonate of soda solution is placed on a white porcelain surface, and a drop of the precipitated mixture added to it by the aid of a glass rod; as soon as a yellow tinge is thus produced, it indicates that the whole of the urea has been thrown down. The matter then becomes merely one of calculation, the mercurial solution being of such a strength that each cubic centimeter used *after the decomposition of the chlorides* corresponds to 0.01 gramme of urea.

Another mode of estimating urea quantitatively is Davy's method, modified by Dr. Russell and Mr. West, which is founded on the decomposition of urea by the hypobromites, and the liberation of nitrogen. For carrying out this process a special apparatus is required, convenient forms of which have been devised by Mr. Apjohn and Mr. Blackley. A solution is prepared containing 100 grammes of caustic soda and 25 c.c. of bromine in 250 c.c. of water, and the nitrogen which is liberated when a measured quantity of this solution acts upon a certain amount of urine is collected, and the proportion of urea present can be calculated therefrom. There are other modifications of this test.

2. URIC ACID.—The test for the presence of uric acid is to place a small quantity of the substance supposed to contain it on a porcelain dish; add a little nitric acid; evaporate over a spirit-lamp until a yellowish-red residue is left; and finally touch this when cold with a glass rod dipped in solution of caustic ammonia. A characteristic bright violet colour is immediately brought out, due to the production of *murexid*. To obtain the acid from urine, it is requisite to add excess of strong hydrochloric or acetic acid to a specimen of this fluid, and to let it stand for 24 hours. The uric acid is then precipitated in a crystalline form, and may be tested as above. This is also the method usually followed for its **quantitative estimation**, though it is not very accurate, a measured quantity of urine being taken, and the precipitate collected on a weighed filter, which is afterwards dried and again weighed.

3. INORGANIC ACIDS.—With regard to the inorganic acids, it must suffice to state that phosphoric acid is best recognized by the ammonio-magnesian test; hydrochloric acid by argentic nitrate; and sulphuric acid by barytic nitrate. The *quantitative estimation* of these substances presents so many practical difficulties, and their proportion is liable to so many variations from different causes, that its consideration would be quite beyond the province of this work.

4. COLOURING MATTERS.—Nothing definite can be said as to the clinical investigation of the colouring matters of the urine; their usual amount is determined in an ordinary way by the appearance of the urine. Blood and bile-pigments are often present in this fluid, but they will subsequently be specially considered. Certain drugs are also liable to affect the colour of the urine. Thus rhubarb and senna impart a deep brownish-yellow colour, due to chrysophanic acid, which becomes bright red on the addition of an alkali; this colour disappears when an acid is

added. *Hæmatoxylum* gives a red tinge; and *santonin* may make the urine bright-yellow or greenish, which changes to orange-colour, cherry-red, or purple when ammonia is added, these colours being discharged by an acid. Carbolic acid or creosote, even when absorbed after local application, are liable to cause the urine to assume a dark greenish-brown or almost black colour, due to the presence of oxidation-products of hydrochinon, and other substances. Gamboge may also colour the urine yellow. *Melanin* is sometimes present in the urine of patients suffering from melanotic cancer; it is not evident at first, but after standing, or the addition of oxidizing substances, such as nitric acid, black pigment is produced. Other abnormal pigments found in the urine are *feburo-uro-bilin*, *uro-erythrin*, and *uro-hæmatin*. *Feburo-uro-bilin* is reddish, and is found in febrile urine, or sometimes in that containing bile. It is related to bilirubin and hæmatin, and is supposed to be derived from a chromogen present in the urine. It can be detected by the spectroscope; by adding a small quantity of chloride of zinc to an alkaline solution, when a green fluorescence appears; and by adding ammonia to the urine, or to an acid solution of uro-bilin, when the colour becomes clear yellow. *Uro-erythrin* or *purpurin* is pinkish-red, and is also often present in febrile urine, or in connection with cirrhosis of the liver, but may occur from slight errors in diet or other causes; it adheres to precipitates of uric acid or urates, giving them a brick-dust colour. Allusion may be here made to *indican*. This substance exists in small proportion in healthy urine, and is identical with the *uroxanthin* of Heller. It is derived from indol, which is formed by pancreatic digestion of albumen in the intestine, and indican will appear in quantity in the urine when this substance is injected subcutaneously. When indican is in excess the urine is dark-yellow; it yields indigo with certain processes, and thus its presence in the urine is detected. This change may occur spontaneously during decomposition of urine, a glistening dark-blue film forming on its surface. In most instances, but not always, the addition of nitric acid develops the colour of indigo, as dark violet, dark greenish-blue, or almost black. The most reliable test seems to be to add an equal quantity of fuming hydrochloric acid to some urine in a test-tube, and then a concentrated solution of chloride of lime drop by drop, until the blue colour is fully developed. If this mixture be shaken up with chloroform, the latter dissolves the indigo, and sinks to the bottom of the test-tube. The supernatant liquid remains reddish or purplish, probably from the presence of indigo-red. Excess of indican seems to be most important clinically as an indication of obstruction of the small intestines, and is said to help to distinguish this condition from obstruction of the large bowel. It has been noticed also in hepatic and gastric cancer, Addison's disease, lymphadenoma, phthisis, tabes mesenterica, and cholera. Indican is said to be present in considerable quantity in the urine in tropical climates; and to be increased by turpentine, oil of bitter almonds, and *nux vomica*. Dr. Robert Maguire has recently (*British Medical Journal*, October 25th, 1884) drawn attention to some interesting observations on the darkening of colour of certain urines on exposure to the air.

5. ALBUMEN.—Different albuminous substances are now recognized in the urine, namely, *serum-albumin*, *egg-albumin*, *serum-globulin*, and *Bence-Jones's albumin*, which is supposed to be identical with *propepton* and *Kuhne's hemialbuminose*. It will only be practicable here to point out the chief tests for albumen in the urine, and to note the most

striking differences between its several forms. The ordinary tests for albumen are *heat* and *nitric acid*, and Dr. William Roberts has lately expressed his confidence in these tests, in preference to those more recently introduced. It will, however, be necessary to notice here other tests which have now come into vogue.

*a.* The **heat-test** is best performed by placing some urine in a test-tube, and heating its upper portion by means of the spirit-lamp, this being then compared with the lower part, and thus the slightest opalescence can be detected. There are some important precautions to be observed. 1. It is essential to see that the urine is duly acidulated. Dr. W. Roberts gives the following directions on this point:—"A test-tube is charged with about three drachms (10 c.c.) of urine, and to this is added a single drop of acetic acid (B.P.). If the urine be alkaline, it should first be carefully neutralized by adding successive drops of acetic acid until the litmus-paper shows a distinct but slight acidity, and then the final single drop of acid is added before boiling. By using this small and definite quantity of acid the precipitation of mucin is almost entirely avoided; and also the risk of preventing the precipitation of albumen by the use of too much acid." 2. The portion of urine employed should be quite transparent and clear, and if there is any permanent turbidity, the urine ought to be filtered; when this is due to urates, however, all that is necessary is to pass the tube two or three times along the flame, when the urates are immediately dissolved, and then the upper part may be further heated. 3. The portion which is being tested must be boiled, because, should the proportion of albumen be small, it is only then that cloudiness is observed. The rapidity of coagulation is in proportion to the quantity of albumen present. 4. After heating, it is well to add a drop or two of nitric acid, because, if the urine is only faintly acid, earthy phosphates may be precipitated, and thus give rise to turbidity. These salts, however, are immediately dissolved by the acid.

*b.* The addition of **nitric acid** to cold urine by the *contact-method* of Heller, is, with certain precautions, a very delicate test for albumen. Dr. W. Roberts regards it as the albumen-test best adapted for clinical work. He affirms that it is less likely to mislead, is more direct and certain in its indications, and requires less control by secondary testings than any other albumen-test. In performing this test the usual plan is to place some urine in a test-tube, incline the latter, and gradually pour strong acid down along its inner surface, so that, owing to its higher specific gravity, the acid may sink to the bottom of the tube without mixing with the urine. Or some of the acid may be placed in the tube, and the urine poured upon it. In order to perform this test delicately, however, the acid should be introduced by means of a pipette. Near the junction of the two liquids more or less turbidity is observed, which gradually spreads upwards through the stratum of urine. When a urine containing both albumen and mucin is tested by this method, the albumen is thrown down just above the line of junction of the two fluids, while the mucin is brought into view towards the upper part of the column of urine, where it gradually forms a diffused haze, quite distinct from the opalescent zone at the line of junction (W. Roberts). The chief fallacies relating to the nitric acid test are as follows:—1. If only a very little acid is added to the urine, the albumen may not be precipitated at all; and, on the other hand, if a considerable quantity is suddenly mixed with it, the same result may follow, even though there is much



albumen present. The solubility in excess of nitric acid applies only to serum-albumin, from which egg-albumin is distinguished by the coagulum being insoluble in excess. 2. Cloudiness may not be observed at once if the proportion of albumen is very small, and therefore it is desirable to wait in doubtful cases for a few minutes—from 15 to 30 at the most—for a possible reaction to take place (W. Roberts). 3. If the urine is highly concentrated, the addition of nitric acid is liable to cause precipitation of urates; in this case, however, the cloudiness begins *at the surface* of the urine, and extends downwards, while heat dissolves the precipitate instantly. 4. When there is great excess of urea, nitric acid may cause its precipitation, but this usually occurs very slowly, and the deposit is crystalline. 5. Opalescence of urine may be due to the patient taking cubebs or copaiba, and this is sometimes increased by adding nitric acid. These ingredients, however, are recognized by their odour; and by the effects of heat, which diminishes the opalescence, and prevents any turbidity with nitric acid. Bence-Jones's albumin is precipitated by nitric acid in the cold, but the precipitate is dissolved on heating, and again appears after cooling. The presence of mucin has already been noticed.

Dr. W. Roberts has recently suggested a modification of the nitric acid test, a mixture being used consisting of one volume of strong nitric acid with five volumes of a saturated solution of sulphate of magnesia. Amongst other advantages, this mixture does not fume, nor stain, nor burn the fingers or garments; and can be carried about in a corked bottle with much less risk.

c. **Picric Acid** is regarded by Dr. George Johnson as the most trustworthy and sensitive test for albumen, being a most delicate albumen-precipitant. It may be used either as a saturated solution, or as a powder or crystals. When unmixed with other agents, Dr. Johnson affirms that picric acid gives no reaction with mucin. The only precipitates which it may produce are urates, peptones, and vegetable alkaloids, but these are readily and completely redissolved by heat. Dr. Oliver recommends a mixture of citric acid in the picric solution (5ij dissolved in 3i).

d. It must suffice to enumerate the other principal tests employed for detecting albumen in urine, namely:—saturated solution of *potassium ferrocyanide*, the urine being freely acidulated by *citric acid* (Pavy); *acidulated brine* (W. Roberts); standard solution of *potassio-mercuric iodide*, the urine being strongly acidified with *acetic* or *citric acid*; and *sodium tungstate*.

**Portable Tests.**—The introduction of portable tests for the urine is a decided advance, and promises to be of much clinical value. In detecting albumen Dr. Johnson employs the powder of picric acid in this way. Dr. Pavy employs *test-pellets*, composed of sodium ferrocyanide and citric acid. The most important portable tests, however, are those introduced by Dr. Oliver,\* of Harrogate, in the form of *test-papers*. These are made with *potassio-mercuric-iodide*, *sodium tungstate*, *potassium ferrocyanide*, and *picric acid* respectively. He also has prepared separate papers of citric acid; and compound papers, containing this ingredient with some of the others. For full instructions respecting the employment of these tests the reader must refer to Dr. Oliver's work. Small cases are made by instrument-makers, which contain the necessary apparatus.

\* "On Bedside Urine Testing," by George Oliver, M.D.

**Quantitative examination.**—For ordinary clinical purposes, a sufficiently exact estimation of the amount of albumen present in a specimen of urine may be obtained by adding a little acetic acid to some of this fluid in a test-tube, boiling, and then setting the specimen aside until the coagulated particles have all subsided, when the depth of the deposit can be compared with that of the urine, the proportion being expressed as “almost solid,”  $\frac{3}{4}$ ,  $\frac{1}{2}$ ,  $\frac{1}{4}$ ,  $\frac{1}{8}$ th, &c, or as mere “cloudiness” or “a trace.” Sometimes the precipitate from a measured quantity of the urine is collected on a weighed filter, which is then washed, dried, and again weighed.

Another method of estimating the amount of albumen in urine has been introduced by Dr. William Roberts, which he terms the *dilution method*. It consists in diluting the albuminous urine with water, until it gives a faint but distinct reaction with nitric acid in between half and three-quarters of a minute after the contact of the acid. Each dilution with an equal volume of water is counted as a “degree of albumen,” and such degree corresponds to 0.0034 per cent. or 0.0148 grain per fluid ounce; from this the total amount of albumen may be calculated. The operation is a delicate one, and should be performed in daylight.

Dr. Oliver employs his test-papers for the quantitative extrication of albumen in urine, and he thinks the potassio-mercuric iodide is the most suitable.

The effect of albumen on the *polarization of light* has also been made use of to determine its quantity in urine.

Before estimating the amount of urea and other urinary constituents, it is requisite to remove any albumen present, by carefully acidulating with acetic acid, heating *just to the boiling point*, and filtering.

*Serum-globulin* is detected and estimated by saturating the urine with sulphate of magnesia in a finely-powdered state, when this substance separates as a white flocculent precipitate. *Propepton* is obtained by acidulating some urine with acetic acid, saturating with sulphate of magnesia, boiling, and filtering while hot; serum-albumin and serum-globulin will remain on the filter, while the propepton passes through in solution, and will precipitate on cooling.

It may be mentioned here that *peptones* have been found in urine in certain conditions, especially in connection with chronic purulent discharges. Little is known as to whether one or several varieties may be met with. Oliver gives the following general properties of the family group:—1. Non-precipitation by heat, nitric acid, or the ferrocyanic test. 2. Precipitation by the picric, mercuric, and tungstate test; solubility of the precipitate by heat below the boiling-point; and re-appearance of it as diffused opacity as the solution cools.

The tests recommended to detect peptones in urine are as follows:—1. A rose or pink tinted zone is observed on superposing a layer of the peptonous urine on Fehling's solution (Ralfe). 2. To a drachm or two of peptonous urine add a drop of honey in solution or glucose, then a drop or two of solution of copper, and finally liquor potassæ. A rich purple tinge will be produced, which on boiling will change to a yellow colour, without any deposit of the suboxide of copper.

6 SUGAR.—Grape-sugar is the variety met with in urine. It has been stated by many observers to be present in healthy urine, but even if such is the case, the proportion of sugar is so minute as to be practically of no consequence from a clinical point of view.

Before proceeding to test for sugar, it is important to ascertain that there is no albumen present in the urine, and should there be any, it must first be got rid of. It has been recommended to filter urine thoroughly through animal charcoal before testing for sugar, and especially before estimating its quantity.

**Qualitative tests.**—1. **Reduction-test.** This is by far the most reliable, and it depends upon the power which grape-sugar possesses of reducing certain metallic oxides to a lower degree of oxidation, or to the metallic state. A salt of *copper* is usually employed, *cupric oxide* being reduced to *cuprous oxide*, which falls as a precipitate. There are two chief modifications of this test, namely, Trommer's and Fehling's.

*a. Trommer's.* A drop or two of a weak solution of *cupric sulphate* is added to some of the urine in a test-tube, and then about half its bulk of *liquor potassæ*, care being taken that sufficient of the latter is added to dissolve all the copper salt. On boiling this mixture, which should be quite clear and free from any precipitate, and of a bluish or bluish-green colour, an orange-red precipitate of cuprous oxide falls, which subsequently changes to reddish-brown. This method is for several reasons unsatisfactory, and Fehling's is much more reliable.

*b. Fehling's.* Here a standard solution is made use of, composed of *cupric sulphate* (40 grammes); *potassic tartrate* (160 grammes); *liquor sodæ*, of sp. gr. 1.12 (750 grammes); with *distilled water* to 1154.5 cubic centimetres. This solution is very prone to decompose, racemic acid being produced from the tartaric, which also possesses the power of reducing cupric oxide, and therefore the test-liquid should be always kept in completely-filled and thoroughly-stopped bottles, in a cool and dark place. The proper mode of using this test-solution is as follows:—A drachm or two is placed in a tube, and heated over the spirit-lamp until it boils: if any decomposition has taken place, a precipitate of cuprous oxide will be thrown down in one or two minutes, and should this happen, the test-solution is unsatisfactory, and it is best to prepare a fresh specimen. When the solution is satisfactory, if the urine is supposed to contain a *considerable amount* of sugar, a drop or two of it is to be added while the solution is boiling, when a brick-red precipitate of cuprous oxide falls immediately, and if more urine is added the deposit becomes yellow. Care must be taken under these circumstances not to add too much urine, as great excess of sugar will cause the precipitate to be redissolved, producing a clear yellow solution. If there is only a *small proportion* of sugar present, the urine must be poured in until nearly as much as the quantity of test-solution employed has been added, but on no account must the quantity exceed an equal bulk. This mixture is again to be boiled, when, if a small quantity of sugar is present, it assumes an intense opaque yellowish-green appearance, and slowly a bright yellow deposit subsides. If there is no immediate precipitation, the mixture must be set aside in a warm place to cool gradually, when, if only a very minute proportion of sugar is present, the liquid by degrees loses its transparency, and assumes a light-greenish opacity or milkiness, which is quite characteristic (W. Roberts). It is highly important to avoid boiling for any length of time, as this is quite unnecessary, while uric acid and other urinary constituents have the power of reducing cupric oxide after prolonged boiling.

Dr. Pavy employs a modification of Fehling's solution, which is more stable, and which contains the following ingredients:—*Sulphate of cop-*



*per*, 320 grains; *potassic tartrate*, 640 grains; *caustic potash*, 1280 grains; *distilled water*, 20 fluid ounces. He has *test-pellets* also made, which contain similar ingredients, for portable tests.

Another *reduction-test* may be mentioned, namely, the *bismuth-test*, originally introduced by Böttcher. Dr. Walter Smith of Dublin speaks highly of this test, and gives the following formula for the test-solution proposed by J. Loewe:—*Subnitrate of bismuth*, 15 grammes, dissolve in *pure glycerine*, 30 grammes, and *solution of sodium hydrate* (sp. gr. 1.34) 60-70 c.c.; *dilute with water*, 150-160 c.c. Heat to 212° Fahr. In employing this solution a little of it is added to the urine, which is then boiled for a few seconds. If sugar be present, the liquid first becomes brownish, then the white precipitate of hydrate of bismuth becomes grey, and rapidly deepens in tint, until the reduction precipitate is perfectly black. One fallacy to which this test is liable is the possible formation of a black precipitate of sulphide of bismuth in presence of sulphuretted organic compounds.

2. **Moore's test.**—This test consists in mixing equal quantities of the suspected urine and of *liquor potassæ* in a test-tube, and boiling the upper portion. A change of colour is observed to a more or less deep brown; or, if there is much sugar present, it may even become almost black. This is by no means a reliable test, for it cannot detect small quantities of sugar; while urine which is concentrated and high-coloured, or which contains excess of phosphates or much albumen, will become darkened on boiling with liquor potassæ, and this is particularly the case with markedly albuminous urine, if the liquor potassæ should have become impregnated with lead from having been kept in glass bottles.

3. **Fermentation-test.**—A small quantity of German yeast is placed in a test-tube, which is then quite filled with the urine, inverted over a shallow dish or saucer containing some of the same liquid, and set aside in a warm place for some hours. If sugar is present fermentation goes on, and carbonic anhydride is set free, which collects at the top of the tube, gradually expelling the urine. The gas may be tested by a lighted taper. This is not a delicate test. Dr. William Roberts makes use of the *loss of specific gravity* in the urine after having undergone this process, both for indicating the presence of sugar, and its amount.

4. **Hassall's test.**—Dr. Hassall considers the growth of the yeast-plant (*torula cerevisiæ*) in urine, visible on microscopic examination, as certain evidence of the presence of sugar. There are several reasons why this test is not very practicable.

5. **Johnson's test.**—Dr. George Johnson has introduced a test for sugar in the urine, which, however, was first discovered by Braun in 1865, and which depends on the fact that when *picric acid* and *liquor potassæ* are boiled with glucose, the acid is reduced to *picramic acid*, and a deep red colour is thus developed. He affirms that this test is very delicate. The picric acid which is used for sugar-testing should be purified by re-crystallization. Dr. Johnson employs grain lumps of caustic potash along with picric acid powder, in his portable apparatus for testing urine.

6. **Indigo-carmin test.**—When carmine of indigo is heated with carbonate of soda and saccharine urine, the blue colour is changed gradually into green, then into red, and finally into yellow. This test in an aqueous form does not seem to be reliable, but Dr. Oliver has prepared *test-papers* charged with the same definite quantity of the constituents, and of these he speaks very highly. When heat is applied to the test-papers in water, a fine blue solution is obtained. A characteristic series of

changes of colour is observed when this is boiled with diabetic urine, the solution finally becoming of a straw colour. Dr. Oliver also employs test-papers charged with a saturated solution of carbonate of soda for certain special purposes.

**Quantitative estimation.**—As regards diabetic urine, where a large quantity of sugar is passed daily, the specific gravity will give an approximate idea of the proportion discharged. For accurate analysis, however, the following are the chief methods adopted. 1. *Volumetric analysis*.—10 c.c. of Fehling's solution are placed in a flask and boiled, and some of the urine, either alone or diluted with a certain proportion of water, according to the amount of sugar present, is added by degrees from a graduated burette, until the blue colour has entirely disappeared, which can be observed by holding the flask between the eye and the light after each addition, allowing it to stand for a minute, so that the sediment may subside. The above amount of the test-solution is decomposed by 1 grain of sugar, and from this the whole quantity of sugar excreted may be determined. 2. *Differential-density method*.—The loss of density in the urine after fermentation is considered by Dr. William Roberts as giving very accurate information as to the quantity of sugar present. Each degree of density lost corresponds to 1 grain of sugar in every fluid ounce of urine. 3. The *polariscope* is sometimes employed to estimate the quantity of sugar, provided the urine is transparent, this being determined by the degree to which the plane of polarization is rotated to the right. 4. Dr. Johnson employs *picric acid* and *liquor potassæ* to estimate sugar in urine quantitatively. 5. Dr. Oliver affirms that his *indigo-carmine test-papers* are available for quantitative determination of sugar.

7. **BILE.**—The following are the chief tests which are practised for the detection of bile in the urine:—

1. **Gmelin's test.**—This is employed for the purpose of indicating the presence of *bile-pigments*. It consists in bringing *strong nitric acid* into contact with the urine, when, if bile be present, a play of colours is developed, from green to violet, blue, and finally to red, which soon disappears. These changes in colour are due to the gradual oxidation of the urinary pigments. The green colour is the most characteristic, being dependent upon the formation of biliverdin, and it must be remembered that a reddish tint is brought out by nitric acid in most specimens of urine, while if much indican is present, a blue or violet or even a green colour may be developed.

Gmelin's test may be performed either by placing a drop or two of the urine and acid separately on a white porcelain surface, and then causing them to come into contact; or by pouring a little nitric acid into a test-tube, and, holding this in an oblique position, allowing the urine to run gently down its interior, so that it may fall on to the surface of the acid. The succession of colours is observed at the junction of the two liquids. The urine may be placed in the tube first, and the acid poured in gradually, so that it sinks to the bottom.

Other oxidizing agents have been employed for the purpose of bringing out the green colour in urine containing bile-pigments. Dr. Walter G. Smith, of Dublin, has advocated the use of *tincture of iodine*, one or two drops of which are allowed to trickle down on to the surface of the urine contained in a test-tube.

2. **Pettenkofer's test.**—By this test the *bile-acids* are detected. It depends upon the development of a deep purple colour when these

acids are acted upon by *cane-sugar* and *strong sulphuric acid*. This reaction is, however, for several reasons most unreliable when applied to urine, and the bile-acids must be separated from the urine by a complicated process, should it be needful to carry this out.

Ordinarily Pettenkofer's test is practised in one of the following ways:—(a.) Strong sulphuric acid is gradually added to some of the liquid to be examined, contained in a test-tube, until the bile-acids, which are first precipitated, are redissolved. Then a small lump of sugar or a drop of syrup is allowed to fall into the mixture, when a series of colours is observed, from pink to red, and finally to purple. (b.) Mix a drop of concentrated syrup with, or dissolve a fragment of loaf sugar in a little of the fluid placed in a white porcelain dish; then add about an equal volume of sulphuric acid; and finally heat moderately. The change of colour to red, and afterwards to purple, will be developed.

### 3. Microscopic Examination.

The objects which may be discovered in urine by the aid of the microscope include:—a. *Extraneous materials*, such as fragments of cotton-wool or flax, hairs, woody fibres, starch-granules, or oil-globules. b. *Unorganized particles*, crystalline or amorphous, including chiefly uric acid and urates, oxalate of lime, phosphates, cystine, xanthine, leucine, and tyrosine. c. *Organized bodies*, namely, renal or other epithelium, renal casts, blood-corpuscles, pus-cells, cancer-cells, fragments of hydatids, pigment, fat, spermatozoa, or low vital organisms, including vibriones, mould fungus, torulæ, and sarcinæ. In order to examine urinary deposits microscopically, a quantity of the urine must be set aside in a conical or cylindrical glass, the supernatant fluid being poured off after standing for two or three hours, and a drop of the sediment then placed on a glass slide, or this may be taken up by means of the pipette. Not uncommonly the microscope is also employed to examine deposits formed during chemical reactions; and, on the other hand, the effects of chemical reagents on objects observed under the microscope sometimes give valuable information.

### 4. Examination of Urinary Deposits.

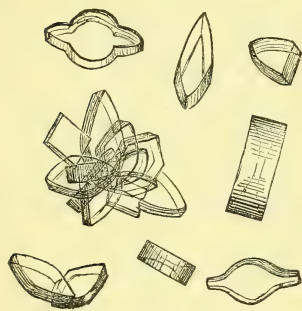
It will be expedient now to consider deposits in urine separately, and to describe the chief characters by which those ordinarily met with are recognized. In examining such deposits, certain *general characters* must first be noticed, namely, their amount; colour and general aspect; mode of aggregation and deposition, whether amorphous, crystalline, or flocculent; and their apparent density and manner of precipitation, which may be observed by shaking up some of the specimen, and then allowing it to stand. Next they must be submitted to the action of *heat*: of *nitric* and *acetic acids*; and of *liquor potassæ*, in order to test whether they are dissolved by these agents. Finally some of the sediment must be examined *microscopically*. The principal urinary deposits, with their main characters, are as follows:—

1. **Uric acid.**—Urine which deposits this substance is always very acid. Generally the uric acid is mixed with urates, and forms some time after the discharge of the urine. To the naked eye it is presented



as more or less brown or brownish-red crystals, either forming a superficial film, adhering to the sides of the vessel, or falling as a heavy reddish deposit like brick-dust. The crystals are not soluble by heat or dilute acids, but they are dissolved by strong alkalis. Chemically they may be recognized by the *murexid-test*. Microscopically they are

FIG. 42.



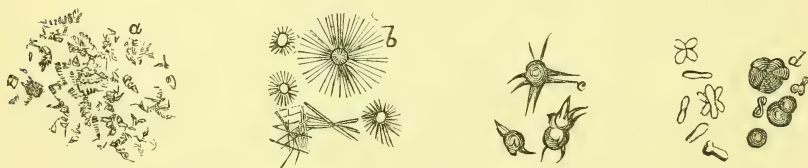
Uric acid crystals.

distinguished by their colour and form, the latter being primarily that of a rhombic prism or lozenge with pointed ends, but the crystals present numerous diversities in shape, becoming ovoid or oval tablets, barrel-shaped, quadrangular, cubes, hexagonal, rod-like, stellate, rosette-like, &c. (Fig. 42.) If a drop of *liquor potassæ* is added, they are dissolved at once, but may be re-precipitated as hexagonal plates by adding a drop of *acetic acid*.

**2. Urates.**—These are very common urinary deposits, even in health. As a rule they appear as *amorphous urates*, which consist of the salts of potash, soda, ammonia, and lime, in variable proportions.

The conditions favourable to their deposit are a high specific gravity, and very acid reaction of the urine; and a low temperature of the air. They form more or less speedily after the discharge of the urine. The precipitate is quite amorphous, pulverulent, and loose; sinks with tolerable rapidity; and presents a variable colour, such as milky, fawn, orange, pink, deep-red, or purplish, owing to the urates carrying down the urinary pigments. A film forms on the surface and sides of the containing glass. *Heat* dissolves the precipitate very speedily and completely, and *liquor potassæ* produces the same effect. Microscopically it appears as minute amorphous granules, of variable size, and more or less dark and opaque. (Fig. 43, *a.*) Urates of soda and ammonia are occasionally deposited in a *crystalline* form, the former as a whitish or yellowish

FIG. 43



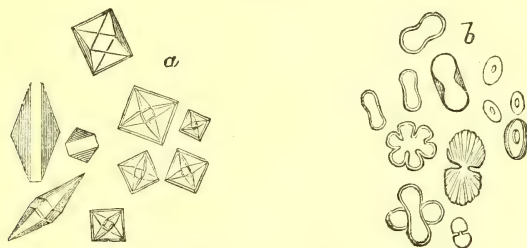
*a.* Amorphous urates. *b.* Crystals of urate of soda. *c.* Hedgehog crystals of urate of soda. *d.* Urate of ammonia.

sediment which sinks rapidly, and usually forms in the bladder; the latter generally as a dense white precipitate, in urine which has decomposed and become strongly ammoniacal. They appear under the microscope as globular, dark, opaque particles, from which project spiny crystals, straight or curved. (Fig. 43, *b. c.*) Urate of ammonia also occurs in the form of minute dumb-bells. (Fig. 43, *d.*)

**3. Oxalate of lime.**—This forms but a very slight, colourless deposit, usually in high-coloured and acid urine. It crystallizes in fine lines on the interior of the containing glass; while the sediment is described by Dr. William Roberts as consisting of two parts—a soft, pale-grey,

mucous-like portion at the bottom; and overlying this a snow-white, denser layer, with an undulating but sharply-limited surface. Oxalate of lime is not dissolved by *heat*, *acetic acid*, or *liquor potassæ*; but is speedily soluble in *mineral acids*. It crystallizes either in the form of minute octahedra, very short in one axis, or of pyramids; or as biconcave, circular or oval discs, with rounded margins. Under the microscope the former vary in appearance according to their position, but commonly they present a characteristic envelope-like appearance, ex-

FIG. 44.

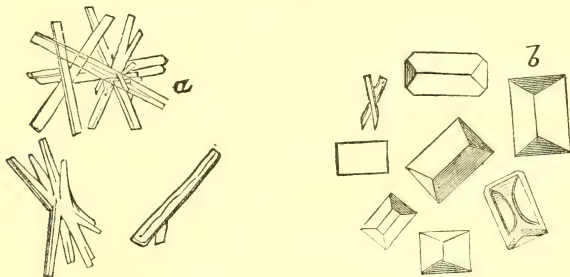


Oxalate of lime.—a. Octahedra and pyramids. b. Dumb-bells and ovoids.

hibiting a square surface crossed diagonally by two lines. (Fig. 44, a.) The latter are presented as dumb-bells, or as ovoids and circles. (Fig. 44, b.)

4. **Phosphates.**—These are deposited in alkaline urine as a rule, but occasionally in that which is neutral or faintly acid. They are *not dissolved by boiling*, which even increases the precipitate, giving rise to

FIG. 45.



a. Stellar phosphates. b. Triple phosphates.

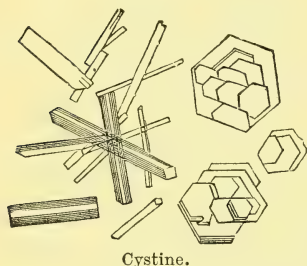
turbidity, and causing the phosphates to fall in flakes. A drop of *nitric acid* dissolves them instantly. Three varieties of phosphates are met with as urinary deposits:—a. *Amorphous phosphate of lime*—*Bone-earth*. This occurs as a whitish, light, flocculent sediment; accompanied with a superficial iridescent film. Microscopically it presents irregular groups or patches of minute pale granules. b. *Crystallized phosphate of lime*—*Stellar phosphate*. Of rare occurrence, the crystals assume very various forms, but most of them resemble crystalline rods or needles, either distinct or grouped in various ways. (Fig. 45, a.) c. *Phosphate of ammonia and magnesia*—*Triple phosphate*. This is the most common form of phosphatic deposit, being generally mixed with the amorphous phosphate. The precipitate is quite white; while brilliant colourless

crystals are seen forming a film on the surface of the urine, and studding the sides of the glass. Under the microscope the crystals are usually very characteristic, appearing as triangular prisms with bevelled ends, but the primary form is liable to numerous variations. (Fig. 45, b.)

5. **Carbonate of lime** occasionally falls as an amorphous deposit in urine, along with phosphates; and it is said to be now and then presented in the form of crystals.

6. **Cystine**.—The urine in which this rare substance is found is turbid on being passed, of a yellowish-green colour, having an oily aspect, and a peculiar odour like sweet-brier. It is faintly acid, but very prone to decomposition, becoming green and evolving hydric sulphide. The deposit which forms on standing appears to be abundant and light, but it weighs very little. This deposit is *not dissolved by heat or acetic acid*, the latter, on the contrary, causing increased precipitation; but it is soluble in *mineral acids* and *caustic ammonia*, being deposited from the latter solution after spontaneous evaporation. Microscopically the crystals appear as brilliant, colourless, hexagonal

Fig. 46.



tablets, having a pearly lustre, often overlapping each other, or being arranged in the form of rosettes. Cystine also crystallizes in square prisms. (Fig. 46.)

7. **Leucine—Tyrosine**.—These substances are stated to form a greenish-yellow sediment, tyrosine appearing under the microscope as delicate needles, grouped in globular masses or bundles; leucine as dark globules, resembling those of fat.

8. **Fat**.—The chief condition in which a deposit of fat is observed is in the so-called *chylous urine*. It causes the liquid to assume a whitish, opaque, and milky aspect, which disappears on the addition of ether, the urine then becoming transparent and clear. On standing the fat collects on the surface as a creamy layer. Under the microscope it appears in the form of extremely fine molecules. Fat is also said to be present in the urine in some cases of pancreatic disease.

The material named *kiesteine* may also be alluded to here. This is a peculiar whitish pellicle, which sometimes forms on the surface of urine after it has stood for a few days, and was formerly supposed to be a characteristic sign of pregnancy, but it is now known that such is not the case. It consists of abundant fat-globules, crystals of phosphates, and the mould-fungus.

9. **Mucus and Epithelium**.—All urines contain a small quantity of these elements, the epithelium being shed from the genito-urinary passages. A light cloud subsides on standing, and the cells may be seen on microscopic examination, differing in character according to the part whence they are derived. In some cases mucus is present in considerable quantity. Mucin is precipitated by both vegetable and mineral acids. Its distinctions from albumin have already been considered. Mucus does not become ropy on adding *liquor potassæ*, and thus differs from pus. Moreover, in order to distinguish mucus from pus, the urine may be filtered; if it contains pus the filtrate will give the tests for albumen; if mucus is present, after acidulating the filtrate with acetic



acid, a precipitate of mucin forms in the cold. In connection with certain diseased conditions, the epithelium of the bladder, ureters, pelvis of the kidney, or of the renal tubules may be present in the urine. The *extra-renal* cells (Fig. 47, *b*) present such varied and curious shapes, that they have been mistaken for cancer-cells. *Renal* epithelium-cells (Fig. 47, *c*) may be separate or in patches, and healthy in appearance,

FIG. 47.



*a.* Vaginal epithelium. *b.* Epithelium from the bladder, ureter, and pelvis of the kidney. *c.* Renal Epithelium, healthy and fatty.

atrophied, granular, fatty, or entirely disintegrated. Usually they are associated with *casts*.

10. **Pus.**—If pus is present in any quantity, the urine containing it is turbid on being passed, and does not become clear when boiled. A yellowish-white sediment forms; and if the urine is ammoniacal, or if solution of *potash* or *ammonia* be added, the pus assumes the characters of a ropy, viscid, tenacious material, which can be drawn out into strings. There is necessarily some albumen present, but it is never abundant when due to pus alone. Under the microscope pus-cells (Fig. 48) are visible, but they are frequently much altered in their characters in decomposed urine.

FIG. 48.



Pus-cells in urine, unaltered, and affected by acetic acid.

11. **Blood.**—Urine containing only a little blood may not give any indication of its presence to the naked eye, but it often presents a characteristic smoky appearance, or it may be of a dirty reddish-brown colour, of varying depth; when more abundant, the fluid has a more or less deep pink or red colour, until in extreme cases it may look almost like pure blood. Occasionally the urine resembles porter. Sometimes the blood is separate from the general mass of urine, and it may be in distinct coagula, or these may form on standing. A brownish, grumous, flocculent deposit falls after a time. The urine is necessarily albuminous. Red corpuscles (Fig. 49) are visible on microscopic examination, but if the urine is very dilute, they are liable to be distended and thus to lose their normal characters; or, if it is ammoniacal, they speedily alter in shape, and may even break up. Minute vermiform coagula or blood-casts may also be seen under the microscope. In certain conditions the urine contains more or less of the colouring matter of the blood, with albumen,

FIG. 49.



Blood-corpuscles in urine.

but without corpuscles or fibrin. The colouring matter of the blood in urine may be detected either by the spectroscope or by the guaiacum-test. In performing this test a drop or two of urine is placed in a small test-tube, a drop of freshly-prepared tincture of guaiacum and a few drops of ozonized ether being then added. The tube is agitated, and the ether allowed to collect at the top. If blood-pigment is present, the ether acquires a blue colour, leaving the urine below colourless. Salines and iodine must be absent from the urine.

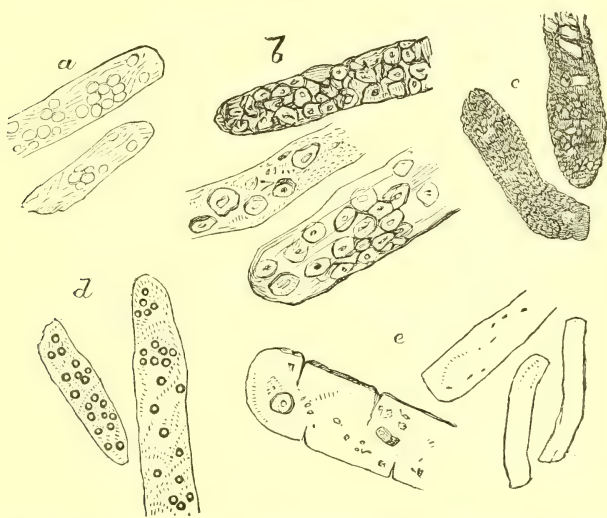
**12. Renal casts.**—In certain morbid conditions of the kidneys minute moulds form in the renal tubules, technically termed *casts*. Their origin in the kidney has been disputed, but of this there can be no doubt, as they have been seen in the convoluted and the straight tubules. These casts are washed away by the urine, in their course outwards being broken up into smaller fragments, and they are visible on microscopic examination, affording information as to the state of the kidneys of the highest importance both for diagnosis and prognosis. As a rule these casts form but a slight cloudy sediment in urine, if any, but sometimes a considerable white deposit falls. Microscopic examination can alone positively reveal the presence of casts, and it is advisable to make a few observations at intervals before coming to any positive conclusion, and also to repeat the examination frequently during the progress of the case. In some instances the examination has to be conducted with the greatest care before casts can be detected, and it may be desirable to introduce a little magenta or carmine staining-fluid beneath the cover-glass; it has also been recommended to add acetic acid to the urine, in order to precipitate uric acid, which will carry down the casts along with it.

Renal casts are generally cylindrical, often bent to a certain degree, and they vary in diameter usually from  $\frac{1}{1000}$  to  $\frac{1}{500}$  of an inch. Occasionally a minute cast seems to be imbedded in a larger one. Care must be taken not to mistake shreds of mucus or other microscopic objects for casts. Certain varieties are met with, but they all consist of a more or less solid basis, in most cases associated with microscopic elements which characterize the several forms. The basis, which is usually transparent or very faintly granular, but occasionally fibrillated, was formerly considered to be coagulated fibrin, derived from blood-plasma of escaped blood, or which had exuded through the walls of the vessels. Undoubtedly it is in many instances of this nature, but in others it has a different origin and composition, but observers are by no means agreed on this matter. Thus it has been supposed that the material is sometimes inspissated albumen, but on no sufficient grounds. The view most in favour at present is that it is derived in some way from the renal epithelium-cells. Thus it has been attributed to colloid degeneration of these cells; to a substance secreted by them; or to the material which in health forms the substance of the epithelial cells. In disease this substance, perhaps somewhat altered or not perfectly formed, collects in the tubes and becomes inspissated. (Beale.)

The chief recognized varieties of casts are as follows, two or more kinds being commonly observed together:—*a. Blood-casts* (Fig. 50, *a*), consisting either of accumulations of blood-corpuscles; or of fibrinous casts studded with these elements. Dr. George Johnson has described "white cell casts," or "exudation cell casts," which he thinks are leucocytes. *b. Epithelial casts* (Fig. 50, *b*), usually of some size, and presenting renal epithelium-cells on the surface or imbedded in their sub-

stance, frequently somewhat changed from their normal condition. *c. Granular casts* (Fig. 50, *c*), usually of moderate size, and characterized by being more or less granular and opaque in appearance, the granules being either coarse or fine, and consisting of protein or fat; these are generally mixed to a variable degree with other elements, such as altered epithelium or crystals of oxalates; and also with free granules. On the addition of acetic acid to the specimen under the microscope, if the granules consist of protein they disappear, if they are of a fatty nature they become more evident. Granular casts are divided by some authorities into *large* and *small*. *d. Fatty casts* (Fig. 50, *d*), which exhibit scattered oil-globules; or collections of these in the form of dark, botryoidal masses. *e. Hyaline, transparent, or waxy casts* (Fig. 50, *e*), varying considerably in diameter, and therefore divided into *large* and *small*. These have either a perfectly clear, transparent, and glassy aspect; or

FIG. 50.



*a.* Blood-casts. *b.* Epithelial casts. *c.* Granular casts. *d.* Fatty casts. *e.* Hyaline cases.

present faint markings on the surface, or a dimly molecular appearance. Sometimes a few nuclei or epithelium scales are visible upon the hyaline casts. In some cases they can only be seen after the addition of *iodine* or *magenta solution*. *f. Pus-casts*, composed of moulded accumulations of pus-corpuscles, which are extremely rare.

It is in connection with certain forms of *Bright's disease of the kidneys* that casts are of so much importance, and valuable diagnostic conclusions may be arrived at as a general rule from studying their characters. 1. If they consist chiefly of the *blood* or *epithelial* varieties, they indicate an early stage of disease, and the condition of the renal epithelium may be gathered from the characters of the cells on the casts. 2. *Fine hyaline casts* are supposed to come from tubules still covered with epithelium, and when they follow the varieties just mentioned, they show that the disease is subsiding. 3. *Large hyaline casts* are believed to be formed in tubules deprived of their epithelium, and therefore to indicate grave organic changes in the kidneys. 4. Abundant *granular casts* also point to advanced disease, and they are frequently mixed with free granules.



5. *Fatty casts* are of very serious import, as proving the existence of fatty degeneration, with destruction of the renal epithelium.

13. **Organisms.**—The chief organisms which may be observed in the urine are *bacteria* and *vibriones*; forms of *torula*; *sarcina urinæ*; certain entozoa or their ova, especially the *bilharzia hæmatobia* and *filaria sanguinis hominis*; and fragments or hooklets of *echinococci*, in connection with hydatids. The *sarcina urinæ* is smaller than the gastric variety. Some suppose that it is always developed after the urine has been voided, but others think that a colony of *sarcinæ* establishes itself in the bladder. Dr. William Roberts has described a form of *bacteruria*, in which the urine at the moment of emission is loaded with bacteria. The urine has the peculiar grey opalescence indicative of commencing decomposition; and a heavy disagreeable odour, like that of stale fish. The reaction is acid, and on keeping the urine shows no tendency to pass into the ammoniacal fermentation. The organism corresponds in its microscopic characters to the common putrefactive bacterium (*Bacterium termo*), and appears as micrococci, and as actively moving short rods composed of molecules, often joined together in zig-zags. Dr. Roberts supposes that a colony of bacteria is established in the bladder, and that the proliferation of the organism, and perhaps certain products of its action as a ferment, give rise to irritation in the bladder, of which symptoms are usually present.

## II. RENAL TUMOUR.

A tumour connected with the kidney has the following general characters:—1. It is *extra-pelvic*, occupying mainly one or other *lumbar region*, and it cannot be separated from the mass of muscles behind. It, however, increases in a forward direction to a variable degree, sometimes attaining an enormous size, and giving rise to general enlargement of the abdomen. 2. The *shape* is generally more or less that of the kidney, the borders being rounded, but irregularity is not unfrequently observed in this respect. 3. As a rule the *consistence* is firm; occasionally there is a feeling of softness, or even distinct fluctuation may be detected. 4. The tumour is almost or quite *fixed*, not being altered by manipulation or by respiratory movements. 5. *Percussion* reveals dullness extending back to the spine, with tympanitic note in front, due to the colon, unless the tumour becomes extremely large. 6. In some cases it may be advisable to use the *aspirateur* or *exploratory trochar* in the diagnosis of renal tumour.

It will be convenient in this connection to allude to certain peculiarities observed in rare instances in the *position* and *shape* of the kidneys, which may give rise to forms of abdominal tumour.

1. **Movable or floating kidney.** Normally the kidneys are nearly fixed, but occasionally one or both, especially the right, are displaced and become more or less freely mobile, floating about in the cavity of the abdomen. This condition has been by far most frequently observed in females, chiefly after repeated or difficult parturition. It has also been attributed to congenital looseness of the attachments of the kidney; sudden or repeated violent effort; pressure by tight-lacing; rapid absorption of the renal investing adipose tissue in fat people; increase of weight of the kidney during the menstrual periods, resulting from congestion, with a consequent tendency to gravitation downwards; or to this organ being dragged down by a hernia. The movable kidney is felt

as a tumour, having the exact form and feel of the healthy organ; and usually lying, when the patient stands erect, in an oblique position, directed upwards and outwards, about midway between the margin of the thorax and the umbilicus. It is mobile in different directions by change of posture, manipulation, and respiratory movements. In some cases the organ may be grasped in the hand, this causing the patient to experience a peculiar sickening sensation. Percussion generally yields a muffled tympanitic sound. On examining the corresponding lumbar region, it will be found flattened or depressed, as well as tympanitic on percussion, owing to the absence of the kidney. In some instances the displaced organ becomes enlarged and painful from time to time, this event being attributed to pressure on its own duct, leading to retention of urine and consequent inflammation. As a result of repeated attacks of this kind the kidney may become permanently fixed by adhesions. Among the most frequent *symptoms* accompanying the floating kidney are mentioned a sense of uneasiness or dragging pain, increased by walking or standing; neuralgic pains; disturbances of the alimentary canal; and other disorders due to compression or irritation. The urine is generally normal, but micturition may be frequent or painful. During the inflammatory attacks severe symptoms may be experienced.

2. Now and then the kidney is fixed in some **abnormal position**, this condition being either *congenital* or *acquired*. The displaced organ is recognized by presenting the characters of the normal kidney, though the shape is usually somewhat altered; and by the signs of its absence from its proper situation.

3. **Horse-shoe kidney.** In this condition the two organs are united by an isthmus passing across between their lower ends. The horse-shoe kidney might possibly be felt in very thin persons with loose abdominal walls, and might thus be mistaken for a tumour. I have never met with it except at *post-mortem* examinations or in the dissecting-room.

### III. DISTENDED BLADDER.

A distended bladder is liable to be met with in medical practice, and it may simulate a tumour or general enlargement of the abdomen. Its characters are as follows:—1. It occupies mainly the *hypogastrium*, extending upwards and laterally to a variable extent, and being quite symmetrical. 2. The *shape* is conical, the apex being directed upwards. 3. *Fluctuation* is usually perceptible. 4. There is *dulness* corresponding to the enlargement in position and shape; while laterally and at the upper part of the abdomen tympanitic sound can be elicited. 5. By examination *per rectum* the distended bladder may be felt. 6. The use of the *catheter* must never be forgotten; or, if this cannot be passed, a small *trochar* or the *aspirateur* may be inserted above the pubes.

---

## CHAPTER LVII.

## ON CERTAIN ABNORMAL CONDITIONS OF THE URINE.

## I. ALBUMINURIA.

ÆTIOLOGY AND PATHOLOGY.—In considering the causes of albuminuria, it must be remembered at the outset that this condition is frequently present in apparently healthy persons. Abstinence from salt may produce it; as well as the use of a highly-albuminous diet, especially one consisting entirely of eggs. Large enemata of eggs will originate a similar condition. Albuminuria is sometimes present after a cold-bath. The condition is directly attributed either to changes in the blood; in the renal circulation; in the structure of the kidneys; or to a combination of these changes. Egg-albumin, if present in the blood, is secreted by the kidneys in their normal state; and so are propepton, and Bence-Jones's albumin. Serum-albumin is not secreted by the healthy kidney, and before it can pass into the urine there must be some alteration either in the renal circulation or its structure. The disorder of the renal circulation which produces albuminuria is increased pressure in the renal veins; and experiments seem to prove that increased arterial tension does not cause it. It may be produced experimentally in animals by ligature of the renal vein; by varnishing the skin; by injecting egg-albumin into the circulation or subcutaneously; or by introducing a large quantity of water into the veins.

Having offered these preliminary remarks, the chief causes of albuminuria may be thus stated:—1. *Admixture of certain materials which contain albumen*, namely, blood or some of its elements; chyle or lymph; pus from any source; or semen. Albuminuria is not uncommon in cases of masturbation, being then attributed to reflex action on the nerves and vessels of the kidney. 2. *Renal venous congestion*, particularly that due to cardiac diseases, to chronic or acute lung-affections, to interference with respiration, or to pressure upon the renal veins or the inferior vena cava by a tumour, a pregnant uterus, or a collection of fluid. 3. *Acute febrile and inflammatory diseases*, for example, the exanthemata, cholera, diphtheria, pyæmia, ague, pneumonia, serous inflammations, rheumatic fever; and also the pyrexial condition which arises in the course of chronic diseases, such as phthisis. 4. Certain affections attended with an *unhealthy state of the blood*, such as purpura and scurvy. 5. *Pregnancy*. 6. *Acute and chronic Bright's disease*. 7. *Chronic lead-poisoning*; and poisoning by inhalation of *arseniuiretted hydrogen* or *carbonic anhydride*. 8. Some forms of *dyspepsia*. 9. Certain *nervous affections*, which act through the circulation, such as epilepsy or exophthalmic goitre. 10. Bence-Jones's albumin is met with in *osteo-malacia*.

SYMPTOMS AND DIAGNOSIS.—The local symptoms, as well as the characters of the urine in cases of albuminuria, will depend upon its cause, and they present considerable diversity. The presence, amount, and variety of albumin are determined by the tests already described. The drain of albumen from the blood may itself cause serious disorder of the general system, such as anæmia and its consequences, wasting, debility,



and ultimate fatty degeneration of structures. The *diagnosis* of the *cause* of albuminuria must be determined by the general history of the case; the characters of the urine; the accompanying general and local symptoms; and the condition of the several organs. It is important to recognize the fact that considerable variations in the degree of albuminuria may be observed in the same case at different times, and under different conditions; that it is sometimes intermittent; and that this symptom may be entirely absent in grave forms of renal disease. Intermittent albuminuria is not infrequent in persons who have been exposed to malaria; and Dr. Quain has noticed that a similar condition in youth is frequently associated with masturbation.

TREATMENT.—Albuminuria does not usually call for any direct treatment, and the chief measures to be adopted are those which have for their object the prevention or removal of the cause of the morbid condition, if this is practicable, such as regulation of diet, improvement of the renal circulation, or the cure of any organic disease. Medicines are sometimes employed with the view of checking the discharge of albumen, of which the principal are tincture of iron, tannic or gallic acid, mineral acids, alum, and iodide of potassium. It is very questionable, however, whether either of these drugs is really useful for this purpose. The effect upon the system of the loss of albumen may be made up for in some cases by nutritious food, and the administration of iron.

## II. PYURIA—PURULENT URINE.

ÆTIOLOGY.—The sources of pus in the urine are:—1. *Abscess in the kidney*. 2. *Pyelitis*. 3. *Cystitis*. 4. *Urethral inflammation*, especially *gonorrhœa*. 5. *Leucorrhœa* in females. 6. The rupture of any *neighbouring abscess* into the urinary passages.

SYMPTOMS AND DIAGNOSIS.—Pyuria is determined by the general characters of the urine; by chemical examination, which reveals the presence of albumen, and the peculiar ropiness with alkalies; and by microscopic investigation, when pus-corpuscles or, rarely, pus-casts may be visible. The amount of pus discharged varies considerably. The urine may be highly offensive. With regard to *diagnosis*, one of the chief difficulties is to determine whether pyuria results from pyelitis, or from chronic inflammation of the bladder and lower urinary passages, especially when these conditions are associated together. The presence of epithelium-cells from the pelvis and infundibula of the kidneys is very important at an early period in revealing pyelitis, but these elements disappear in course of time; if the complaint exists alone, however, the local symptoms, and the discharge of acid urine containing much pus, especially if combined with a history of some obvious cause of pyelitis, are sufficiently distinctive. When pus comes from the bladder, it is frequently ropy and tenacious, on account of the urine being ammoniacal; while it is also discharged mainly towards the end of the act of micturition. When disease of the lower passages and bladder has been in existence for a length of time, it is highly probable that the kidneys are likewise involved. When pus originates in urethral inflammation, there are the local signs of this condition; while pus escapes before the urine, and can be pressed out independently. In doubtful cases where there is leucorrhœa, it has been recommended to

pass a catheter, and thus to remove some of the urine directly from the bladder for examination.

**TREATMENT.**—Should pyuria require special treatment, the principles are:—1. To remove any obvious cause of the suppuration, if possible. 2. To administer remedies to check the formation of pus, the chief being alum; astringent preparations of iron; mineral acids; tannic or gallic acid; *vegetable astringents*, particularly decoction of uva ursi; buchu; *metallic astringents* in obstinate cases; balsams and resins, especially balsam copaibæ; and turpentine. If the bladder is affected, it may be necessary to wash out this organ with warm water, *antiseptics*, or even *astringents*, which must be used with due care. 3. To support the general health; and to treat the constitutional state by good diet, change of air, sea-bathing, *tonics*, and cod-liver oil.

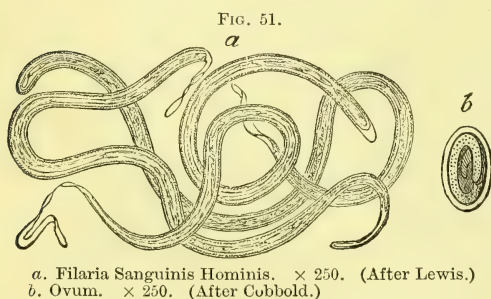
### III. CHYLOUS URINE—CHYLURIA—FILARIA SANGUINIS HOMINIS.

**ÆTIOLOGY AND PATHOLOGY.**—There has been much discussion as to the cause of the peculiar condition of the urine, termed *chyluria* originally by Prout: but the investigations carried on during the last few years seem to have clearly demonstrated the fact that, at any rate in most cases, it is in some way related to the presence of the embryos of a peculiar entozoon in the system, termed the *Filaria sanguinis hominis*, and it will be expedient in this connection to give at the outset a brief account of this entozoon and its pathological relations.

The embryos of the filaria were first discovered in 1870 by Dr. Lewis of Calcutta, in the urine and blood in connection with chyluria. Since then they have been studied by several observers, and the mature worm was discovered by Dr. Bancroft of Brisbane, in 1876, in a lymphatic abscess in the arm, and subsequently in a hydrocele of the cord. Hence it has been named *Filaria Bancrofti*. Two living specimens were also found by Dr. Lewis in 1877, a male and a female. The worm has been observed by Dr. Manson *in situ* in a lymph-channel.

The embryo filaria is very minute, averaging about  $\frac{1}{5}$  inch in length, and about  $\frac{1}{3300}$  inch in breadth. It has a rounded anterior end, a pointed tail, with slightly granular contents, and no definitely differentiated organs. It is enclosed in a tubular sac, extremely delicate and transparent, within which

it can be seen to alternately contract and elongate itself. The sac may sometimes be seen collapsed and folded like a ribbon; and after death the worm may be so contracted as to leave the tube empty at one or both ends. The embryos move about with great activity. The filaria belongs to the nematoid worms, the mature



female form measuring from three to four inches in length, and being about as thick as a hair; the male is smaller. A male and female live together. The ovum measures about  $\frac{1}{600}$  to  $\frac{1}{800}$  inch in diameter, the embryo appearing to be simply the ovum stretched out; it is oval in form, and thin-walled.

With regard to the habitat and dissemination of the filaria, Dr. Manson states that the parents inhabit the lymphatic trunks for the most part, where they may live and procreate for years, if undisturbed. They are viviparous, and discharge their young in large numbers into the lymphatics. He noticed that the embryo filariæ in the blood were almost invariably totally absent during the day; that they begin to make their appearance about six or seven o'clock in the evening; then rapidly increase; and generally again disappear about eight or nine o'clock in the morning. His observations seemed to show that they are taken along with the blood into the stomach of a certain form of mosquito, in which they undergo developmental changes, and it is supposed that they are discharged into water with the larvæ of this insect, and are by this medium conveyed into the human system, through drinking the water. The embryos are then believed to bore their way into a lymphatic vessel, and passing against the current of lymph, become finally located in a distant lymphatic vessel. Dr. Stephen Mackenzie has, however, found that by reversing the habits of a patient under his observation, and turning night into day, the filariæ were found during the daytime, and disappeared at night.

A few remarks may now be offered as to the supposed pathological relations of the filaria and its embryo. Different species of filaria are found in abundance in many of the lower animals; and in many parts of the world, especially tropical and sub-tropical countries, human beings are in large proportion infested by the filaria sanguinis hominis. It is only under certain circumstances that they give rise to pathological conditions, for the active and minute embryos pass readily along the lymphatics and through the glands into the thoracic duct, and thence into the blood. Dr. Manson believes that they occasionally abort, and that unhatched ova are discharged, which are too large to pass through the glands, but block them up as emboli. Once this has occurred, it is apt to occur frequently, and thus tends, sooner or later, to cause more or less serious and wide-spread obstruction of lymphatics. He supposes that in this way disease may be set up; or by inflammatory or other changes caused by the parasite. The affections which have thus been specially referred to filaria are *chyluria*, *lymphorrhagia*, *elephantiasis lymphangiectodes*, and *true elephantiasis*. Nothing definite is known as to the pathological relations of the mature worm. In a case of chyluria and filarial disease observed by Dr. Stephen Mackenzie, the thoracic duct was found after death to be impervious, and lost in a mass of inflammatory material about the middle of its course; the part below, with the abdominal lymphatics and those connected with the kidneys, being enormously dilated. There were no filariæ. These had disappeared from the blood three months before death, when double pleurisy and empyæma came on, and after death acute cystitis and suppurative nephritis were also found. It was believed that the parent worms had been lodged in the thoracic duct; had caused inflammation and obstruction, followed by general dilatation of the lymphatics; and that the chyluria was connected with the dilated lymph-channels in the kidneys.

It now remains to discuss briefly the ætiology and pathology of chyluria. The complaint is met with almost exclusively in tropical and sub-tropical climates. Rare instances have been recorded in which it originated in Europe, one by Dr. William Roberts in Lancashire, another by Dr. Beale in Norfolk.



The principal views entertained as to the pathology of chyluria are as follows:—1. That it is but a symptom of *piarhæmia* or fatty blood, which is but the normal condition of the blood after food, aggravated and made permanent by derangement of the digestive organs, especially the liver (Bernard, Robin). 2. That there is a direct communication between the lymphatics and the urinary tracts, and consequent leakage of chyle (Vandyke Carter). 3. That there may be a hypertrophy of the lymphatic channels in some part of the urinary tract, which subsequently acquire the properties of glands (W. Roberts). 4. That chyluria depends upon the *filaria sanguinis hominis*. This view was originally advanced by Dr. Lewis, and it has since received much support. The *filariæ* are found in abundance in the urine and blood in cases of chyluria, and also in chylo-lymphous discharges; and they have been detected shortly before the condition has manifested itself. How they act has not been determined. It may be that they cause the delicate walls of the lymphatics to rupture; and Dr. W. Roberts suggests that aggregations of *filariæ* may lead to this result. Another notion is that they give rise to derangements of the liver and other organs, which lead to *piarhæmia*. Dr. Lewis further suggests that they may in some way tend to the production of minute secreting structures, along the urinary tract or in other situations, which might permit of the filtration of the ordinary nutritive fluids of the body in a more or less modified condition.

So far as can be determined from the present state of knowledge, it seems that chyluria is certainly connected in some way with the *filariæ* in many cases, but that in some instances it is independent of any parasite. It may be affirmed that the condition does not depend on any structural disease of the kidney or other organ. Chyluria occurs at all ages, from childhood to extreme old age; and rather more frequently in females than males (Lewis).

**SYMPTOMS.**—Chyluria presents an extremely varied clinical history, and the descriptions given of cases are most diverse. Its course is marked by an irregularity and capriciousness which cannot be explained. The only constant symptom is the presence of so-called *chylous* urine. This fluid usually presents a peculiar whitish, opaque, milky appearance, which disappears when it is shaken up with ether. Sometimes the urine is not chylous, but lymphous. In some instances it is of a pink colour, from the presence of blood, but Dr. Lewis states that more commonly, at least in India, the blood, when present, is seen forming an adherent coagulum at the bottom of the vessel. In some countries, however, and occasionally in India, hæmaturia is the prominent feature in connection with *filaria*. Chylous urine gives out a strong milky or whey-like odour, which is increased by heat. A semi-solid tremulous coagulum forms speedily after standing, resembling blanc-mange. This soon breaks down, and forms a creamy scum; while the urine rapidly decomposes. The specific gravity varies greatly, even in the same individual at different times; and the appearance of the urine also differs at different periods of the day, and in relation to food. Chylous urine contains the constituents of chyle or lymph, namely, fibrin, albumen, and fat, which are indicated by the ordinary tests. Their relative proportion varies much; and it does not correspond to that of either of the nutritive fluids of the body, but appears to be nearest to that of lymph. As a rule all the constituents are scanty in the morning before meals, unless the circulation has been quickened by exercise or in

other ways, when the albumen is increased, but not the fat. This element is usually most abundant shortly after meals; occasionally it is chiefly observed in the morning. Microscopic examination of chylous urine reveals fat in the molecular form, leucocytes, and red corpuscles in some cases; it seldom, if ever, presents any casts. Filariae are also often present. The ordinary urinary constituents are generally in deficient proportion to the amount of urine passed.

The discharge of chylous urine usually occurs very suddenly; it may be constant, but more frequently is intermittent; and after its occurrence may cease for years or permanently. In most cases symptoms referable to the urinary organs are noticed, such as uneasiness across the loins, over the hypogastrium, along the urethra, and especially towards the perinæum in males. Occasionally chylous urine coagulates in the bladder, causing more or less discomfort in micturition, or not uncommonly suddenly stopping the flow of urine during the discharge of the clots, which become visible after their escape. Patients suffering from chyluria may enjoy good health, but generally there is marked debility and wasting, with mental depression, owing to the continuous drain of the nutritive fluid. The blood seems to vary in its composition, according to the analysis of different observers; and it often contains filariæ in abundance. The serum and corpuscles do not, according to Dr. Lewis, present any abnormality indicative of the presence of fatty matter in any form. Occasionally chyluria is associated with chylous discharges from various parts of the body; with elephantiasis lymphangiectodes; or with true elephantiasis.

Chyluria usually runs a very chronic course. Occasionally patients apparently in fair health have been known to die very unexpectedly from no recognized acute disorder. After apparent recovery, the complaint will probably return again and again, even after a complete change of climate and avocation (Lewis).

TREATMENT.—Medicines seem to have but little effect on chyluria, but the complaint is sometimes cured spontaneously. The chief drugs which have been employed or recommended in its treatment are tincture of iron; *astringents*, especially large doses of gallic acid; and large doses of iodide of potassium. Dr. William Roberts quotes a case which was benefited by decoction of mangrove bark, which has a reputation in Guiana. Salt-water baths may be used; and it seems best to restrict animal diet, at the same time giving nutritious food. Rest may be of service.

#### IV. HÆMATURIA.

ÆTIOLOGY.—The blood in hæmaturia may come from the kidneys; from their pelves or infundibula, or the ureters; from the bladder; from the urethra; or, in females, it may be connected with uterine or vaginal hæmorrhage, including ordinary menstruation. Excluding the latter, the causes of hæmaturia may be arranged thus:—1. *Traumatic*, for example, external injury affecting any part of the urinary apparatus; severe exertion and straining; injury by instruments; and laceration of the mucous membrane of the pelvis of the kidney, ureter, bladder, or urethra by a calculus. 2. *Renal affections*, namely, congestion, including the active hyperæmia induced by certain articles, especially turpentine and cantharides; acute Bright's disease; suppurative nephritis; cancer; tubercle; renal embolism; minute calculi in the tubules; hydatids and

other parasites. 3. *Affections of either pelvis or ureter*, including cancer, tubercle, and parasitic diseases. 4. *Affections of the bladder*, namely, congestion; acute cystitis; cancer, especially if of a villous and fungous nature; and varicose veins. 5. *Gonorrhœa* and other *urethral inflammations*. 6. *Endemic*. This calls for special notice, being a form of hæmaturia observed in certain hot climates, especially the Mauritius, which has now been proved to be due to a small parasite—*Bilharzia hæmatobia*—affecting the mucous membrane of the pelvis of the kidney and of the bladder. In other cases hæmaturia depends on the *filaria sanguinis hominis*; or possibly upon other parasites. 7. *Abnormal conditions of the blood*, particularly in connection with purpura and scurvy; but also in malignant fevers, cholera, and other affections. 8. *Vicarious*, chiefly of the menstrual discharge. 9. *Mental emotion* in rare instances, it is said.

**SYMPTOMS AND DIAGNOSIS.**—For purposes of diagnosis it is highly important to notice whether blood present in urine is passed constantly, or only at intervals, or under particular circumstances, as after riding, jolting, or taking certain articles of food; the mode of its discharge, whether before or after the urine, or along with it, and also if it escapes independently of micturition; its amount; and the degree in which the urine and blood are mingled, whether they are intimately mixed, or more or less separate, or if the blood forms distinct coagula.

The characters of hæmaturia associated with most of the local lesions mentioned above will be hereafter pointed out. At present all that need be said is, that in *renal* hæmaturia the blood and urine are intimately mixed, the colour being frequently smoky, while under the microscope minute moulded coagula or blood-casts are usually visible, being in some cases numerous and decolorized, and accompanied with other renal structures; in bleeding from the *renal pelvis* or *ureter* there is also an intimate admixture, and moulded vermiform coagula of considerable length may be discharged; in *vesical* hæmorrhage the blood is expelled chiefly or only towards the end of the act of micturition; while in the *urethral* variety it may escape or be pressed out independently of micturition, and when urine is passed, blood precedes it or colours the first portion, and then the urine may become quite clear, blood again appearing at the close of the act. It may, however, flow back into the bladder from the urethra, thus colouring the urine contained in this organ. Blood is sometimes purposely mixed with urine by hysterical patients and malingerers.

The *diagnosis* of hæmaturia may be further aided by a consideration of the history of the case, and of the previous symptoms, which might reveal some cause, such as a calculus; the seat of local urinary symptoms, whether pointing to the kidney, bladder, or urethra; the results of thorough *physical examination*; and the general symptoms present.

**TREATMENT.**—This must be conducted on similar principles to those followed in the treatment of other hæmorrhages. The most valuable internal *astringents* in hæmaturia are gallic or tannic acid, pyrogallie acid, oil of turpentine, acetate of lead, hamamelis, iron, alum, or full doses of dilute sulphuric acid combined with opium. The subcutaneous injection of ergotine deserves trial. The local use of *cold* is also often highly beneficial, in the form of ice applied to the loins, hypogastrium, or perinæum, or of cold injections into the bladder. *Astringent* injections are permissible in some forms of vesical hæmorrhage. Dry-cupping over



the lumbar regions is frequently very useful when the blood comes from the kidneys; and occasionally local removal of blood is desirable. Pressure can be applied in the case of urethral hæmorrhage, and for this purpose it may be requisite to pass a catheter or sound. After the occurrence of renal hæmaturia it is important to watch the case for some time, as coagula may remain in the tubules and thus set up serious mischief.

#### V. HÆMATINURIA—HÆMOGLOBINURIA.

**ÆTIOLOGY AND PATHOLOGY.**—The urine occasionally contains more or less of the colouring matter and albumen of the blood, but no corpuscles or fibrin, and to this condition the term *hæmatinuria* has been applied. It has been observed in connection with septic and malignant fevers; occasionally in purpura and scurvy; after poisoning by arseniuretted hydrogen or carbonic anhydride; and as a distinct affection, named *paroxysmal* or *intermittent hæmatinuria*. This complaint has been met with almost entirely amongst adult males. In nearly every case it seems to have followed exposure to cold or draughts. Occasionally it has been attributed to injury over the renal region; or to malaria, some of the patients having previously had ague. With regard to the *pathology* of paroxysmal hæmatinuria, some regard it as due to a diseased condition of the blood, the red corpuscles becoming disintegrated and dissolved; others consider that the kidneys are at fault. There is no organic lesion of these organs, but probably an intense congestion occurs before the paroxysms, due to vaso-motor disturbance, leading to temporary dilatation of the renal vessels, with the consequent escape of some of their contents, without rupture. Chilling of the skin is supposed to be the immediate cause of this vascular disturbance in the kidneys. It has been stated that paroxysmal hæmatinuria has some relation with oxaluria, ague, and rheumatism. It is highly probable that in some of the cases described corpuscles have been present in the urine originally, which have subsequently broken down and become dissolved, and, indeed, blood-cells have been seen when the urine has been examined immediately after its discharge.

**SYMPTOMS.**—*Intermittent hæmatinuria* comes on in sudden and usually irregular paroxysms, varying much in their frequency in different cases, only occurring during the day, and generally lasting from three to twelve hours; being preceded for a brief period by chills or rigors, languor, a sense of weight or dull pain over the kidneys, severe aching pain or stiffness in the legs, occasionally retraction of the testicles, and nausea or vomiting. In some cases the initial symptoms consist of immoderate yawning and stretching of the limbs. The attacks are usually clearly traceable to exposure to cold, and may come on during such exposure, but it may be very slight. The symptoms may, however, be very severe, the patient feeling extremely cold, presenting marked pallor or duskiness, and the pains being very prominent. As a rule there is no pyrexia, and the temperature often falls below the normal at first, as much as 2° or 3°. The urine becomes in a short time very dark, resembling porter or port wine; usually turbid; generally faintly acid; of variable specific gravity; and highly albuminous; while it deposits an abundant chocolate-coloured, grumous sediment, which microscopically is seen to consist chiefly of granular matter, sometimes mixed with hæmatine crystals, and often with a few dark granular or hyaline casts, and crystals of oxalate of lime. Spectroscopic examination shows

two absorption bands between the orange and green portions of the spectrum, characteristic of the presence of oxy-hæmoglobin; and Drs. Forrest and Finlayson of Glasgow have reported cases in which they noticed in addition "a third, somewhat narrow, absorption band about the middle of the red in the spectrum," which they regard as due to the presence of meta-hæmoglobin, though they are not certain as to the nature of this substance. When the urine comes to present its abnormal characters, the general symptoms rapidly abate, and the patient feels quite well in a few hours; there may be a slight rise of temperature. In the intervals the urine seems to be quite natural; and the change to the healthy condition may be equally sudden with the onset of the paroxysm, but it is usually gradual. While generally irregular, the paroxysms sometimes present a marked periodicity. They may come on once or twice a day, once or twice a week, or less frequently; and may cease altogether in warm weather. If they are frequent, the patient tends to become weak and anæmic, but there may be no marked deterioration of the general health even in prolonged cases.

TREATMENT.—Full doses of quinine and tincture of iron have been found of most service in the treatment of hæmaturia. Other remedies employed are arsenic, gallic acid, acetate of lead, digitalis, and ergot of rye. The patient should wear warm clothing, with flannel next the skin; protect the loins and feet especially; and avoid every cause of cold. During the paroxysm he should be kept warm in bed.

## CHAPTER LVIII.

### URÆMIA—URÆMIC POISONING.

ÆTIOLOGY AND PATHOLOGY.—*Uræmia* is the term applied to a group of symptoms which are liable to occur as the result of grave interference with the urinary functions. The conditions under which they arise may be stated generally as:—1. *Diseases of the kidneys*, especially Bright's disease; or nervous and vascular derangement of these organs, which prevents them from performing their excretory work properly. 2. *Obstruction of both ureters*, so that the urine which is formed cannot escape into the bladder, though it is a remarkable fact that the symptoms are by no means characteristic in these cases. 3. *Retention of the urine in the bladder* from any cause, when the uræmic state is supposed to be partly due to re-absorption of the urinary constituents.

Most authorities regard the phenomena of uræmia as due to the accumulation in the blood of poisonous materials, the circulation of which through the nervous and muscular systems occasions the effects characteristic of this condition. Formerly they were attributed to non-excretion of urea, or to the decomposition of this substance, and the consequent formation of carbonate of ammonia; more recent observations, however, seem to indicate that the chief poisonous agents are materials produced as the result of imperfect tissue-metamorphosis, which in the normal course of events should be further converted into urea and uric

acid, and then excreted. The watery state of the blood; and œdema with anæmia of the brain substance, have also been made to account for the phenomena of uræmia.

**SYMPTOMS.**—The clinical phenomena which may be met with indicative of uræmia are headache, sometimes fixed behind the neck or at the back of the orbits, or a sense of weight and pressure over the forehead or vertex; vertigo; increased irritability of the voluntary muscles, evidenced by muscular twitchings or fits of epileptiform convulsions, in the latter the face being pale, and the pupils dilated, several fits sometimes occurring in rapid succession, with more or less stupor in the intervals, though consciousness is often partially restored; cerebral disturbance, usually in the direction of drowsiness, heaviness, and confusion of ideas, culminating in stupor or profound coma, delirium being only exceptionally observed; disturbance of vision, in the way of dimness of sight from time to time, or actual temporary blindness, there being no necessary organic changes revealed by the ophthalmoscope; deafness in rare instances; vomiting and diarrhœa, the matters discharged containing a quantity of ammonia, which may be perceptible to the smell; occasionally an urinous or ammoniacal odour of the breath and sweat; and in exceptional cases paroxysms of dyspncea. The exact combination of symptoms in any individual case varies considerably, as well as the mode and rapidity of their onset. As a rule they come on gradually, beginning with headache and vomiting. Occasionally uræmia is revealed by a sudden apoplectic or epileptiform attack; by blindness; or by severe vomiting.

**DIAGNOSIS.**—Certain cases of uræmia are particularly liable to be mistaken for those of apoplexy, epilepsy, or opium-poisoning. The diagnostic points by which it is distinguished from these conditions will be considered in a future chapter; at present I would only draw attention to the great importance of testing the urine in all cases of sudden or unexplained insensibility. Cases of belladonna-poisoning may also simulate uræmia. The possibility of headache or giddiness, disturbances of sight or hearing, and vomiting or diarrhœa being due to this cause, must not be forgotten.

**TREATMENT.**—In the uræmic state the main indications are to remove any cause of urinary obstruction; to use measures for promoting excretion of urine, especially free dry-cupping, or the application of heat and moisture over the loins; to encourage the action of the skin by the aid of warm, vapour, or hot-air baths; and to treat symptoms. Inhalation of chloroform is useful during the epileptiform attacks. Venesection is often employed for apoplectic seizures, but is not admissible should there be advanced disease of the kidneys. Sinapisms may be applied to the nape of the neck and limbs. Vomiting must be treated in the usual way; but it is not advisable to check diarrhœa too speedily, and a brisk *purgative* is not unfrequently very serviceable, such as a full dose of compound jalap powder.

---



## CHAPTER LIX.

## RENAL CONGESTION—EMBOLISM—INFARCTION.

**ÆTIOLOGY.**—Congestion of the kidneys may be *active* or *mechanical*. *Active* renal congestion, also named *catarrhal nephritis*, may result from:—1. Any pyrexial condition, but especially that accompanying the exanthemata. 2. Exposure to cold and wet. 3. The use of certain medicines in excess, namely, cantharides, turpentine, nitre, cubebs, or copaiba. 4. Irritating urine in connection with diabetes. 5. Morbid formations in the kidney; or emboli. 6. The early stage of inflammation. 7. It is said hypertrophy of the left ventricle; or, possibly, active dilatation of the vessels, as in cases of hysteria. *Mechanical* hyperæmia is a common consequence of:—1. Some cardiac or pulmonary disease interfering with the general venous circulation. 2. Pressure upon one or both renal veins, or upon the inferior vena cava above their point of junction, by an abdominal tumour or a pregnant uterus.

**ANATOMICAL CHARACTERS.**—At first the kidneys present the characters ordinarily accompanying congestion, namely, enlargement and increase in weight; increased redness, with points of vascularity, especially corresponding to the Malpighian bodies; and sometimes minute ecchymoses. In many forms of *active* hyperæmia there is a catarrhal state of the ducts of the pyramids, with shedding of their epithelium. After *mechanical* congestion has lasted for some time the usual pathological changes are set up, leading ultimately to grave disorganization of the kidneys, these organs becoming contracted, indurated, and sometimes granular or irregular, while their cortical substance undergoes more or less atrophy. Microscopic examination reveals alteration in the shape of the tubes, with thickening of their walls; changes in the epithelium, which is often destroyed; increase in the intertubular connective tissue; and permanent distension of the minute vessels. By some pathologists this condition is looked upon as a form of Bright's disease.

*Emboli* not unfrequently lodge in the kidneys, and give rise to *infarctions*, which are almost invariably confined to the cortical portion; differ in size; and are usually well-defined and wedge-shaped, with the base towards the surface. At first they present a dark-red colour, but become decolorized from the centre towards the circumference, leaving yellow masses, which may be ultimately absorbed, depressed cicatrices alone remaining. Rarely an infarction softens and breaks down, forming a pseudo-abscess; or it is said that even actual pus may be produced.

**SYMPTOMS.**—Congestion of the kidneys is ordinarily indicated by the urine becoming diminished in quantity, high-coloured, and concentrated, and depositing urates abundantly on standing; while it afterwards contains some albumen, occasionally a little blood or clear fibrinous casts, with a few renal epithelium-cells. In some forms of *active* hyperæmia, however, a copious flow of pale and watery urine takes place, which is of low specific gravity. There may be a sense of fulness about the loins, or even a certain degree of heavy pain, and tenderness is frequently com-

plained of. If the congestion subsides, the urine assumes its normal characters; but if it continues and leads to organic changes in the kidneys, this fluid presents more marked and permanent changes, which will be hereafter considered. As a rule there are no symptoms indicative of *renal embolism*. If the embolus is large, its lodgment may cause a sudden severe pain in the renal region, shooting towards the pelvis; followed by albuminuria or hæmaturia. Should an abscess form in the kidney after embolism, symptoms indicative of this event might set in.

TREATMENT.—If renal congestion calls for any positive interference, the main indication is to remove or mitigate its cause as soon as possible, especially in the *mechanical* form. Rest in the recumbent posture; free dry-cupping, or the application of heat and moisture over the loins, or in some cases the removal of a little blood from this region; the use of the warm bath; and active purgation, are the chief direct remedies to be employed.

## CHAPTER LX.

### SUPPURATIVE INFLAMMATION IN CONNECTION WITH THE KIDNEYS.

#### 1. SUPPURATIVE NEPHRITIS—RENAL ABSCESS.

ÆTIOLOGY.—The causes of renal inflammation ending in suppuration are:—1. *Injury* from without. 2. Some *direct irritation* in the substance of the kidney, especially from a calculus. 3. *Suppuration in the bladder or urinary passages*, that in the kidney being set up either by extension or independently, the latter being probably the result of a kind of local pyæmia. 4. *Embolism*. 5. General *pyæmia*. 6. *Extension* of inflammation from surrounding structures.

ANATOMICAL CHARACTERS.—Whatever may be the origin of the inflammation, the alterations in the kidney are in most cases similar at first, namely, enlargement of the organ; hyperæmia, much blood flowing on section; and diminution in consistence. It is supposed that an interstitial exudation then forms. Suppuration usually commences in separate points, which extend and coalesce so as to form one or more abscesses, these varying much in size. In most forms of the disease only one kidney is usually involved, and there is finally but a single abscess, which may attain large dimensions. If not opened, it generally bursts either into the pelvis of the kidney; externally in the loins; into the peritoneum or sub-peritoneal tissue; into the intestines; or into the thorax. Occasionally inspissation of the contents takes place, followed by caseation and calcification, a cure being thus effected. In pyæmia numerous scattered abscesses of small size are observed. It is said that pus is sometimes infiltrated through the kidneys; and also that it may form within the tubules.

SYMPTOMS.—Acute suppurative nephritis is generally accompanied with local pain in the corresponding lumbar region, often severe, increased by movement, and frequently shooting towards the bladder, testis, or thigh; as well as with tenderness. The testis may be drawn

up. The urine is diminished in quantity and concentrated, or even actually suppressed; and it frequently contains some blood or merely a little albumen, but these elements may be quite absent. As a rule distinct rigors usher in the complaint, followed by marked pyrexia, which has a great tendency to assume a typhoid type, especially when suppuration commences, this being accompanied with repeated shiverings. Sympathetic vomiting is not uncommon. Uræmic symptoms are also liable to arise. Should a large abscess form, it presents as an elastic or fluctuating fulness or tumour, usually in the lumbar region, where it may afterwards burst. If it opens into the pelvis of the kidney, a copious discharge of pus takes place along with the urine, and this may afterwards continue, either persistently or at intervals. Various symptoms may result from the bursting of a renal abscess into other parts. When the kidneys are involved in pyæmia there are no prominent local signs; and such is often the case when renal inflammation follows some morbid condition of the urinary passages, when it also frequently runs a somewhat chronic course.

## II. PYELITIS—INFLAMMATION OF THE PELVIS OF THE KIDNEY— PYONEPHROSIS.

ÆTIOLOGY.—Pyelitis signifies inflammation involving the mucous lining of the pelvis and infundibula of the kidney. Its important causes are:—1. *Direct irritation* by foreign matters lodged in the renal pelvis, especially a calculus or gravel, parasites, and blood-clots. 2. *Morbid deposits* in the membrane, namely, cancer or tubercle. 3. *Extension* of inflammation from the bladder along the ureter. 4. Irritation by *accumulated urine*, resulting from closure of the ureter owing to pressure or internal obstruction, especially if this urine has become decomposed. 5. In rare instances pyelitis seems to be set up as an *idiopathic* affection, from exposure to cold and wet, or other injurious influences. 6. A certain degree of catarrh of the renal pelvis and infundibula may also arise in the course of *other diseases*, especially various febrile affections, organic affections of the kidneys, and diabetes; or from the use of certain *drugs*, such as turpentine or cantharides.

ANATOMICAL CHARACTERS.—Pyelitis may be *acute* or *chronic*, and the appearances vary accordingly. The *acute* form, in which the inflammation is usually *catarrhal*, is characterized by injection of the lining membrane, occasionally with slight ecchymoses or extravasations of blood; relaxation and softening; shedding of epithelium; and the subsequent discharge of a purulent mucus, or of actual pus. Occasionally diphtheritic or croupous inflammation is observed, along with a similar condition of other mucous surfaces. The *chronic* variety may either follow the acute, or commence independently. The membrane is then pale, though some of its veins may be permanently distended; often grey or slate-coloured from pigment; much thickened; and unusually firm. Pus is constantly formed, and if there is no obstruction it flows away with the urine; should there, however, be an impediment to its escape, the pus accumulates in the pelvis of the kidney, which it distends more and more, giving rise to the condition named *pyonephrosis*; here it is commonly mixed with other materials, such as urine, which is usually decomposed and ammoniacal, deposits or incrustations of uric acid and urates or phosphates, calculi or other materials which have



excited the pyelitis, or blood. By degrees the substance of the kidney is compressed and invaded upon, until ultimately the organ may be completely destroyed, a mere sac remaining, containing pus and other matters. In other instances distinct disease is set up in different parts of the kidney—*pyelo-nephritis*; or it may simply shrivel up and become atrophied. The accumulation may burst in any of the directions which renal abscess takes; or occasionally ulceration of the mucous membrane is set up by some foreign body, and perforation occurs before any particular distension of the pelvis is observed. In some cases the pus becomes inspissated, and abundant calcareous deposits are formed, or even imperfect bone, the cavity contracting and shrivelling up.

**SYMPTOMS.**—In the majority of cases pyelitis is preceded by, or accompanied with, symptoms due to its cause, for instance, those of calculus or disease of the bladder. The *local* clinical phenomena associated with this complaint are uneasiness or pain over one or both lumbar regions, often of an aching character, or shooting downwards; tenderness; a sense of local weakness; generally frequent micturition; changes in the urine; and in some instances the presence of a renal enlargement. The alterations in the urine may be the only deviation from health. This fluid is often increased in quantity; generally acid; and at first contains a little blood intimately mixed with mucus and the variously-shaped epithelium-cells detached from the pelvis and infundibula; gradually it becomes mixed more and more with pus, until finally this morbid product may be present in large quantity, and it comes away persistently so long as no obstruction exists to prevent its escape. Albumen is only observed in proportion to the admixture of blood and pus. Some important differences are noticed in the characters of the urine under certain circumstances. If the flow of pus along the ureter is impeded in any way, as by the lodgment of a calculus, the urine may become quite natural, provided only one kidney is involved; if both are implicated, or if the closure of the ureter is incomplete, the quantity of pus is merely lessened. Should the obstruction be removed, a copious flow of purulent urine again takes place suddenly; this course of events may be repeated from time to time, or the obstruction may remain permanently. Further, if the urine is retained in the renal pelvis, it tends to decompose, and is then frequently discharged in an ammoniacal state. Should the different fluids accumulate here, a fulness or tumour is produced, having the general characters of a renal enlargement, but presenting an elastic or fluctuating feel. This fulness will increase in size from time to time should the ureter become obstructed, being then also more painful and tender, and it may suddenly subside when the impediment is removed. Occasionally the enlargement attains very large dimensions.

The *general* symptoms are those of pyrexia in the acute form of pyelitis, preceded by rigors. When suppuration is set up, there are commonly repeated rigors, in some cases recurring at regular intervals; and in prolonged cases signs of hectic fever appear. The bowels are often disturbed, there being either diarrhoea or obstinate constipation, the latter resulting from pressure on the colon. If the kidneys become independently implicated, symptoms indicative of Bright's disease set in. In some cases recovery takes place, provided only one kidney is affected, and the cause of the complaint can be removed, though often with complete destruction of the involved organ. Most commonly, however, death ultimately ensues from gradual exhaustion. This event

may also result from perforation or rupture of the distended pelvis, the symptoms differing according to the direction in which the opening takes place, and the structure with which the purulent collection communicates.

### III. PERINEPHRITIS.

In *perinephritis* the tissue surrounding the kidney becomes the seat of inflammation, the process usually terminating in suppuration. It may be caused by injury; exposure to cold; or by previous suppurative nephritis or pyelitis. Clinically it presents a history very much like that of the diseases just mentioned, but is distinguished from these affections by the absence of any marked disturbance of the renal functions, or of any changes in the urine. There may also be a greater intensity and superficialness of the pain and tenderness, with more marked exacerbation on movement; and subcutaneous œdema over one lumbar region may be observed. The purulent accumulation generally opens posteriorly, but may rupture in various other directions.

#### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. DIAGNOSIS.—The inflammatory affections just described are generally attended with much pain referred to the renal region; functional disturbance of the kidneys, except in the case of perinephritis; and pyrexia. They can in most instances be distinguished by the circumstances under which each occurs; and by the characters of the urine. Acute Bright's disease is diagnosed from these affections by the dropsy and other characteristic symptoms which accompany it; as well as by the conditions of the urine. In *pyelitis* the presence in the urine of epithelium from the pelvis and infundibula is highly important; while later on pus appears. *Suppurative nephritis* may give rise to the physical signs of an abscess in the renal region. *Perinephritis* is, as a rule, but not invariably, distinguished by the absence of any morbid characters of the urine. *Active renal congestion* might possibly be mistaken at first for some of the inflammatory affections, but the milder character of the symptoms, and their speedy subsidence, sufficiently characterize this condition. *Acute cystitis* sometimes simulates renal inflammation.

2. PROGNOSIS.—Suppurative inflammatory diseases in connection with the kidney are very dangerous, on account of the interference with the renal functions; the constitutional disturbance which they occasion; and the dangers incident to the rupture of any purulent accumulation. *Pyelitis* differs much in its gravity according to its duration; its cause; and whether it is single or double. When confirmed, this complaint is always serious, but even after complete destruction of the kidney recovery may follow, provided only one organ is involved. Calculous pyelitis is much more favourable than that which follows chronic disease in the lower urinary passages; or than that which is excited by tubercle or cancer.

3. TREATMENT.—The general measures applicable at the outset in all the forms of *acute renal inflammation* are to keep the patient in bed, completely at rest; to cup over the loins to the extent of from 6 to 10 or 12 ounces, if the case is favourable, or otherwise to dry-cup freely; to

apply hot poultices or fomentations constantly over the lumbar region ; to give low diet, with abundance of diluent drinks ; and to act freely upon the bowels. In *suppurative nephritis* or *perinephritis*, should signs of an abscess appear, this should be encouraged towards the surface, and the pus evacuated when the proper time arrives, for which purpose the *aspirateur* is most serviceable. At this time abundant support, along with *stimulants*, may be required, especially if typhoid symptoms should set in. In *pyelitis* it is very important to remove the cause of the disease if possible ; and when the complaint is due to a calculus, a considerable amount of opium, or free subcutaneous injection of morphia is often needed, in order to relieve the pain. The measures requisite for the treatment of purulent discharge in the urine have already been considered.

## CHAPTER LXI.

## BRIGHT'S DISEASE.

NUMEROUS and diverse have been the classifications adopted by different authors of the morbid conditions included within the term *Bright's disease*. In general language it is commonly employed to signify any structural disease of the kidneys accompanied with albuminuria and dropsy, but these symptoms are not essential. Primarily Bright's disease is divided into *acute* and *chronic* forms, under which its several varieties may be discussed.

## I. ACUTE BRIGHT'S DISEASE—ACUTE DESQUAMATIVE NEPHRITIS—ACUTE TUBAL NEPHRITIS—ACUTE INTERSTITIAL NEPHRITIS.

**ÆTIOLOGY.**—In the large majority of cases acute Bright's disease is associated with scarlatina ; or results from "taking cold." It may follow excessive drinking ; or is occasionally set up in the course of other exanthemata besides scarlet fever, for example, measles or typhus fever ; in the collapse-stage of cholera ; after ague or exposure to malaria, it is affirmed ; or during pregnancy. There is good reason for believing that the *pathological* cause in many of these conditions consists in undue exercise of the functions of the kidneys, these organs having to excrete materials which are either formed in excess or of an unusual character, or such as ought to be removed by the skin. The chief *predisposing* causes are the early period of life ; want of cleanliness of the skin ; intemperate habits ; and occupations which involve exposure to wet and cold. In some cases Bright's disease probably exists in a chronic form without giving rise to any evident symptoms, until one of the above causes leads to an acute exacerbation.

**ANATOMICAL CHARACTERS.**—Acute Bright's disease is usually of the nature of tubal or desquamative nephritis, the morbid changes taking place in connection with the renal tubes. They are of various degrees of intensity, and the inflammation may be sub-acute, rather than acute. The kidneys present obvious changes. They are enlarged and



increased in weight, in most cases considerably. At first they are deeply congested, the surface and a section presenting a deep dusky-red colour, with darker spots corresponding to the Malpighian corpuscles or to minute hæmorrhages; while the superficial veins are distended, and a quantity of bloody fluid escapes on section. The surface is quite smooth, and the capsule is easily separated. The enlargement is mainly due to increase in the cortical substance, which is found to be much thickened, softened, lacerable, and friable. The pelves and infundibula are also injected, and a bloody liquid is often found here. In a more advanced stage the colour of the cortical portion changes, usually in a short time, becoming either more or less white, yellowish-white, or pale-buff, as well as opaque and dotted, or presenting a mottled surface of red and white; while the pyramids remain dark-red and striated, red lines radiating in a fan-like manner from their bases. If the process subsides quickly in the early stage, the kidney may completely recover, the inflammatory products passing away in the urine.

Microscopic examination reveals capillary distension; and the presence of fibrinous exudation within the tubules of the kidney, along with red blood-corpuscles, and extremely abundant epithelium-cells, which become detached, rapidly increase in number, and accumulate in the tubules, many of which appear crammed full of cells, while some of them are dilated. Most of the epithelial particles are altered in their characters, presenting the condition known as "cloudy swelling," being enlarged, cloudy or opaque, and more or less granular from the presence of protein granules or sometimes of fat; or they may be quite disintegrated. Abundant young cells are also present, resulting from proliferation. They accumulate more and more as the disease advances, which mainly accounts for the pale colour, this also partly resulting from compression of the vessels. In some tubules the epithelium may be completely detached, only hyaline fibrinous moulds remaining. These changes are much more evident in the cortical than in the pyramidal portion of the kidneys.

The special characters of the changes in the kidney in connection with scarlatina—*scarlatinal nephritis*—have already been described, and also the condition named *glomerulo-nephritis* by Klebs. (See page 141.) Peculiar appearances have been observed in cases of fatal puerperal eclampsia by Dr. Angus Macdonald and Dr. Hamilton, the epithelium-cells being small, and their nuclei abnormally distinct; while many tubes were blocked with a peculiar hyaline or colloid material.

When the inflammatory process is prolonged in cases of acute Bright's disease, or when repeated attacks of sub-acute inflammation occur, the intertubular connective tissue always becomes involved, an interstitial nephritis being set up.

In fatal cases of acute Bright's disease morbid appearances due to *complications* are commonly observed, especially serous inflammations; endocarditis; pneumonia or bronchitis. Dropsy and its consequences are also generally present. In some cases the heart is hypertrophied.

**PATHOLOGY.**—Acute Bright's disease consists, as a rule, essentially in an intense *catarrhal inflammation* of the tubules of the kidney. As a result, in addition to capillary engorgement and rupture, the epithelial cells undergo rapid proliferation. In some cases there is more or less *interstitial nephritis*. The renal functions are greatly impeded, and hence the blood becomes overloaded with excrementitious matters, as well as unusually watery, and highly deficient in albumen and red

corpuscles. The elements formed in the kidneys are washed away by the urine, in which they are visible on microscopic examination.

**SYMPTOMS.**—As a rule the onset of acute Bright's disease is definite and marked. Frequently the complaint sets in with chilliness or rigors, general pains, headache, and nausea or severe vomiting; in other cases dropsy rapidly extending is the earliest symptom observed; and occasionally uræmic phenomena first attract attention. When the disease is established, the characteristic clinical signs include peculiar changes in the urine; more or less general anasarca, frequently accompanied with effusion into serous cavities and œdema of organs; extreme pallor, puffiness, and dryness of the skin; a tendency to uræmia, serous inflammations, endocarditis, pneumonia, or bronchitis; and pyrexia. Generally some degree of dull pain, with tenderness, is experienced over the renal regions, but these sensations are not prominent; micturition also is in most cases too frequent, especially at night, though the quantity of urine passed is greatly diminished, and sometimes this excretion is almost or quite suppressed. The urine which is discharged has the following characters:—It is dark in colour, from excess of pigment, and from the presence of blood, the latter often causing the urine to assume a smoky, brown, or dark-red tint; the specific gravity is high—1025 to 1030, 1040, or more; the reaction is almost always acid; the normal odour is replaced by one compared to that of beef-tea, or to the washings of flesh, or whey; an abundant sediment forms, brown and flocculent; while urates are frequently deposited. Chemical examination reveals abundance of albumen, the urine sometimes becoming almost solid on boiling. The excretion of urea and inorganic salts is greatly diminished, but uric acid is about normal. Under the microscope the deposit is seen to consist of red blood-corpuscles, in some cases much altered in their characters; renal epithelium-cells, usually more or less swollen, cloudy or granular, or partially disintegrated; remnants of these cells, in the form of nuclei or granular matter; extra-renal epithelium; amorphous particles of fibrin; and numerous casts, chiefly of the *blood* and *epithelial* varieties at first, and of medium size, with a few large or small *hyaline*, and some opaque *granular* casts. The casts change during the progress of a case of acute Bright's disease, and it is very important to study these alterations; not uncommonly a little fat appears in connection with the casts and with the epithelium, which disappears as the disease subsides. The presence of "white cell casts" has been regarded by Dr. George Johnson as a sign of *glomerulo-nephritis*.

Dropsy often comes on with great rapidity in acute Bright's disease, in some cases rendering a patient irre recognizable in a few hours; while the face assumes a characteristic blanched, pasty, and puffy aspect. Hydrothorax, ascites, and œdema of the lungs are common; while œdema glottidis sometimes proves highly dangerous. The patient generally feels dull and heavy, or complains of headache, distinct uræmic symptoms being also liable to supervene at any time. Inflammatory complications will be indicated by their special signs, those chiefly to be borne in mind being pericarditis, pleurisy, peritonitis, endocarditis, bronchitis, and pneumonia. Fever is often high, with a full hard pulse; while there is complete loss of appetite, great thirst, and usually constipation. The blood is hyperinotic.

The late Dr. Sibson drew special attention to certain signs in connection with the heart and vessels in cases of Bright's disease, and in the acute form he frequently noticed the following phenomena:—

tension and hardness of the radial artery; a second beat over the aorta in the first and second right intercostal spaces; an intensified metallic second sound and muffled first sound over the aorta; reduplication of the first sound, variously distributed, but usually best heard over the septum ventriculorum; and in most cases a doubled second sound. The left ventricle was hypertrophied in many instances, but in others this effect of Bright's disease was prevented by the co-existence of some wasting and exhausting complaint. The correctness of these observations has been abundantly confirmed, and the signs in connection with the vascular system in cases of acute Bright's disease are now generally regarded as of considerable importance.

COURSE, DURATION, AND TERMINATIONS.—Acute Bright's disease presents considerable variations in these respects. *Recovery* may follow, either speedily or gradually. Complete restoration is indicated by disappearance of the dropsy; by subsidence of pyrexia, and return of the functional activity of the skin; and by the urine becoming abundant, clear, and of low specific gravity, while blood, albumen, and casts disappear, many of the latter assuming the *hyaline* character during the progress towards convalescence. Some fatty changes may be noticed, but they disappear in a favourable case. As a rule the dropsy subsides before the albuminuria, and the latter may hold on for a considerable time. Not uncommonly acute Bright's disease passes into a *chronic* form. *Death* may result from dropsy affecting important parts, such as the glottis; from inflammatory complications; or from uræmia.

DIAGNOSIS.—The circumstances under which it occurs; the peculiar train of symptoms; and the characters presented by the urine, in most cases render the diagnosis of acute Bright's disease quite easy. When the affection sets in insidiously, as with uræmic symptoms, there may be much obscurity at first. It must be remembered that *acute* Bright's disease may occur as an exacerbation of the *chronic* form, and an important matter bearing upon diagnosis is to determine whether such is the case, or whether the complaint is actually recent. This conclusion is founded upon the past history; the presence or absence of any sufficient and obvious cause of the acute attack; and the characters of the urine. Should much blood and renal epithelium be discharged, and should the microscopic elements in the urine not show signs of degenerative changes, the disease is probably recent and entirely acute.

PROGNOSIS.—Any organic disease of the kidneys is serious, especially if it is extensive, and if both organs are involved. Hence acute Bright's disease is a grave affection. However, a large number of patients affected with this complaint recover completely; but there is always a danger lest it should lapse into the chronic state, and therefore it is necessary to watch the urine carefully for some time before giving a final prognosis. If, along with the subsidence of the symptoms, the albumen and other abnormal urinary ingredients steadily diminish, and the urine is gradually restored to its normal characters and composition, the prognosis is favourable. Even should slight albuminuria hold on for some time, accompanied with a few casts, the case may end in ultimate recovery. If albumen continues to be discharged in abundance for a length of time, the prognosis becomes more serious; much will depend also on the presence and characters of the casts, whether these show that the disease is subsiding, or that it is becoming confirmed, and that the epithelium is undergoing degenerative changes and destruction. Recovery cannot be considered satisfactory until every trace of albumen



has permanently disappeared. The immediate prognosis is more grave if the urine becomes very scanty; and if it contains a large quantity of albumen, blood, and casts. The chief signs of proximate danger are the supervention of uræmic symptoms; œdema of the glottis or lungs; abundant pleuritic or pericardial effusion; severe erysipelas affecting dropsical parts; and the development of acute inflammatory complications.

TREATMENT.—A patient suffering from acute Bright's disease should be kept completely at rest in bed, in a warm and comfortable room, being well protected from draughts. In some cases it might be advisable at the outset to cup over the loins to the extent of from 6 to 12 ounces; but the removal of blood requires particular caution in this affection, on account of the tendency to anæmia, and it should be omitted if the patient is at all weak, and especially if chronic renal disease has previously existed. Free dry-cupping is often of great service, and may be resorted to when blood cannot be taken away. The diet must at first be kept low, especially as regards nitrogenous food, abundance of diluent drinks being allowed.

The most important object in the treatment of acute Bright's disease is to endeavour to get the skin to act freely and persistently. This is best effected by clothing the patient in flannel; placing him between blankets; and employing warm, hot air, or vapour baths, repeated daily or less frequently, as circumstances indicate. Dr. William Roberts recommends the warm "blanket-bath." Internally full doses of citrate or acetate of potash or of liquor ammoniæ acetatis may be given, freely diluted, with a few minims of tincture of henbane. Some authorities highly recommend small doses of tartar emetic or antimonial wine. Jaborandi and subcutaneous injection of pilocarpin have also been employed with advantage. There is much difference of opinion as to the use of *diuretics*. Experience has proved, however, that certain of these agents may often be given with great benefit. In the first place, the patient should drink water freely, for the purpose of eliminating and washing away the urinary solids and other materials which accumulate in the kidneys. All *stimulants* must be forbidden in the acute stage. In addition to the vegetable salts of potash already mentioned, cream of tartar, digitalis, citrate of caffeine, and infusion of fresh broom-tops have been beneficially employed in combating the dropsy of acute Bright's disease. The bowels should be acted upon freely by means of a dose of compound jalap powder every morning or on alternate mornings. Later on elaterium or other powerful *hydragogue purgatives* may be required, if the dropsy does not subside.

Various *symptoms* frequently call for attention during the course of acute Bright's disease, especially vomiting and uræmic phenomena. The management of inflammatory complications, particularly those within the chest, is often a matter of much difficulty. Lowering treatment is decidedly not admissible, and on no account must mercury be given, as in renal diseases the smallest dose is liable to produce most serious salivation. The application of blisters or of turpentine likewise requires great care, as they tend to irritate the kidneys. Opium must also be avoided, or only given very cautiously. Sinapisms, warm fomentations or poultices, and chloroform epithems over the loins are the best local applications, and they are frequently of much service.

After the more acute symptoms have subsided, the use of the *diaphoretic*, *diuretic*, and *purgative* remedies must be moderated, and at this

time the most valuable medicine is iron. Care is needed in commencing the administration of this drug, which should be given at first in a mild form and in small doses, its effects being carefully watched. The tincture of the sesquichloride, syrup of phosphate, ammonio-citrate, or ferrum redactum are the best preparations, and if the first is tolerated in full doses, excellent results are frequently brought about. Quinine may be combined with the iron, and this remedy is particularly recommended after scarlatina. The diet should be gradually improved, being also nutritious and digestible; and during convalescence a little wine may be given, provided it agrees with the patient. The greatest care is necessary at this time to guard against a relapse. The patient should always wear flannel all over the body, and avoid every possible exposure; indeed, it is often advisable to enforce confinement to the bedroom until the albumen has quite disappeared, and for some time special precautions are needed. Afterwards a change of air to a warm and well-protected region is very beneficial; or this may be recommended if the disease shows a tendency to become chronic. Baths should be employed from time to time.

## II. CHRONIC BRIGHT'S DISEASE.

There are certain well-marked varieties of this complaint recognized by most authors. The subject will, perhaps, be presented most clearly by first discussing the *general ætiology* and *clinical history* of the disease; and afterwards considering the main facts pertaining to each special form.

**GENERAL ÆTIOLOGY.**—The chief causes of chronic Bright's disease are:—1. A previous acute attack. 2. Constant or frequent exposure to cold or wet, or to sudden changes of temperature. 3. Abuse of alcohol, particularly of ardent spirits. 4. Some constitutional diathesis or form of blood-poisoning, especially gout, syphilis, tubercular or scrofulous disease, chronic saturnism, and the fatty diathesis. The opinion is held by some pathologists that all forms of Bright's disease are of constitutional origin, the renal affection being but a local development of a general disorder. 5. Chronic disease of the pelvis of the kidney, bladder, urethra, or prostate gland. 6. Pregnancy. 7. Prolonged disordered digestion. Dr. George Johnson regards this as a cause of Bright's disease, and this opinion is supported by the experiments of Stokvis, who found that if egg-albumen is made to pass through the kidneys for a length of time, glomerulo-nephritis is induced.

**Predisposing causes.**—Chronic Bright's disease is more prevalent among males, probably from their more frequent exposure to its exciting causes; in adults; in those whose occupation involves exposure to cold and wet, or to sudden changes (as cabmen, labourers, puddlers, workers in glass), or greater temptations to intemperance; and among the poor. Want of cleanliness of the skin is a predisposing cause, and this is often combined with exposure and intemperance, the three together being peculiarly prone to originate Bright's disease.

**GENERAL CLINICAL HISTORY.**—In general terms the symptoms of chronic Bright's disease comprehend morbid conditions of the urine, especially albuminuria, the presence of casts and renal epithelium or sometimes of blood, and diminution in the excretion of urea and other urinary ingredients; frequent micturition, particularly by night;

dropsical accumulations, liable to come and go, or to alter their seat rapidly; deficient action of the skin, which is almost always dry, and often rough and harsh; and changes in the blood, which becomes hydræmic and deficient in albumen and red corpuscles, with consequent pallor or sallowness of the skin, shortness of breath, and other symptoms, while excretory elements accumulate in it. Sometimes uneasiness or tenderness is experienced over the region of the kidney. Headache and giddiness are frequently complained of; and serious uræmic symptoms are liable to arise at any moment. Serous inflammations, endocarditis, bronchitis, and pneumonia are also apt to supervene. Derangements of the digestive organs are very common, in the way of loss of appetite, dyspeptic symptoms, nausea or vomiting, flatulence, and irregularities of the bowels. Other *complications* liable to be met with are phthisis; cardiac disorder or disease; morbid conditions of the vessels; and hepatic affections. In certain forms of Bright's disease apoplexy is of frequent occurrence. The signs in connection with the heart and vessels described under acute Bright's disease are also noticed in many cases of the different chronic forms; which likewise tend to originate hypertrophy of the left ventricle.

The late Dr. Sparks and Dr. Mitchell Bruce have arrived at the following practical conclusions from a number of careful observations of the effects of different kinds of diet, rest, and exercise upon the amount of albumen passed in chronic Bright's disease:—1st. That the albumen was reduced by an absolute milk diet, and by an absolutely non-nitrogenous diet; and that the effect of a non-nitrogenous diet was neither immediately produced, nor immediately arrested by the re-ingestion of nitrogen. 2ndly. That eggs in excess did not appreciably reduce the amount of albumen. 3rdly. That alcohol in the form of wine decidedly increased the albumen; and that digitalis had probably a similar effect. 4thly. That the albumen was remarkably reduced in amount by absolute rest, in the recumbent posture; and decidedly increased by exercise.

As regards the *mode of onset*, chronic Bright's disease not uncommonly remains after an acute attack; in most cases, however, it sets in gradually, and the affection may be quite latent until some grave uræmic or other symptoms reveal the serious condition present. In other instances there may only be albuminuria or slight dropsy. The disease is generally subject to remissions and subacute or acute exacerbations, the latter often coming on from a slight, or even without any obvious cause. The *duration* is very variable, some cases lasting for many years, and it differs in the several forms. Death is usually hastened at the close by uræmia; serous inflammations; pneumonia or bronchitis; dropsy, either on account of its dangerous situation, or from its being complicated with erysipelas or gangrene; or by apoplexy. A few cases terminate gradually by asthenia; in others death results from independent complications, such as phthisis. Recovery may ensue even after the disease has existed for a considerable time.

It is requisite to draw special attention to certain changes which may be observed in connection with the eye. The occurrence of temporary attacks of disturbance of vision associated with uræmia has already been alluded to. Dr. Gowers has noticed on ophthalmoscopic examination that the arteries of the retina are sometimes distinctly lessened in size; and considers that this depends on their contraction, and is as a rule in direct proportion to the tension of the arterial blood, as measured by the



incompressibility of the radial pulse. The most important condition, however, which is met with in cases of chronic Bright's disease is a form of amaurosis, attended with definite morbid changes in the retina, as observed with the ophthalmoscope, namely, *albuminuric retinitis* and *hæmorrhages*. The loss of sight under these circumstances creeps on slowly, and is permanent, though usually liable to sudden increase from various causes, with subsequent improvement. At first the appearances noticed are increased vascularity, with enlargement and tortuosity of the retinal veins, but shrinking of the arteries; and slight swelling around the disc, the margin of which becomes indistinct, while it becomes suffused and dark-red, a grey filmy exudation afterwards forming. The more characteristic appearances, however, are noticed later on, being due to the presence of numerous whitish or yellowish-white brilliant-looking spots or patches around the disc; with hæmorrhagic extravasations into the retina in the same locality. By the increase and union of the patches the disc is often surrounded with a zone, "the inner line of which is irregularly circular, or melts into the grey interval and the disk itself, while the outer presents salient angles, which correspond to the course of the larger vessels" (Allbutt). Whitish streaks are also seen radiating outwards along the vessels and nerve-fibres. In course of time the disc is itself invaded by the spots and hæmorrhages. Some observers affirm that the white spots always result from changes in blood-clots, but probably most of them are the result of independent exudation. Ultimately these may be absorbed, many of the vessels being obliterated or removed, causing retinal anæmia: while it is then seen that the choroid has also undergone marked changes, and that it presents yellow patches. Important structural changes are set up in the retina, choroid, and vitreous body. Both eyes are always involved, but not to the same degree.

These ocular changes are unquestionably found most frequently and distinctly in connection with the *granular contracted kidney*, but they have also been noticed in other forms. As to their immediate cause, they have been attributed to hypertrophy of the heart accompanying the renal disease; to some constitutional condition attended with changes in the vessels generally; to alterations in the blood, namely, uræmia or deficiency of albumen; or to extension of disease along the optic nerve from the brain.

Having given this general outline of Bright's disease, the prominent characters of the several varieties will now be pointed out.

# 1. LARGE, WHITE, SMOOTH KIDNEY—CHRONIC DESQUAMATIVE OR TUBAL NEPHRITIS.

**ÆTIOLOGY.**—This form is most frequently a sequel of acute or sub-acute Bright's disease. It may come on gradually from taking cold; as the result of repeated pregnancies; or in the course of phthisis. It is chiefly met with in comparatively young persons.

**ANATOMICAL CHARACTERS.**—The kidneys are enlarged and heavy, their surface being smooth and pale, but variegated with vessels; the capsule is readily separated, being unaltered or somewhat opaque. A section shows great thickening of the cortical substance, which is white or yellowish-white and opaque, often also presenting numerous small yellow spots or streaks, due to fatty degeneration—"granular fatty kidney"?

(Johnson). The consistence is diminished. The pyramids retain their normal colour, and contrast markedly with the cortex, though they are also affected to a less degree. Microscopic examination reveals enlargement of many of the tubes, which contain a great number of epithelium-cells, with exudation. The cells are always much altered, being swollen, clouded, and more or less granular, also frequently containing fat or oil-globules, or they may be quite disintegrated, so that only a granular *débris* remains, with masses of fat and oil-globules. Some tubules may be quite denuded and empty, or only present hyaline fibrinous moulds. The Malpighian corpuscles are either normal in size or a little enlarged, and their capsules are thin.

It is now generally conceded that in very chronic cases this form of kidney occasionally gradually contracts and wastes, and it may become markedly small and irregularly atrophied. How this change is brought about is, however, not positively determined. Some authorities maintain that it results from an interstitial inflammation, as in the cirrhotic kidney; others that it is entirely due to changes within the tubules. In this condition of kidney the capsule is more or less thickened, opaque, and adherent at parts; superficial depressions form, causing a somewhat granular appearance; while there is some increase in the interstitial tissue, with thickening of the blood-vessels. These changes are, however, not so marked as in the cirrhotic kidney, and they are mainly noticed in those regions where the tubular changes are most evident, and where blocked tubules are seen in all stages of destruction.

**PATHOLOGY.**—The generally accepted view of the pathology of the large white kidney is, that it is the result of *tubular nephritis*, beginning as an acute or chronic affection, attended with great increase and desquamation of the epithelium, the cells of which gradually undergo changes, ending in their complete fatty transformation and destruction. More or less loss of tissue, with consequent atrophy of the kidney, may follow in course of time, as above described.

**SYMPTOMS.**—Either remaining after an acute attack, as frequently happens, or being chronic from the outset, this variety of Bright's disease presents the following clinical features:—The urine is usually deficient in quantity; pale and often somewhat turbid, depositing a whitish sediment, or from time to time being smoky or tinged with blood; of normal or rather high specific gravity; while it contains a considerable quantity of albumen, as well as various casts, with renal epithelium or its remains. The microscopic elements are not nearly so abundant as in the acute disease. The chief casts met with are *epithelial*, the epithelium-cells being always more or less altered; *granular*; *large* or *small hyaline*; and *fatty*, as the renal structures undergo fatty changes. By studying their prevailing characters much information may be gained as to the exact state of the kidneys. Anasarca is generally a prominent symptom, and serous effusions are not uncommon. The general surface, but more especially the face, presents the characteristic dull-white, puffy, and pasty aspect, being often also smooth and glossy, these appearances becoming more marked as fatty degeneration proceeds. There is considerable tendency to uræmia, and to the occurrence of serous inflammations. Dr. George Johnson states that mucous hæmorrhages are frequent in the advanced stages, especially epistaxis. Exacerbations are very liable to arise from time to time.

## 2. GRANULAR, CONTRACTED, OR CIRRHOTIC KIDNEY— CHRONIC INTERSTITIAL NEPHRITIS.

**ÆTIOLOGY.**—In this variety the onset is always very chronic and insidious, being independent of any obvious immediate *exciting cause*. The contracted kidney is chiefly associated with gout; chronic lead-poisoning; chronic alcoholism; a tendency to general degenerative changes; or, it is said, with repeated exposure to cold. The subjects of this form of renal disease are usually advanced in years. It must be remembered that the large white kidney may become contracted in course of time.

**ANATOMICAL CHARACTERS.**—Chronic interstitial nephritis is characterized by a gradual increase of the intertubular connective tissue of the kidneys, and by wasting of the tubular structures. At the same time there is no absolute line of demarcation between *inter-* and *intra-*tubular changes. The prominent changes presented by the cirrhotic kidney are gradual contraction and atrophy, until the organ may weigh only an ounce or two; granulation of the surface, the granules ranging from the size of a pin's head to that of a pea or more, there being also irregular depressions, giving rise to a lobular appearance; thickening, opacity, and adhesion of the capsule, which is inseparable and sinks into the depressions; increased resistance and toughness of the renal tissue. These changes are observed in very different degrees of advancement in different cases. On making a section of the kidney it is seen that the cortical substance is chiefly wasted, having in some instances almost completely disappeared, what remains being of a red or brownish-red colour, and coarsely granular. There may be signs of fatty degeneration. According to the colour, the granular kidney has been described as "red," "white," "yellow," and "mixed." Cysts are frequently observed, varying in size from very minute points to spaces as large as a nut, or even larger, and containing an albuminous fluid. In the gouty kidney a white deposit of urates forms in connection with the tubules. Calcareous deposits are also sometimes visible as white streaks between the tubes of the pyramids.

The intimate changes usually consist in a great increase of the intertubular connective tissue; with consequent alterations in the tubules, Malpighian corpuscles, and vessels. Many of the tubules are denuded of their epithelium, contracted, or obliterated; others are blocked up with disintegrated epithelium-cells; while still others contain clear fibrinous moulds. Fat-granules and oil-globules are often visible. The Malpighian bodies appear shrunk and abnormally close together, their enclosed glomeruli being more or less compressed by fibrous tissue. Many vessels are obliterated, and the walls of the smaller arteries are thickened, so that it is difficult to inject the kidney through its main artery. This thickening of the arteries has been attributed to muscular hypertrophy, and to increase in the external fibrous coat. The cysts so frequently seen are probably due to obstruction or constriction of the ducts at intervals, with distension of the intervening portions. The late Dr. Mahomed held that in the *red* granular kidney the changes are chiefly vascular, namely, thickened vessels, thickened Malpighian corpuscles, and fibro-hyaline intertubular thickenings; and that the *yellow* or *mixed*



granular kidneys have, in addition to these, interstitial small-celled growths and epithelial proliferation.

**PATHOLOGY.**—It is in connection with the *cirrhotic kidney* that important differences of opinion have arisen regarding pathological questions. With respect to its *nature and mode of origin*, most German authorities consider it as being merely a later stage of the large white kidney, which, if it only lasts long enough, will become atrophied and granular; and some of their pathologists describe several stages in the progress of the morbid alterations in the kidneys. Though recognizing the fact that now and then the large kidney does waste and become granular, as has been already pointed out, yet most English writers are of opinion that the true cirrhotic kidney does not so originate, but that it is the result of *chronic interstitial nephritis*, attended with proliferation of the intertubular connective tissue, which becomes much increased, and compresses surrounding structures. Dr. Grainger Stewart, who formerly regarded this disease as a result of excessive growth of the renal connective tissue, now agrees with this view. Dr. George Johnson, however, considers that the *epithelial cells* are first affected, undergoing degeneration in consequence of having to perform unusual excretory work. Dr. Dickinson believes that the disease begins superficially, immediately under the capsule, and gradually extends inwards.

Before alluding to an important view held in relation to the cirrhotic kidney, it will be convenient to consider more particularly the morbid conditions of the heart and arteries which may be induced by Bright's disease, and which are found with special frequency associated with the contracted kidney. It must be borne in mind that the heart may be primarily diseased, and by inducing mechanical congestion may lead to organic changes in the kidneys, ending in contraction and atrophy, this condition being, as previously stated, regarded by some pathologists as a variety of Bright's disease. Again, affections of the cardiac valves may co-exist with renal disease independently, or as the result of the same constitutional dyscrasia; or they may be the consequence of endocarditis complicating Bright's disease. It is, however, to cardiac hypertrophy, especially involving the left ventricle, and presumed to be a secondary result of the renal disease, that it becomes requisite to draw particular attention. There can be no doubt but that this condition of the heart does arise in many cases of chronic Bright's disease, and the question is, how is it originated? Formerly it was attributed to the altered state of the blood, which was supposed to exercise an undue stimulant effect upon the heart, or to pass with difficulty through the capillaries, thus causing the heart to act with excessive vigour, and so to become hypertrophied. Traube advanced the opinion that destruction of the renal secreting tissue, and obstruction to the circulation in the kidneys lead to the hypertrophy, chiefly because under such circumstances a sufficient amount of water is not removed from the blood, and thus the arterial tension is increased; this view, however, he seems to have subsequently relinquished. Dr. George Johnson made the important discovery that the walls of the small arteries, not only in the kidneys, but also in various other structures throughout the body, become greatly thickened. This eminent observer maintains that the arteries contract, and oppose the passage into the tissues of the unhealthy blood associated with Bright's disease, with consequent rise in arterial tension; and that this is followed by hypertrophy of the muscular coat, which is the cause of the thickening of their walls. Dr.

Gowers thinks that his ophthalmoscopic observations as to the contraction of the retinal vessels afford a direct proof in favour of the first part of this theory. Owing to the condition of the arteries it is supposed that the walls of the heart become also hypertrophied, in order to overcome the resistance thus offered.

Sir William Gull and Dr. Sutton have denied that the thickening of the small arteries is due to muscular hypertrophy; they affirm from their observations that these vessels, as well as the capillaries throughout the body, become the seat of a peculiar *hyalin-fibroid* change, which leads to thickening of their walls with loss of elasticity, and they attribute the cardiac hypertrophy to this *arterio-capillary fibrosis*, as it is termed. On these observations they found another view as to the pathology of the granular kidney, namely, that it is but a part of a general morbid condition, beginning in the smaller vessels and affecting these throughout the body, which leads to atrophy of the tissues. In their opinion the vascular changes outside the kidneys are not the secondary result of the renal disease, but merely a part of the general disorder.

Dr. Mahomed maintained that high arterial pressure occurring in young and apparently healthy persons, independent of renal disease, will, if it remains as a chronic condition, produce the cardio-vascular changes of Bright's disease, and that the *red granular kidney* is thus produced, this being unattended with albuminuria. In a further stage the *yellow or mixed granular kidney* is produced, with albuminuria.

**SYMPTOMS.**—The contracted kidney may be clinically latent for a very long period. As regards the urine, this is as a rule abundant, being in some cases very copious; of light colour and low specific gravity; while it contains comparatively little albumen, or even none at all at times; there being also but few or no casts, which are chiefly *hyaline* or *granular*, with but little epithelium or fat. Dr. Mahomed affirmed that there is no albuminuria in the red granular kidney. At the close the urine often becomes very scanty or suppressed. Dropsy is absent in a considerable number of cases from first to last, and generally is but slight, or is only observed at intervals. The skin is dry and harsh, but does not exhibit the peculiar pale and pasty aspect; while the face has often a sallow and pinched appearance. In most cases there is marked debility and constitutional cachexia. Dyspeptic disturbances are frequently very prominent. The *complications* most liable to be met with are cardiac hypertrophy, and morbid conditions of the vessels, the latter not uncommonly leading to apoplexy. Uræmia often supervenes; but some authorities affirm that serous or pulmonary inflammations are much less frequent than in connection with the large white kidney.

### 3. FATTY KIDNEY.

Fatty changes are observed in connection with all forms of Bright's disease, but some authorities are of opinion that the kidney may become primarily the seat of a *fatty infiltration*, the renal cells being loaded with fat, the liver being in most cases affected at the same time. Dr. Johnson applies the terms *simple fat kidney* or *general fatty infiltration of the kidney* to this condition. It is stated that it may be associated with any of the usual causes of fatty infiltration. The kidneys are frequently enlarged, their cortical substance being uniformly pale or mottled with red, and occasionally hæmorrhagic spots are observed. The consistence



is diminished, the kidney having often an cedematous feel and appearance. The microscopic shows uniform distension of the renal cells with oil. There may be albuminuria and other symptoms of renal disease, but generally no obvious signs of functional derangement of the kidneys are observed (Johnson).

#### 4. LARDACEOUS OR ALBUMINOID KIDNEY.

Most writers now classify the *lardaceous kidney* as a form of Bright's disease. Its *etiology* and *pathology* are those of the general disease.

ANATOMICAL CHARACTERS.—There is by no means an agreement in the descriptions given by different observers as to the appearances presented by the albuminoid kidney. Dr. Grainger Stewart recognizes three stages of the disease, in the first stage the vessels only being affected; in the second the renal tissues being infiltrated with the albuminoid material, and many of the tubules blocked with it; and in the third the organ being atrophied and contracted. Dr. Dickinson describes a somewhat analogous course of morbid changes. When the disease is marked the kidneys are enlarged and pale, and their surface is smooth, the capsule separating readily. The consistence is very tough and hard. A section is sharp-cut, and shows the cortical substance to be pale, anæmic, waxy, and translucent, often dotted over with glistening spots, which correspond to the infiltrated Malpighian bodies, in which the deposit first occurs. The cones seem natural. The usual chemical reactions characteristic of albuminoid material are yielded. The renal cells are often cloudy, withered, or fatty, but they are stated not to afford the tests of albuminoid deposit. Transparent hyaline moulds are found in some of the tubules. In the advanced stage the affected organs may become much atrophied and irregular. In a case observed by Dr. Stewart the kidneys were found after death to weigh two and a half ounces each; the tubes were wasted, the Malpighian bodies were large and waxy, and closely grouped together, the fibrous tissue was relatively more abundant than in the healthy organ, but the capsules were at all points readily separable, and the organs did not present the appearances characteristic of cirrhosis. In opposition to this view, German writers deny that this marked contraction can take place in cases of simple albuminoid disease of the kidney, but that it is due to a simultaneous or previously existing cirrhotic process.

SYMPTOMS.—There is also much difference of opinion as to the clinical signs of albuminoid disease implicating the kidneys, and some writers maintain that they are very variable in different cases. Dr. Stewart affirms that when the disease is simple and uncomplicated, he has invariably found the symptoms distinct and uniform throughout. They seem to be of the following character. There is polyuria, the urine being very copious, pale and watery, depositing scarcely any sediment, and of low specific gravity, varying from 1005 to 1012 or 1015. At first albumen is either absent altogether, or is present only in very small quantity; later on it increases, and often becomes exceedingly abundant. Cases, however, have been described in which there was no albuminuria throughout, and Dr. Stewart states that this happens in rare instances. The urine presents little or no sediment, and very few casts are present, consisting mainly of the *small hyaline* and *finely granular* varieties; there may be some epithelium scales upon them, or these may be



separate, being usually wasted, or containing oil-globules. The epithelial particles occasionally yield the reaction of albuminoid substance. Dropsy is absent, or almost completely absent, throughout. Cardiac changes are also wanting or comparatively slight; and uræmic phenomena are rare. Owing, however, to the frequent association of albuminoid disease with other forms of Bright's disease, the symptoms are liable to be much modified. If chronic inflammatory changes set in, the urine becomes much diminished in quantity, and its specific gravity raised; while there may be much sediment, with numerous *large hyaline* and *granular casts*. Dropsy also supervenes, and may be very considerable, this symptom likewise occurring in cases in which albuminoid disease is added to previous inflammatory disease. There will usually be evidences of this condition affecting other organs; and its general symptoms will also be present.

### 5. MIXED TYPES.

It need only be remarked here that kidneys are sometimes met with presenting combinations of the morbid changes just described. Thus, there may be a combination of the *interstitial* and *tubal* forms of disease; or of *lardaceous disease* with either of these forms. As already remarked, *fatty* changes are common in all forms of Bright's disease.

### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. DIAGNOSIS.—The existence of one or other form of chronic Bright's disease is often quite evident from the *history* of the case; and from the *symptoms* present, the characters of the *urine* being especially important. Albuminuria may be the only sign of renal mischief, and therefore it is most desirable to adopt a routine practice of examining the urine, especially if an individual is persistently out of health without any obvious cause, or suffers habitually from dyspeptic symptoms, from headache or giddiness, or from cardiac disturbance. Of course it must be remembered that albuminuria may be due to other causes besides renal disease, and especially to cardiac disease obstructing the venous circulation, or to admixture of albuminous fluids; and also that this symptom may be absent in serious forms of kidney mischief. Careful microscopic examination is necessary, in order to discover casts or renal epithelium, if these elements are present. The ophthalmoscope is highly important in diagnosis in some cases, and its employment has not unfrequently been the means of revealing Bright's disease where it was previously unsuspected.

The diagnosis between the different *varieties* of chronic Bright's disease is founded upon the conditions under which they severally arise; and upon their special symptoms, which have already been pointed out. The stage of degeneration of the kidneys, and the actual changes which are going on, may often be determined with much accuracy by a careful observation of the microscopic elements. The development of an acute inflammatory affection or of uræmic symptoms in the course of chronic Bright's disease not previously known to exist, may prove very puzzling. The rule of always examining the urine will generally lead to the detection of the renal affection under such circumstances.

2. PROGNOSIS.—The prognosis in chronic Bright's disease, while always more or less unfavourable, differs much in different cases. The termination is most rapid in connection with the large white kidney, but much will depend on the exact nature and extent of the changes in the kidneys, as revealed by the urine. Patients suffering from this affection may live for many years, and may even enjoy comparatively good health. The circumstances which render the prognosis more unfavourable are a prolonged duration of the disease; steady diminution in the quantity of the urine, without corresponding increase in density; excessive albuminuria, with abundant granular and fatty casts or oil-globules; extensive dropsy, with serous effusions; obstinate dryness of the skin; marked cardiac hypertrophy and vascular changes; persistent dyspepsia or disturbances of the bowels; and constant pyrexia. It is remarkable, however, how much improvement in symptoms may be brought about in some apparently hopeless cases. There is always a danger at any moment of the supervention of uræmia; of acute exacerbations of the kidney disease; or of inflammatory complications, the last being very easily set up, and being much more grave than in healthy persons. Patients suffering from Bright's disease are extremely unfavourable subjects for injuries or operations.

3. TREATMENT.—Chronic Bright's disease requires very careful and varied management, and it is difficult to indicate any definite line of treatment which shall be applicable to all its forms. At the same time there are certain principles to be followed out, to which attention will now be directed.

*a.* It is very important to find out the *cause* of the disease, and to remove this if possible, such as abuse of alcohol, constant exposure, or suppuration setting up albuminoid disease. *b.* *Hygienic and dietetic* management demands careful and constant attention. In those cases in which there is merely albuminuria this is often all that is needed. The patient must be completely clad in flannel, and avoid exposure, especially to cold or wet weather, as well as to anything likely to cause a chill, and should take moderate exercise daily. If possible, a residence in a tolerably warm, equable, and sheltered district is advisable, or a temporary change to such a district should be recommended. A sea-voyage is sometimes highly beneficial in cases not far advanced. It is very necessary to keep up a free action of the skin, by means of warm baths with friction, or it may even be advisable to employ occasionally a hot-air or Turkish bath. The diet requires supervision, and should be of a nutritious and digestible kind, though it is necessary to restrict more or less the consumption of nitrogenous elements of food. Milk may be usually taken in large quantities; and skimmed-milk has been specially recommended in the treatment of Bright's disease. I am acquainted with one case in which the patient has lived absolutely on skimmed-milk for several months, and has derived much benefit therefrom. Strong stimulants had better be avoided, but light wines in moderation, or a glass of good bitter ale, do good in many cases. The bowels must be kept well opened daily, and the digestive functions maintained in good order. *c.* Treatment directed to the *constitutional state*, and the *condition of the blood*, is of the highest value. The administration of iron, regularly and perseveringly carried out, often produces the most beneficial results, in the way of improving the state of the blood and general system. If it can be taken, the tincture of steel or the solution of perntrate is the best preparation, but the saccharated carbonate,

ferrum redactum, syrup of iodide or phosphate, ammonio-citrate, or citrate of iron and quinine, are also very useful. Among other conditions affecting the constitution which require special attention in chronic Bright's disease are phthisis; albuminoid disease; gout; and saturnism. *d.* Some authorities consider it desirable to diminish the amount of *albumen* discharged in cases of Bright's disease, by means of the agents previously mentioned. *e.* Dropsy is the principal *symptom* calling for interference in a large proportion of cases of Bright's disease. *Purgatives* and *baths* are chiefly to be relied upon for its removal. Among the former jalap, cream of tartar, elaterium, scammony, gamboge, and podophyllin are the most serviceable. Jaborandi or subcutaneous injection of pilocarpine may also be useful. Apocynum cannabinum or American Indian hemp has been well spoken of. Some practitioners give liquor ammoniæ acetatis freely with iron, to act as a *diaphoretic*; others recommend James's or Dover's powder. Opinions are much divided with regard to *diuretics*, both as to the propriety of giving them, and as to those which are most efficacious. In my experience certainly they are not of much use as a rule, and may do considerable harm. Copaiba or its resin, and caffen have been used with advantage in some cases. Dr. Leech has found the best results from *saline diuretics*, especially tartrate, bitartrate, and acetate of potash. From iodide of potassium he has obtained no definite effects. Digitalis and caffen he found much less useful than in cardiac dropsy. The inhalation of oil of juniper appeared to give better results than its administration by the mouth. In extreme dropsy acupuncture, the use of Southey's trochars, or free incisions into the skin of the legs or scrotum may be required. Great care is necessary when carrying out these measures, in order to guard against erysipelas, which may be prevented by applying warm moist flannels, frequently changed and thoroughly cleansed before being re-applied; and by sponging the parts well before each application is made. Antiseptics may be employed with advantage. Particular care is needed to avoid pressure upon dropsical parts, or their irritation by the contact of urine. Among other symptoms which are likely to require treatment are those indicative of dyspepsia; vomiting; derangement of the bowels; headache or vertigo; and uræmic phenomena. The remarks made under acute Bright's disease with reference to inflammatory complications apply likewise to the chronic forms of the disease. *f.* It is necessary to warn patients suffering from Bright's disease against the dangers to which they are exposed; and to impress upon them the necessity of paying strict attention to the hygienic matters already mentioned. Should cardiac hypertrophy be set up, with changes in the vessels, the danger of the occurrence of cerebral hæmorrhage should be borne in mind.

---



## CHAPTER LXII.

## ON CERTAIN RARE DISEASES OF THE KIDNEYS.

## I. CANCEROUS AND OTHER GROWTHS.

OF rare occurrence, renal cancer may be *primary* or *secondary*. It is most frequent in very young children, or after adult age; and in males. The variety met with is almost invariably *encephaloid*. The deposit is always in the nodular form when secondary; but when primary it may be either nodular or infiltrated. Secondary cancer does not attain any large size; but primary cancer frequently grows to enormous dimensions, usually giving rise to an irregular tumour. The consistence varies considerably, being sometimes very soft and almost fluctuating, while it is rarely uniform throughout. Hæmorrhage, softening, degeneration, and suppuration are liable to occur in the growth. The uninvolved renal texture is generally atrophied from pressure, or otherwise altered. Thickening of the capsule, and adhesions to neighbouring parts are generally observed; while adjoining structures are frequently displaced or destroyed by pressure, the colon always lying in front of the tumour. The pelvis of the kidney and the ureter are often involved. In the great majority of cases only one kidney is affected. Secondary deposits are common, especially in the neighbouring glands.

**SYMPTOMS.**—The important clinical signs of renal cancer are severe pain in the lumbar region, generally shooting towards the hypochondrium and thigh or in some other direction, and subject to remissions or intermissions; tenderness; hæmaturia, in many cases profuse and irregularly intermittent, occurring without any obvious cause; and the presence of a *renal tumour*, the special characters of which are the rapidity of its growth, and the great size which it may attain, especially in children; its absolute immobility; as a rule its irregularly lobular feel; and its more or less firm though unequal consistence. Occasionally there is an obscure sense of fluctuation over some parts of it. In some cases the superficial veins over the tumour are much enlarged; and it has been known to present pulsation. Symptoms may arise from the pressure of the growth on surrounding structures. The detection of cancer-cells in the urine has been considered important, but several excellent observers doubt the possibility of recognizing these structures. Marked emaciation and debility, with signs of the cancerous cachexia, are frequently observed; and there may be evidences of cancer in other parts of the body. The *course* of the disease is very rapid in children, but comparatively chronic in adults. There may be no symptoms in secondary cases.

Of the *non-malignant* growths very exceptionally found in the kidneys may be mentioned osseous tumours; fibromata; sarcomata; lipomata; enchondromata; lymphatic or glandular growths; and syphilitic gummata. Some of these may form an evident tumour.

## II. TUBERCLE.

There are three classes of cases in which tubercle is found in connection with the renal apparatus, namely:—1. As a part of *acute miliary tuberculosis*, the kidneys being studded with grey granulations. 2. *Secondary* to tubercular disease in the lungs or other organs, when it does not usually give rise to any local symptoms. 3. As a *primary* formation, generally involving the kidneys, their pelves and ureters, the bladder, and sometimes the urethra; and being not uncommonly followed in the male by deposits in the prostate gland, testes, or vesiculæ seminales. The last constitutes much the most important group of cases. In the kidneys tubercle is seen at first in the form of grey or yellow nodules, occupying the cortex, which afterwards coalesce, become caseous, and break down, forming irregular abscess-like cavities, which burst into the urinary passages, discharging disintegrated tuberculous matter and pus. Generally both kidneys are implicated; and they are frequently extensively or completely destroyed. In the pelvis and ureter the growth starts in the submucous tissue, where it forms granules, and ultimately inflammation of the overlying membrane is excited, ending often in extensive ulceration and destruction. Occasionally one ureter becomes completely rigid, and its canal is blocked up, leading to pyonephrosis.

**SYMPTOMS.**—During the early period *primary renal tuberculosis* may be indicated by a dull pain in the region of the kidneys, with frequent micturition. The important symptoms, however, are those of chronic pyelitis or pyonephrosis, often associated with symptoms of cystitis; great wasting, debility, and hectic fever; and in time signs of implication of the lungs, intestines, or other organs. The urine is almost always deficient; slightly acid; and contains abundance of pus, frequently a little blood, but not in any large quantity, extra-renal epithelium cells, much granular detritus, and in some cases connective-tissue or elastic fibres. If the ureter is blocked up, a painful fluctuating tumour forms in the corresponding renal region, which may subside with coincident appearance of much pus in the urine, should the obstruction be removed. Uræmia is liable to arise if both kidneys are affected.

## III. PARASITIC GROWTHS.

1. Occasionally one kidney, especially the left, is the seat of a *hydatid tumour*, which may ultimately attain a great size. It tends to burst into the renal passages, its contents escaping with the urine; very rarely it opens in some other direction; or it may undergo any of the changes to which hydatid-cysts are liable.

**SYMPTOMS.**—There may be none throughout. The most prominent sign of hydatid disease of the kidney is the existence of a renal tumour, rounded in form, though often somewhat irregular and lobulated; having an elastic or more or less fluctuating feel; and occasionally yielding hydatid-fremitus. As a rule there are no renal symptoms. Should the cyst burst into the urinary passages, important symptoms generally arise, namely, those of one, or more commonly, of several intermittent attacks of *nephritic colic*, due to the escape of the vesicles by the ureters; preceded by a sharp pain in the renal region, and

occasionally by a sense of something having burst; and followed by signs of the passage of the hydatids along the urethra, that is, by great pain to the end of the penis, and constant desire to pass urine, with more or less retention; and finally by the appearance of the vesicles or their remains in the urine, frequently accompanied by some blood or pus. Occasionally a cyst blocks up the ureter, and thus leads to hydronephrosis. Inflammatory symptoms arise should the tumour become inflamed; or various symptoms may occur from its bursting in different directions.

2. The *cysticercus cellulosus* has been found in the kidneys.

3. The following *entozoa* are met with occasionally:—*a. Bilharzia hæmatobia*. This worm is found in some other structures, but it is most injurious in connection with the bladder, ureter, and pelvis of the kidney, being deposited in the minute veins of the mucous membrane lining these parts. It belongs to the *trematoda*, being about 3 or 4 lines long, of soft texture, and bisexual. The morbid effects which it may occasion are hæmaturia, it being, as previously stated, regarded as the cause of the *endemic hæmaturia* of certain hot countries; the formation of raised, injected, and ecchymotic patches in the mucous membrane; local inflammation ending in suppuration; obstruction of the ureters, with consequent hydronephrosis or pyonephrosis; and the formation of calculi, owing to the masses of ova forming a nucleus for the urinary deposits.—*b. Strongylus gigans*. This is a *nematoid* worm, resembling in general characters the *ascaris lumbricoides*, but being much larger, having a reddish colour, and presenting six nodular papillæ about the mouth. It is found in the kidney and urinary passages, and necessarily tends to give rise to considerable disturbance, but of no definite character. *c. Pentastoma denticulatum*. Supposed to be the larva of a worm, this appears as a very minute encysted parasite,  $1\frac{1}{2}$  lines long, club-shaped, with a double pair of hooks, and devoid of sexual organs. *d. Filaria sanguinis hominis*. This parasite has already been fully considered, in its relation to chyluria and hæmaturia.

#### IV. CYSTIC DISEASE.

Dr. William Roberts describes the following varieties of cysts which may be met with in connection with the kidney:—1. Scattered cysts in kidneys otherwise healthy, which now and then attain a great size, so as to form a fluctuating tumour. 2. Disseminated cysts in the atrophic form of Bright's disease. 3. Congenital cystic degeneration. 4. General cystic degeneration in adults. The last affects both organs, but to different degrees. They become much enlarged, and are converted into a mass of closely aggregated but distinct cysts, lodged in an abundant matrix of connective tissue; varying much in size; and containing either a limpid yellowish or reddish serum, or a gelatinous substance, this yielding albumen but not urinary ingredients; subsequently other materials are often added. The renal tissue is partially or almost completely destroyed. The cysts do not as a rule open into the pelvis, which, with the ureter and bladder, is usually quite healthy. As to the *origin* of these cysts, they have been attributed to dilatation of the Malpighian capsules; or to distension of limited portions of the tubules which have been obstructed at each end. During life this condition may give rise to a tumour, which is sometimes extremely large. The



urine is occasionally increased in quantity, and is generally of low specific gravity. The fatal termination is often preceded by uræmic symptoms.

#### V. HYDRONEPHROSIS—DROPSY OF THE KIDNEY.

Hydronephrosis may result from any permanent closure of the ureter. It is frequently congenital, but may arise subsequently from rupture of the ureter due to injury; impaction of a calculus or other body in its interior; organic changes in its walls leading to stricture, such as ulceration followed by cicatrization; or external pressure upon it by a tumour. As the result of this obstruction, the pelvis and the portion of the ureter above the impediment become dilated from accumulation of urine; this subsequently causes flattening of the papillæ, and gradual compression and atrophy of the pyramids of the kidney, followed by wasting of the cortex, with distension of the capsule, until ultimately nothing may be left but a membranous sac containing fluid, either single or divided into chambers, and sometimes attaining an enormous size. The fluid consists usually of altered urine, this being much more watery than the normal secretion, almost always a little albuminous, and sometimes presenting an admixture of blood, pus, or epithelium. As a rule only one kidney is affected, while the healthy organ becomes hypertrophied.

**SYMPTOMS.**—Evidence of some cause likely to give rise to obstruction of the ureter may help in the recognition of hydronephrosis. The only positive sign, however, is the development of a painless, soft, and more or less fluctuating renal tumour, which sometimes feels lobulated; unaccompanied with any unusual characters of the urine. Occasionally the obstruction is removed, and the tumour suddenly subsides with copious discharge of urine, which is highly characteristic; the sac may afterwards shrivel up. It may be necessary for diagnostic purposes to use an exploratory trochar or the aspirateur. The tumour may occasion symptoms by pressing on surrounding structures. It is a curious fact that in cases of double hydronephrosis uræmic symptoms do not arise for a considerable time. Most cases ultimately terminate fatally in various ways. In extremely rare instances the sac ruptures spontaneously.

#### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. **DIAGNOSIS.**—In most of the rare affections just considered, the chief matter in diagnosis is to determine the nature of a *tumour in the renal region*. It will be well, therefore, to enumerate all the conditions to which such a tumour might be due, and they include *renal abscess; pyonephrosis; perinephritis; cancer* or a *non-malignant growth; hydatid disease; hydronephrosis*; or *cystic degeneration of the kidney*. The distinctive features of these morbid conditions have been sufficiently pointed out in their several descriptions, the characters of the enlargement, as well as those of the urine, being important elements in diagnosis. It may be requisite to employ an exploratory trochar or the aspirateur before any positive conclusion can be arrived at. The tumour may become so large as to fill the abdomen, so that it becomes impossible, except by the history, to recognize its origin; and when due to an

accumulation of fluid, it may come to simulate ascites. A renal tumour may be mistaken for one in connection with the ovary, uterus, supra-renal capsule, liver, spleen, or neighbouring absorbent glands; or for an accumulation of fæces in the intestines.

2. PROGNOSIS.—The prognosis of the diseases described in this chapter is very unfavourable. Cancer is necessarily fatal. Accumulations of fluid in connection with the kidneys, especially if of a purulent character, are exceedingly dangerous, owing to their effects upon the renal structure; the constitutional disturbance which they often set up; and the dangers of the supervention of uræmia, or of the discharge of the fluid into the abdominal cavity.

3. TREATMENT.—If anything can be done for the rarer forms of kidney disease now under consideration, *operative interference* is generally called for. In *hydronephrosis* the first principle is to endeavour to remove the obstruction which causes the retention of urine, and frequent manipulation or shampooing over the renal region has sometimes been found effectual for this purpose. If this does not succeed, and there are indications of danger, tapping must be resorted to, by means of the aspirateur or a small trochar, and the operation should be repeated if necessary. *Hydatid tumour* must be treated in the same manner as hydatid disease of the liver. The removal of the kidney for *cancer* or other solid tumour is scarcely permissible, but has been performed. *Tubercular pyelitis* requires similar treatment to other forms of this disease, the constitutional condition being attended to at the same time.

---

## CHAPTER LXIII.

### URINARY CALCULUS AND GRAVEL.

THE full consideration of this subject comes more appropriately within the scope of surgical works, and here it is only intended to give a brief outline of its main facts. By *gravel* is meant very small concretions, which are often passed in the urine in large numbers.

VARIETIES OF URINARY CALCULI AND THEIR CHARACTERS.—1. *Uric Acid*.—This is very common, both in the form of calculi and gravel, being especially associated with the gouty diathesis, and hence occurring mainly in elderly persons, and among those of the upper classes. The concretions are formed in very acid, high-coloured, concentrated urine. They are hard; heavy; minutely tubercular or smooth on the surface; generally oval and compressed; as a rule small or of moderate size; and variously coloured by urinary pigments. There may be several. 2. *Urates*, chiefly consisting of urate of ammonia. These form soft, irregular concretions, which are deposited from acid urine, almost always in the kidneys, and nearly limited to young children. They are soluble in hot water. 3. *Oxalate of lime* or *Mulberry calculus*.—This variety is characterized by the surface being rough and tuberculated, like a mulberry. The calculus is of moderate size; generally spherical; very hard; and dark brown or almost black in colour. 4. *Phosphatic calculi*.—The important variety is the *fusible calculus*, consisting of a mixture of calcic and

ammonio-magnesian phosphates. It is almost always formed in the bladder, and on a nucleus of some other material. There is no limit to the size to which it may attain. The texture is loose and friable, easily breaking down, and presenting a chalky or earthy appearance. Crystals of triple phosphate often stud the surface. By heating with the blow-pipe, this calculus fuses into an enamel-like material. Another rare variety is the *basic phosphate of lime* or *bone-earth calculus*, which is very white, chalky-looking, and soft. The exceptional urinary calculi include:—5. *Carbonate of lime*. 6. *Cystine*, usually ovate; yellow, but changing to pale green on long exposure to light; lustrous; mammillated on the surface; friable and soft. 7. *Xanthine*. 8. *Fatty or saponaceous concretions*. 9. *Fibrinous and blood-concretions*. 10. *Alternating*, consisting of alternate layers of two or more primary deposits.

**PATHOLOGY AND ANATOMICAL CHARACTERS.**—Most of the calculi mentioned are of *renal* origin, being derived from a deposit from the urine as it is first excreted, and being formed either within the tubules of the kidney, in its pelvis, or in the infundibula. Such are termed *primary calculi*, and the theories as to the cause of their formation are:—1. The presence of excess of certain normal ingredients in the urine (uric acid, oxalates, &c.); or of some sparingly-soluble abnormal ingredient (cystine, xanthine). 2. Certain conditions of the urine diminishing its solvent power over some of its constituents, such as excessive acidity, or deficiency of chloride of sodium and alkaline phosphates, diminishing the solubility of uric acid and urates; or alkalinity from fixed alkali leading to a deposit of bone-earth phosphate, or of carbonate of lime. 3. The presence of some body suitable to form a nucleus for deposits, for example, a clot of blood, ova of entozoa, or little clumps of urate of soda. 4. The presence of mucus or other colloid substance in some part of the renal apparatus, causing precipitation of urates or oxalate of lime in a globular form, intimately mixed with the animal matter, and thus forming a nucleus for further deposit. (Vandyke Carter and Ord.) The *mixed phosphatic calculus* is almost always formed in the *bladder*, and results from decomposition of the urine, which becomes ammoniacal, this condition being, as already explained, favourable for the deposition of earthy phosphates, which are often mixed with a little urate of ammonia and carbonate of lime. Hence it is described as a *secondary calculus*, and the deposit always takes place on some nucleus, this being generally itself a calculus which has passed into the bladder. Should the urine be retained in the renal pelvis until it becomes ammoniacal, a phosphatic calculus may form there.

In structure a urinary calculus usually consists of a *central nucleus*, surrounded by the *body*, and outside all there may be a phosphatic *crust*. The nucleus may or may not be of the same composition as the rest of the calculus; or sometimes it consists of some foreign body, or of mucus or blood. A section generally shows a stratified arrangement, but it may be partly or entirely radiated. Blended into the structure of urinary calculi there is always a little organic matter, including mucus, epithelium, pus, or pigment.

The morbid effects liable to be excited by renal calculus are:—1. *Hæmorrhage*, from direct injury to some part of the urinary apparatus. 2. *Renal congestion*; or *inflammation* ending in *abscess*. 3. *Pyelitis* or *pyonephrosis*. 4. *Hydronephrosis* and *renal atrophy*, as the result of impaction in the ureter. 5. *Cystitis*. Occasionally urinary calculi



become lodged in cysts or pouches, and give rise to no further mischief. It sometimes happens that one ureter is already occluded, and a stone lodges in the pervious one, leading to complete suppression of urine, with consequent uræmia. Very rarely a stone makes its way out of the renal apparatus into other parts, such as into the peritoneum or intestines.

**SYMPTOMS.**—The clinical signs of urinary calculus are chiefly the consequence of the effects above mentioned, and need not be described here. It is only requisite to point out what symptoms are suggestive of the lodgment of a stone in the kidney or its pelvis; and to describe those which are characteristic of its passage along the ureter to the bladder.

The symptoms of *calculus in the kidney* are pain over the renal region, of a dull aching character, but also frequently shooting towards the testes and thighs; pain at the end of the penis; frequent micturition; and the presence in the urine of blood, pus, epithelium from the pelvis and infundibula, or of considerable unorganized sediments, such as uric acid or oxalates. These phenomena are usually aggravated by anything which disturbs the position of the calculus, especially by violent exercise or jolting, after which the symptoms often assume the characters of *nephritic colic*, this in its typical form being due to the passage of a calculus along the ureter to the bladder. *Nephritic colic* is characterized by sudden excruciating pain in one renal region, shooting in various directions, but especially towards the hypogastrium, testis, end of the penis, and inside of the thigh; great restlessness, the patient tossing about in all directions in order to try to obtain relief; constant desire to micturate, the urine, however, being very scanty or suppressed, any that may be passed being high-coloured, often bloody, and discharged in drops with much burning pain; retraction of the testicles; collapse and faintness, with cold clammy sweats, and a very feeble pulse; generally distressing nausea and vomiting; great anxiety; and sometimes spasmodic movements of certain muscles, or general convulsions. The attack lasts a variable time, there being commonly temporary remissions, and if the calculus reaches the bladder, the symptoms usually subside with equal suddenness, affording a sense of intense relief, and the patient may be conscious of something having fallen into the bladder. If the attack lasts for some time, more or less pyrexia is liable to be set up.

**DIAGNOSIS.**—Patients often complain of pain in the renal region, and imagine that they are suffering from gravel or stone, but such pain is commonly due to extra-renal conditions, such as myalgia, neuralgia, accumulations in the colon, or other causes. Should there be reason to suspect the existence of a *renal calculus*, careful and repeated examination of the urine must be made, particularly for uric acid and oxalates, for epithelium from the urinary passages, or for traces of blood or pus; and it may be well to do this after the patient has taken some severe exercise. As a rule *nephritic colic* is easy to diagnose, but the same symptoms may result from the transit of a blood-clot or of a hydatid vesicle. An attack may also be simulated by the passage of a gall-stone; by severe neuralgia; or by intestinal colic. When a stone reaches the bladder, it can usually be discovered by surgical examination. If there is good reason to believe that a renal calculus exists, it may, under certain circumstances, be permissible to make an exploratory incision to clear up the diagnosis.

PROGNOSIS.—Urinary calculus may be attended with a good many dangers. It may produce extensive disorganization of the kidney; or its passage to the bladder may prove fatal. If a stone is very large, or if there are several calculi, the prognosis is more grave. Calculus is a disease liable to recur.

TREATMENT.—The treatment of urinary calculus is very important. 1. In the first place measures should be taken to *prevent* its formation, if the urine gives indications that there is any danger of the occurrence of this event; or if there has been a previous history of stone. The chief general measures requisite for this purpose are to recommend the patient to drink water freely, so as to maintain the urine in a diluted state; not to allow too long intervals between meals, but to take four or five light meals during the day; and not to remain too much in the recumbent posture, the hours of sleep being moderate. Uric acid calculus is further guarded against by strict regulation of diet, which must be mainly farinaceous, all heavy meals, as well as much meat and rich wines, being avoided; and by administering moderate quantities of bicarbonate or citrate of potash. Oxalate of lime calculus is prevented by keeping the urine very dilute; maintaining the activity of the skin; avoiding certain vegetables rich in oxalates, especially rhubarb and sorrel, and also calcareous waters; and giving alkaline carbonates. Phosphatic calculus is obviated by careful attention to the bladder, if this organ is diseased; and by endeavouring to change the character of the urine. Should this excretion be ammoniacal, it may be desirable to wash out the bladder with dilute acids. 2. It has been deemed possible to *dissolve* calculi after their formation—those of uric acid in the kidneys by administering acetate or citrate of potash freely, in large and frequently repeated doses, continued for a long time; phosphatic calculi by dilute acid injections into the bladder. 3. For *nephritic colic* the remedies are the free administration of opium by the mouth or rectum, or subcutaneous injection of morphia; belladonna, if opium is not admissible; warm baths, with fomentations or poultices over the loins; and the abundant use of warm demulcent drinks. It may be necessary to cup over the loins. Change of posture and manipulation along the ureter have been said to aid in the passage of a calculus. If the pain is extreme, it may be desirable to administer chloroform. Vomiting and collapse must be attended to. 4. *Surgical treatment* is of course usually required when a stone reaches the bladder; and at the present time *nephrectomy* or even *nephrotomy* are coming into more and more favour in the treatment of suitable cases of calculus in the kidney. The treatment of the pathological conditions which may be induced by stone have already been sufficiently considered in their respective chapters.

---

## CHAPTER LXIV.

## CYSTITIS—VESICAL CATARRH.

DISEASES of the bladder are mainly surgical, but it is necessary to allude to cystitis, as this complaint is not uncommon in medical practice.

ÆTIOLOGY.—The causes of vesical catarrh are:—1. *Direct irritation*, especially by calculi and morbid growths; or resulting from certain conditions of the urine, as after taking excess of cantharides, copaiba, beer or spirits, but particularly when this fluid becomes ammoniacal, either as the result of retention from some impediment to its escape, or from paralysis of the bladder in consequence of spinal disease. 2. *Extension* of inflammation in the vicinity, especially that of gonorrhœa. 3. *Exposure to cold or wet*. 4. *Acute exanthemata* occasionally.

ANATOMICAL CHARACTERS.—*Acute* cystitis is characterized by redness, swelling, and softening of the mucous membrane; with the formation of excess of mucus, and the detachment of epithelium with numerous young cells. In the *chronic* form the colour becomes often dirty grey or brown; and there is thickening of the tissues, with, in time, hypertrophy of the muscular coat of the bladder, the walls becoming much thickened and tough. Abundant muco-purulent or purulent matter forms in the bladder, and the surface may ulcerate or even become gangrenous, or suppuration between the coats may take place, ending in extensive destruction and structural changes. The urine is generally decomposed and ammoniacal. This has been supposed to be the result in some cases of an alkaline fermentation set up by the mucus formed in the bladder. Niemeyer and others, however, have advanced the opinion that this decomposition is generally the consequence of the repeated use of dirty catheters, by which low vital organisms are introduced into the bladder.

SYMPTOMS.—The prominent symptoms of *acute* cystitis are uneasiness and a sense of heat over the bladder, in the perineum, and along the urethra; in some cases tenderness over the hypogastrium; constant inclination to micturate, and a difficulty in retaining the urine, a few drops being passed, causing great pain and a sense of burning; and the presence of more or less mucus in the urine. There may be some degree of pyrexia. The chief indication of *chronic* cystitis is derived from the characters of the urine, which contains much mucus and epithelium, or pus, or sometimes blood; and if the urine is ammoniacal, the pus is converted into a gelatinous, ropy, adhesive substance, which can only be poured with difficulty from one vessel into another, and may be drawn out into strings. After a while much constitutional disturbance is often excited, with a tendency to hectic fever; and if extensive suppuration or gangrene should be set up, low typhoid symptoms are liable to arise, or those indicative of peritonitis may supervene.

TREATMENT.—In the first place the *cause* of cystitis must be removed, if possible, especially when this is in the form of a local irritant. In acute cases warm baths and hot fomentations or poultices over the hypogastrium, to which opium may be added, are of service. In some instances removal of blood, by means of a few leeches, is advisable.



The bowels should be kept well-opened, for which purpose enemata may be employed. Suppositories of opium or belladonna are valuable for relieving the local sensations. Barley-water and similar drinks should be allowed freely; and citrate of potash administered, well-diluted, along with tincture of henbane or opium.

In *chronic cystitis* it is important to see that the bladder is properly emptied, and should a catheter be required, care must be taken that this instrument is quite clean, and it may be well to smear it over with carbolized oil. If there is irritability of the bladder, liquor potassæ or the bicarbonate or a vegetable salt of potash should be given, freely diluted, and the salts may be combined with tincture of hyoscyamus. Repeated warm baths are serviceable, or local fomentations may be employed. Stimulants must be avoided, and diluent drinks given freely. Should there be a catarrhal condition of the bladder, attended with the formation of much mucus or pus, it may be desirable to wash out this organ with warm injections containing some *antiseptic*, or with very dilute *astringent* or *acid* injections. Under these circumstances the best internal remedies are dilute nitric acid, with decoction of pareira, buchu, uva ursi, or triticum repens, and tincture of henbane; or balsam of copaiba with liquor potassæ.

---

## CHAPTER LXV.

### DISEASES OF THE ABSORBENT SYSTEM.

THE absorbent system occupies a prominent place in relation to several important pathological processes and conditions, and is at the present time receiving considerable attention from pathologists. As illustrations of these relationships, it may be mentioned that this system is undoubtedly concerned in many septic conditions, and the late Mr. Messenger Bradley classified glanders, malignant pustule, snake bite, dissection-wounds, and erysipelas as forms of septic lymphangitis. Moreover, it is supposed to play an important part in relation to various zymotic diseases, such as plague, typhus, typhoid, and diphtheria. The serous cavities are also now regarded as parts of the lymphatic system, and some cases of inflammation of serous membranes, for instance, of puerperal peritonitis, are considered as being due to lymphangitis. Again, the absorbents are concerned in conveying to various parts of the body morbid products, such as those of cancer, syphilis, and tubercle; and they are often involved in connection with diseases of internal organs. Some structures consist mainly of lymph-follicles, and their chief diseases are associated with these follicles. Certain skin-diseases, such as erythema, have been attributed to inflammation of the lymphatic rootlets; and changes in the lymphatics seem to constitute an important element in the morbid anatomy of elephantiasis and several other affections. These illustrations will suffice to show the importance of the pathological relations of the absorbent system; but in this chapter it is intended to deal only with the more obvious local affections of its vessels and glands, and these need be only briefly discussed, as they belong chiefly to the domain of surgical practice.

## A. CLINICAL CHARACTERS.

The signs to be sought for as indicating disease of the absorbent vessels and glands are:—1. **Morbid sensations** in these structures, especially pain, tenderness, and often a feeling of stiffness about the glands. 2. **Objective changes**.—The superficial lymphatic vessels are visible in certain conditions, and may be dilated. The glands are also often enlarged, this being generally accompanied with some change in consistence. The enlargement may be limited, or may affect the glands extensively throughout the body, not uncommonly giving rise to considerable tumours. The superficial glands are either separate and distinct; or they tend to form nodular, irregular, firm masses, owing to a number of glands being aggregated together; or chains of these structures may be involved. Those of the neck, axilla, and groin are most commonly affected. In the chest they may give rise to the *physical signs* of a mediastinal tumour. They can in certain cases be felt in the abdomen by making steady deep pressure, or by grasping portions of the abdominal walls, either as separate nodules, or as a distinct tumour. The latter is deeply situated, nodular, and fixed, being usually not very large. 3. **Interference with the passage of lymph and chyle**.—It has been supposed that obstruction to the passage of lymph may lead to its coagulation, and to the production of a kind of solid œdema. Interference with the progress of the chyle will gravely affect the nutrition of the body, thus inducing more or less emaciation. 4. **Escape of lymph or chyle**.—This may take place from the vessels or glands, and may also lead to serious consequences. 5. **Evidences of pressure upon, irritation, or destruction** of neighbouring structures.—These phenomena result from enlarged glands, and will of course vary with their situation. Neuralgic pains and localized œdema are not unfrequent symptoms, owing to contiguous nerves and veins being interfered with. Venous thrombosis may also be caused by obstruction of the circulation. In the chest and abdomen more or less pressure-signs may be present, as in the case of other tumours. By the irritation of the glands, inflammation of serous membranes and other structures may also be excited. They are further liable to undergo destructive changes, which may implicate neighbouring parts, thus tending to cause serious mischief. For instance, they may suppurate and destroy the cutaneous structures; in the chest they may lead to destruction of portions of the lungs, or to perforation of air-tubes or vessels; in the abdomen to perforative peritonitis, or to perforation of the bowels. 6. **Constitutional symptoms**.—These are of much importance, necessarily differing in their characters according to the nature of the morbid condition. They may depend upon the disease of the lymphatic system, which, for example, may induce pyrexia or septicæmia; or such disease may be but a part of some constitutional affection, which also causes the general symptoms.

## B. SPECIAL DISEASES.

1. **Acute Inflammation—Lymphangitis—Angeioleucitis—Adenitis**.—The lymphatic vessels may be alone inflamed—*lymphangitis* or *angeioleucitis*; or merely the glands—*adenitis*; or both sets of structures may be implicated. Usually the affection is limited to some

particular part, but in the septic forms of inflammation the absorbent system is extensively involved.

**ÆTIOLOGY.**—This class of affections may be of *traumatic* origin, being due to various forms of injury, such as a wound, contusion, or strain; or they result from various kinds of irritation, such as that induced by neighbouring inflammation, suppuration, ulceration, or diseases of joints. External irritation, as, for instance, the strong heat of the sun, may induce superficial lymphangitis. Special forms of inflammation of the absorbent system are set up by particular kinds of irritation, such as that of gonorrhœa or the syphilitic virus; and septic forms of the disease are induced by various septic poisons. The lymphatics connected with the internal organs are often inflamed when these are the seat of any irritation. Pus has been found in the neighbouring lymphatics in cases of purulent pleurisy. Some forms of pelvic cellulitis have also been regarded as being due to lymphangitis. Inflammation may be immediately excited in the vessels, and then travel along to the glands; or the irritation may be conveyed by the current of lymph to a more or less distant part, the intervening portion being unaffected; or the glands may be implicated by extension from the surrounding cellular tissue. When a gland is involved, while the vessels between it and the source of irritation are unaffected, the inflammation is said to be *sympathetic*. Lymphangitis may set in very rapidly.

**ANATOMICAL CHARACTERS.**—Lymphangitis is distinguished as *reticular* or *tubular*, according as the fine capillary network or the trunks of the vessels are involved. In the former case the skin and its capillaries are usually implicated. In the latter variety the vessels become dilated, and their walls are thickened; the endothelium often disappears; and the internal coat becomes opaque and uneven. The lymph coagulates in their interior, blocking up their channels, and the clot may become organized, obliterating the vessels permanently; or occasionally it softens and suppurates in the centre, and the pus may find its way into the circulation, leading to septicæmia or pyæmia. Exudation also takes place, while the surrounding cellular tissue undergoes hyperplasia and becomes thickened. Lymphangitis may lead to inflammation in joints, which may be of a purulent character. In adenitis the affected glands become congested and swollen, as well as the seat of exudation, while the passage of the lymph through them is impeded. Resolution may take place after a time, but not uncommonly the inflammation terminates in suppuration, this beginning in the centre, the cavities of the glands becoming filled with pus, and the surrounding cellular tissue being also involved. In other cases the glands remain more or less indurated, and they may form adhesions to the surrounding structures, especially if the irritation is repeated several times.

**SYMPTOMS.**—When the superficial lymphatic vessels or glands are inflamed, this condition is evidenced by objective signs. Lymphangitis is indicated by wavy or straight red lines, running towards the glands; or sometimes there are isolated red patches, the skin and its capillaries being involved along with the lymphatics. The large vessels may be felt as firm and knotted cords. If the glands are affected, these can be felt and seen to be more or less enlarged and swollen, at first feeling firm. At the same time pain is experienced, often very acute, with sensations of heat, stiffness, and tenderness. When the affected structures are deeply situated there are no red lines, and redness is not always



present. There is induration of the part, more like that of œdema than inflammation. The inflammation may, however, pass through the intervening tissues from the deep to the superficial lymphatics, and *vice versâ*. Owing to the interference with the passage of the lymph, more or less swelling from lymphatic œdema is often present, of a firm character, and a limb may be much enlarged from this cause. If suppuration should take place in glands, this will be evidenced by the ordinary signs characteristic of an abscess. There is more or less pyrexia, in proportion to the extent and intensity of the inflammation. In septic forms of lymphangitis signs of general septicæmia are likely to arise.

2. **Chronic Adenitis.**—The lymphatic glands are liable to chronic inflammation, which either remains after one or more acute or subacute attacks, or comes on gradually. The glands are then enlarged and indurated, and may be a little painful. They may subsequently suppurate, or undergo a caseous degenerative change, but often remain unaltered for a considerable time. This condition of the glands interferes with the passage of the lymph through them; and also renders them more subject to attacks of acute inflammation from slight causes.

3. **Obstruction of the Absorbent Vessels.**—This condition may involve the lymphatic capillaries, their main trunks, or the thoracic duct itself. It may result from the blocking-up of their channels by coagulated lymph; from inflammation of the walls of the vessels; or from external pressure. Thus the thoracic duct may be more or less obstructed, or even completely obliterated, by the pressure of enlarged glands in the thorax, or of an aneurism. The lymphatic trunks in the limbs may also be compressed by glands, aneurisms, and other morbid conditions; and even the capillaries are subject to pressure in consequence of inflammation of the surrounding cellular tissue. Obstruction of the thoracic duct is said to arise from disease of its valves. It may be remarked, further, that a certain degree of obstruction to the flow of the lymph and chyle may arise from marked interference with the venous circulation, as the result of cardiac disease or direct obstruction of the principal veins.

If the thoracic duct is obliterated, grave general symptoms arise, namely, marked wasting and anæmia, tending towards a fatal issue, if the chyle cannot reach the venous system by the establishment of a collateral circulation. Various degrees of obstruction of this channel have, however, been found in several cases at *post-mortem* examinations, in which no symptoms had been observed during life. The most obvious direct effects of obstruction in the absorbents are dilatation of the vessels behind the impediment, in the course of the circulation; and the development of lymphatic œdema. These conditions necessarily vary much in their extent and degree, according to the situation and character of the obstruction. The dilatation may ultimately lead to rupture of the vessels.

4. **Lymphatic Dilatation—Lymphangiectasis.**—Dilatation may affect the capillary network of the lymphatics; more commonly the larger trunks; or occasionally the thoracic duct or the receptaculum chyli. It presents various degrees, and assumes different forms. Thus there may be merely a localized reticular dilatation of the lymphatic capillaries; or more frequently varicose, saccular, tubular, fusiform, or cirsioid dilatation of the trunks; or the enlarged vessels may form a distinct growth, named *lymphangioma* or *lymphangiectodes*, which has been divided by Wagner into the three varieties—(a) *simple*; (b) *cavernous*; and (c) *cystoid*, in

which cysts are developed. Moreover, enlarged lymphatics constitute an important element in the structure of elephantiasis and other growths, especially elephantiasis lymphangiectodes. The thoracic duct and receptaculum chyli may be enormously dilated, the former in extreme cases reaching the size of the little finger, or even attaining larger dimensions than this. The deep lymphatics and the lacteals are liable to dilatation, as well as those on the surface.

Lymphangiectasis is in many cases congenital, and it has been supposed that this may be due to a want of specialization in the lymphatic system of certain parts. The condition is attributed to different causes. Thus it may follow lymphangitis, in consequence of which the larger tubes are blocked, and the afferent vessels become therefore dilated. In other cases it is not preceded by marked inflammation of the lymphatics, but there is considerable hypertrophy of the cellular tissue, and the vessels enlarge, forming a very free anastomosing network. Again, it is supposed that dilatation may arise from mere hypertrophy of lymphatic plexuses; or from paralysis of the coats of the vessels. Any obstruction from internal plugging or external pressure may lead to enlargement of the vessels, the circulation through which is thus impeded; and probably the obstruction may be occasionally seated in the glands. Elephantiasis lymphangiectodes has been attributed in some cases to the *filaria sanguinis hominis*, as has been already pointed out. Lymphatic dilatation is most frequently met with in warm and moist climates.

When lymphangiectasis occurs on the surface of the body it can be recognized by objective examination. Dilatation of the superficial lymphatics is generally observed on the inner side of the thigh, the sides of the belly, the scrotum, and the penis. It is characterized by vesicles like grains of sago, grouped regularly or irregularly. (Curnow.) Sometimes only ampullæ are formed, which are generally soft and painless. The vessels may rupture subcutaneously, forming vesicles containing a clear or milky fluid. They are also liable to rupture externally, or into various internal parts when situated internally, and it is only thus that the latter can be at all recognized clinically, the escaped chyle or lymph appearing in the stools or urine. Even superficial dilatation of the lymphatics has been not uncommonly mistaken for other conditions, such as hernia, abscesses, and strumous enlargement. The discharge of lymph confirms the diagnosis. If inflammation attacks dilated lymphatics it tends to spread rapidly, and may prove fatal. The clinical characters of lymphatic growths and tumours do not call for consideration here. Congenital cystic formations connected with the lymphatics occur chiefly on the tongue, upper lip, and neck.

**5. Lymphorrhœa—Lymphorrhagia.**—By these terms is signified the discharge of lymph or chyle from the vessels or glands, either on the surface of the body or into some internal part, the amount varying much in different cases. This is sometimes of *traumatic* origin, and in rare cases it may occur from slight wounds, especially in the neighbourhood of joints, which is probably due to a constitutional defect—a *lymphorrhagic diathesis*, corresponding to the hæmorrhagic diathesis. (Bradley.) Usually traumatic lymphorrhœa results from wounds of the thoracic duct, of the larger lymphatic trunks, or of the glands. *Idiopathic* lymphorrhagia is generally due to previous dilatation of the vessels, which ultimately give way. A most interesting case has been reported by Dr. Cayley :\* in which the receptaculum chyli gave way spontaneously as

\* "Pathological Transactions," Vol. xvii. p. 163.



the result of previous extreme dilatation, and fatal peritonitis ensued. Lymphorrhagia may be associated with chyluria, and is then believed to be due to the *filaria sanguinis hominis*.

When lymphorrhagia occurs on the surface of the body, the discharge of the lymph is the clinical sign of the condition. The amount of fluid which escapes varies considerably, ranging from an ounce to five or even ten pounds during the twenty-four hours. It also differs at different times, and the flow has even been known to assume a periodic character, increasing during digestion. The fluid which escapes after injury may be clear and limpid lymph, or mixed with inflammatory products or blood. That which comes away in cases of rupture from dilatation of the vessels is more or less white and milky, like chyle, and it contains a variable quantity of fat. The quantity of fibrin present varies much, and therefore the power of spontaneous coagulation of the fluid. When lymphorrhagia takes place internally, it can only be recognized by the presence of the fluid in the urine or fæces respectively; in the former case giving rise to chyluria, in the latter to fatty stools. Mr. Bradley has called attention to the probable origin of certain cases of hydrocele, hydrocephalus, pleuritic effusion, and ascites from a lymphorrhagia into the respective serous cavities. I have met with a case of ascites which seemed at any rate to be partly due to this cause. As proved by Dr. Cayley's case, the escape of chyle into the peritoneum may set up fatal inflammation. The general condition is more or less affected in cases of lymphorrhagia, in proportion to the amount of fluid lost.

**6. Simple Glandular hypertrophy—Lymphadenoma—Hodgkin's disease—Adenie.**—Hypertrophy of the absorbent glands is an important morbid condition, which occurs, as has been already pointed out, in one form of leucocythæmia, but which constitutes the main anatomical change in the affection known as *Hodgkin's disease*, to which attention will now be briefly directed.

**ÆTIOLGY AND PATHOLOGY.**—Very little is positively known on this matter, but it is assumed that Hodgkin's disease is a primary affection of the lymphatic system, depending upon some special constitutional condition or diathesis, which has been named *lymphadenosis*. By some pathologists it is regarded as malignant, and is placed by Wilks between cancer and tubercle. In many cases the disease seems to begin without any obvious cause, but in others it evidently starts from some local irritation, and such irritation has probably been present in other instances where it has been too slight to attract attention. This complaint is said to be common among French soldiers, and has been attributed to the irritation of the stiff military stock. It may be associated with a distinct scrofulous habit. As regards *predisposing causes*, Hodgkin's disease is most common in early and late adult life; in males; and amongst the poor, its development being aided by bad food, insufficient clothing, cold and damp, and unfavourable hygienic conditions.

**ANATOMICAL CHARACTERS.**—The lymphatic glands more or less throughout the body present various degrees of enlargement. This is first observed usually in the neck, and especially in the sub-maxillary glands, but in most cases other groups are involved in succession, especially the axillary, inguinal, and mediastinal. The disease may, however, begin in other parts, even in internal glands, to which it may be mainly or almost entirely limited. The glands may increase until they attain a very large size, forming considerable masses or tumours. At first they are



distinct, but gradually neighbouring glands become fused into one growth. In exceptional cases they involve the skin. These glandular enlargements usually show no disposition whatever to suppurate or to undergo degeneration. Rarely caseous degeneration does take place, but this is due to an associated strumous diathesis. On section the enlarged glands are seen to be whitish or yellowish-grey. The consistence varies considerably, and the glands may be very soft, yielding a juice on pressure; or firm and dry. The distinction between their cortical and medullary portions becomes lost. Microscopic examination reveals that the enlargement is due to hypertrophy of the glandular tissue, the whole structure being converted gradually into lymph-cells, with a fine network of cellular tissue. In the firmer varieties there is more fibrous tissue.

In addition to the hypertrophy of the lymphatic glands just described, other organs in course of time present growths of a similar nature, especially the spleen, and less commonly the liver, lungs, kidneys, and alimentary canal. Even the canals of the bones may be filled with lymphoid cells. The heart is sometimes atrophied and fatty. Signs of inflammatory and other complications may be met with at the *post-mortem* examination.

**SYMPTOMS.**—When the affected glands in Hodgkin's disease are superficial, their enlargement is evident on objective examination, and their extension and growth can be observed. In the large majority of cases there is neither pain nor tenderness, but if the enlargement is very acute and rapid, sharp shooting pains may be complained of. When situated in internal cavities, the existence of lymphadenomatous growths can generally be made out by *physical examination*. Some of the most important symptoms result from pressure and irritation by the enlarged glands, these necessarily varying according to their position, and their relation to adjoining structures. Obstructive dyspnoea is often a marked symptom when the growth is situated within the chest. Along with the local signs of this disease, the constitution is obviously affected as a rule. This may occur before any local symptoms appear, but usually the general symptoms are gradually developed as the glands progressively enlarge, including emaciation; anæmia and its attendant phenomena, often combined with an appearance of serious illness; marked muscular weakness, the patient often tottering and trembling; and feeble circulation. More or less pyrexia is present in most cases, especially in young patients. Free perspirations are common, and the skin is pale and usually moist. Oedema of the legs is a frequent symptom. The blood does not present any excess of white corpuscles, but is often very watery and wanting in its normal colour, the red corpuscles being markedly deficient. The patient is much depressed and low-spirited, and attacks of syncope are not uncommon. Bronzing of the skin has been said to arise from enlarged retro-peritoneal glands surrounding and compressing the solar plexus.

Unless death should occur from the local effects of the enlarged glands, the *course* of Hodgkin's disease is generally chronic and progressive. Occasionally it is very acute, attended with high fever, profuse perspiration, vomiting and purging, and mental wandering at times. The *termination* is generally fatal, and in most cases death occurs within two years, either from gradual asthenia and exhaustion; from the effects of pressure; rarely from hæmorrhage, owing to perforation of a blood-vessel; or from some intercurrent complication, such as pneumonia,

pleurisy, erysipelas, or Bright's disease. It must be remembered, however, that cases of extensive lymphadenoma may go on for many years. the system being apparently but little affected, and the patient enjoying fair or even good health. In some instances the glands may be much reduced in size by appropriate treatment; while in others they remain enlarged, but show no tendency to progressive increase.

**7. Scrofulous or Tubercular disease.**—In scrofulous subjects, especially children, one of the prominent clinical phenomena in many cases consists in chronic enlargement of the external lymphatic glands, especially those of the neck, there being a tendency to subsequent degeneration and breaking down of their structure, with unhealthy supuration or caseation. In other cases the glands within the abdomen and chest are extensively affected. The enlargement has been attributed either to an unhealthy chronic inflammation; to a deposit of tubercle; or to hyperplasia of the lymphatic elements. Bacilli have been found in the affected glands. They have a very low vitality, and are liable to become speedily disorganized and caseous; they may finally dry up and calcify, or go on to unhealthy suppuration, forming chronic abscesses, which subsequently burst or destroy neighbouring tissues. When the diseased glands are superficial, they are easily recognized. In the chest they constitute the disease named *bronchial phthisis*; and, in addition to giving rise to the signs of a mediastinal tumour, the glands are liable to soften and to form excavations, ultimately involving the lungs, or opening into the trachea or a bronchus, into the pleura, or into one of the great vessels. If they communicate with the air-passages, there is much expectoration of muco-purulent or purulent matter, as well as in many cases of blood, caseous matter, or calcareous particles. When the mesenteric glands are implicated—*tabes mesenterica*—they may be felt separately or as an agglomerated mass, and often give rise to symptoms of peritoneal irritation or inflammation; as well as to flatulence, colicky pains, and various digestive disturbances. Owing to the accumulation of flatus, the abdomen is generally much distended. Appetite may be excessive, deficient or lost, or capricious. The bowels are frequently irregular, being either constipated or relaxed, the stools being also unhealthy. Uncontrollable diarrhoea is likely to be present if the intestines are ulcerated. In exceptional instances the softened glands rupture into the peritoneum or intestines. This variety of glandular disease is usually attended with considerable constitutional disorder, indicated by emaciation, debility, and fever tending towards a hectic type. The loss of flesh is frequently extreme when the lacteal glands are involved; and it is also very marked in bronchial phthisis, if the glands break down. In children, in whom this complaint is much the most common, there is in many cases no evidence of tubercle in other organs; but in adults the lungs or other structures are generally implicated. Recovery may be brought about even when the glands throughout the body have been extensively affected, many of them perhaps remaining as calcified masses.

**8. Albuminoid disease.**—The absorbent glands are often the seat of albuminoid disease. They are then very firm and small; on section presenting the characteristic pale, homogeneous, waxy appearance. In the abdomen they can be felt as little hard masses, separate, and readily movable. The constitutional symptoms are those of the general disease.

**9. Cancer.**—As a secondary deposit, cancer is very liable to implicate the absorbent glands in the neighbourhood of any structure which may

be affected with this disease. It may also commence in them primarily. All forms are met with; and large, hard, nodulated tumours are often formed. The clinical phenomena are mainly those due to the presence of the tumour; with evidences of the cancerous cachexia. The glands are generally very painful and tender.

10. **Rickets.**—In this disease the superficial glands often become hard and shotty; and the mesenteric glands may be enlarged.

#### GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. **DIAGNOSIS.**—As regards the *lymphatics*, sufficient has been already said to indicate the data upon which the diagnosis of their several affections is founded. In forming a diagnosis of *glandular diseases*, special attention must be paid to the constitutional condition; and to the physical characters presented by the glands. The main difficulty lies in determining the existence of disease of the glands in internal cavities; and in making out their exact condition. Glandular disease must always be borne in mind as a prominent cause of wasting in children, and especially affections of the mesenteric or bronchial glands.

2. **PROGNOSIS.**—This will depend on the nature and cause of the disease present; the state of the constitution; the situation and extent of glands which are affected; and the changes which they undergo. Acute inflammatory affections may prove very serious. In internal cavities enlarged glands, by their mere pressure or destructive effects, are liable to give rise to serious mischief. Extensive glandular disease in children, particularly when the mesenteric glands are involved, is very dangerous.

3. **TREATMENT.**—*a.* In *acute lymphangitis* and *adenitis* the principles of treatment are to remove any cause of the inflammation; to keep the affected part at rest; and to use warm fomentations and poultices freely. Sometimes leeches may be applied with advantage. Should suppuration be set up, this must be managed on ordinary principles. *b.* In *chronic* affections connected with the absorbent system, constitutional treatment is often of the first importance, especially when the glands are involved. This must be adapted to the nature of the disease, but the measures which are generally serviceable include the administration of good nutritious diet, with plenty of milk; attention to all hygienic matters, with change of air, especially to the sea-side, sea-bathing being often very beneficial; regulation of the digestive functions; and the use of cod-liver oil, quinine, iron in some form, especially syrup of the iodide or phosphate, and other *tonics*. Iodide of potassium and liquor potassæ have been supposed to influence the size of the glands. It is the custom to use various external applications over enlarged glands, with the view of diminishing their size, especially ointments of iodine or iodide of potassium or lead; tincture or liniment of iodine painted over the skin; lotions of iodine and iodide of potassium; spirit lotions, or those containing nitrate or chloride of ammonium; or sea-weed poultices or fomentations. In many instances undoubtedly much good may be thus effected, but certainly it is necessary to be careful in employing strong applications, such as those of iodine, and also in practising violent friction, as injurious irritation and inflammation may thus be induced. Gentle friction with some simple oleaginous substance is frequently beneficial. Should abscesses form, they must



be treated by poulticing and incisions. Symptoms due to enlarged glands in internal cavities must be attended to as they arise. Some practitioners recommend irritant injections into the substance of chronically enlarged glands. Extirpation has been occasionally practised, but this is not a desirable mode of treatment. The administration of phosphorus may diminish the size of the glands in Hodgkin's disease, but is for other reasons not to be recommended. The constant current has been employed in some cases with success. For the chronic conditions connected with lymphatic vessels, such as dilatation or lymphorrhagia, careful bandaging is useful, or elastic pressure may be employed; kneading may help to remove lymphatic œdema.

---

## CHAPTER LXVI.

### DISEASES OF THE THYROID GLAND—BRONCHOCELE—GOITRE.

ONLY two classes of disease affecting the thyroid body, which are likely to come under the notice of the physician, will be described in this chapter.

I. In certain districts enlargement of the thyroid gland—**bronchocele** or **goitre**—prevails as an endemic disease. It is particularly observed at the base of high mountains. In this country goitre prevails especially in Derbyshire—hence named “Derbyshire neck;” and in certain parts of Yorkshire, Sussex, Hants, and Nottinghamshire. It has been attributed to various meteorological and other causes, but the mass of evidence goes to prove distinctly that it is due to impregnation of the drinking water with excess of lime and magnesia salts, these salts having been dissolved in the percolation of the water through the geological strata containing them. The disease is observed most commonly in females; and is rare before puberty, though it has been known to be congenital.

**ANATOMICAL CHARACTERS.**—The degree of enlargement of the thyroid varies considerably, and the gland may attain very great dimensions. It begins in the isthmus, or in one lobe, especially the right, but ultimately usually involves the entire organ. The shape of the gland is generally altered, distinction of its several parts being lost, but this is not always the case. At first the tumour is soft, but gradually becomes firmer, and may ultimately be exceedingly hard. In the early stage there is simple hypertrophy of the gland—*simple bronchocele*—with the formation of a glutinous, ropy, colloid fluid in its cells; afterwards the blood-vessels increase in number and become dilated, while numerous cysts form—*cystic bronchocele*—containing either the gelatinous material or a bloody-looking fluid. Ultimately calcareous matter is deposited, and the gland may be converted into a calcified capsule, enclosing cysts, various kinds of fluid, and calcareous aggregations. Inflammation and suppuration or ulceration may arise, altering materially the characters of the enlargement.

**SYMPTOMS.**—The thyroid gland presents an obvious swelling, varying in its size and other characters; and the whole or part of the gland being involved, it may compress the neighbouring structures, and lead to serious dyspnœa, dysphagia, or interference with the circulation in the neck. The general system is usually below par, there being often much debility and anæmia. In some valley-districts bronchocele is associated with *cretinism*, a condition characterized by marked mental deficiency, with atrophy of the brain; as well as by bodily deformity.

**TREATMENT.**—In this form of bronchocele the principles of treatment are to change the residence; to avoid drinking the impregnated water; to employ iodine both internally and externally; and to improve the condition of the system by means of iron. Iodine has gained the reputation of being almost a specific for goitre, and the best mode of administration is to give the tincture in small doses with iodide of potassium, freely diluted. Iodide of iron is also very useful. Externally applications of the tincture of iodine, iodine ointment, or ointment of iodide of mercury are chiefly employed. The combined employment of ointment of biniodide of mercury, and exposure to a tropical sun or to the heat of a strong fire, has been specially recommended. Pressure may be beneficial in reducing the enlargement. If this treatment is unsuccessful, surgical interference is advocated, especially injection of the gland with some irritant, such as diluted iodine or tincture of steel; the passage of a seton or wire through the tumour; ligature of the thyroid arteries; or, as a final resource, extirpation of the enlarged organ.

**2. Exophthalmic goitre—Graves's or Basedow's disease.**—This is a highly interesting complaint, characterized by palpitation of the heart; marked pulsation in the vessels of the neck and head; enlargement and usually pulsation of the thyroid gland; and prominence of the eyeballs or exophthalmos. It is observed by far most commonly in young women from twenty to thirty years of age, but may be met with in males, who are usually somewhat advanced in age. Almost always, but not invariably, the condition is associated in females with marked anæmia and menstrual derangements. The patients are often of nervous temperament, and the complaint may be directly traceable to some powerful nervous disturbance. Pathologically exophthalmic goitre is believed to be the result of paralysis of the vaso-motor nerves supplying the vessels of the thyroid gland, and of the head and neck; and of undue stimulation of the accelerating nerves of the heart. The enlargement of the thyroid, which is not usually very great, is due mainly to dilatation of its vessels, partly to serous infiltration of its tissues, and after a time to hypertrophy; very rarely do cysts form. The exophthalmos is supposed to result either from the eyes being pushed forward, owing to the dilated vessels, and increased vascularity, œdema, and probably hyperplasia of the fat behind them; to contraction of the muscular tissue in the membrane which covers the sphenomaxillary fissure; or to a combination of these causes. There is no satisfactory evidence in support of the notion that the nervous disturbance is due to any obvious change in the lower cervical ganglia of the sympathetic, as has been suggested, although increase in the connective tissue and diminution of the nerve-cells in these ganglia have been described.

**SYMPTOMS.**—As a rule the subjects of exophthalmic goitre are very anæmic or chlorotic, and they often become low-spirited or irritable before the actual symptoms appear. Palpitation is usually noticed

for some time before the other characteristic phenomena, and these generally come on very gradually. The enlarged thyroid feels soft and elastic, while it pulsates or presents a peculiar thrilly sensation; and frequently a hæmic murmur is heard over it. The degree of enlargement varies in different cases, and the gland is usually unequally affected. The pulsation may even be visible at a distance. The degree of exophthalmos also varies, but it may become so great that the eye-balls project considerably, so that the eye-lids cannot cover them; hence grave destructive changes are liable to be set up in these organs, due to inflammation and ulceration. Their movements may be much impaired, and often a certain degree of impairment of the co-ordination between their movements and those of the eyelids is observed, so that when the eyes are quickly cast down the eyelids do not follow them, and the sclerotic is visible below the upper eyelid. Vision is generally unaffected. The exophthalmos is sometimes preceded by a spasmodic contraction of the levatores palpebrarum, which is said to be very characteristic. The cardiac action is often very rapid, and may be irregular. A basic murmur is not uncommon, due to the excited cardiac action, and the anæmic condition of the blood. The carotid arteries are frequently seen to throb violently; and they are sometimes dilated. A murmur may be audible in them and in the subclavians. The increased pulsation is evident even in the smaller arteries. Uncomfortable sensations of throbbing and fulness in the head, giddiness, and headache are often complained of; while the face is liable to flush. Patients suffering from this affection often feel weak; they are liable to free perspirations; and their temperature is frequently raised. They not uncommonly suffer from dyspeptic symptoms. The urine may be excessive and watery; or sometimes it is albuminous. Among other phenomena noticed may be difficulty of breathing, hoarseness, or aphonia, due to the enlarged thyroid; and a sensation of fulness and throbbing in the throat. The characteristic symptoms are usually much increased by exertion and emotion. Many cases improve or recover under proper treatment, but the course and duration of exophthalmic goitre are very variable. Death may result from the consequences of gradual dilatation and weakening of the heart; from general wasting; from interference with respiration; or from intercurrent disease.

TREATMENT.—In cases of exophthalmic goitre the treatment must be mainly directed to the general state, iron, quinine, and other *tonics*, with nutritious digestible food, careful attention to hygienic conditions, especially as regards fresh air, and moderate exercise, being the chief remedies required. Digitalis is useful, on account of its influence upon the heart; and belladonna is also decidedly efficacious in some cases, in combination with iron. Ergot has been recommended; and also galvanism of the sympathetic in the neck. Care must be taken to prevent the eyes from becoming injured, and with this object a shade may be worn; or the eyelids may be closed by means of a light bandage, if necessary.

---



## CHAPTER LXVII.

## MYXŒDEMA.

MYXŒDEMA is a peculiar disease, originally described by Sir W. Gull, but more recently investigated and named by Dr. Ord, in which the tissues of the body are invaded by a form of dropsy, the fluid yielding mucus and having a jelly-like consistence; there being no albuminuria or other signs of primary disease of the kidneys.

ÆTIOLOGY AND PATHOLOGY.—In Dr. Ord's original experience myxœdema almost always occurred in adult women, especially in married women; and the complaint occasionally followed pregnancy. Subsequent observations have shown, however, that men are not uncommonly affected, and in Sir Andrew Clark's experience males suffered more frequently than females, in the proportion of about seven to three. Dr. Amand Routh has brought before the Medical Society a case of sporadic cretinism and myxœdema combined, in which the latter condition probably commenced in very early life. No definite causes of the complaint have yet been made out, but in some cases severe hæmorrhage has previously occurred. There is much doubt as to the pathology of myxœdema, but the condition of the thyroid body in this disease, the results of experimental removal of this body, and the remarkable observations of Prof. Kocher, of Berne, on the effects of extirpation of the thyroid in patients, seem to point to atrophy of this gland being in some way the cause of myxœdema, and to an important relation existing between this complaint and the cretinoid state. Kocher's description is the description of myxœdema, although he knew nothing of the disease. The condition of the thyroid body varies considerably in cases prior to the development of myxœdema. Sometimes it is preceded by exophthalmic goitre; or this condition may supervene on myxœdema.

ANATOMICAL CHARACTERS.—In myxœdema the connective tissue in all parts of the body presents a remarkable overgrowth, with a kind of retrograde degeneration. Its fibres are increased, and unnaturally defined; the corpuscles are enlarged and multiplied; and the interstitial material is enormously augmented. The skin is much swollen, translucent, deficient in secretion, and infiltrated with mucus, yielding an immense amount of mucin. The thyroid body is markedly atrophied. The mucous membranes, glands, muscles, and central ganglia of the nervous system are similarly affected; and the outer coat of the arteries seems to be involved to a great degree. The condition of the kidney closely simulates subacute interstitial nephritis. The proper structural elements of the different tissues are gradually absorbed, by the effects of the pressure of the new material. The increase in the neuroglia in the nerve-centres is often very marked, leading to destruction of their elements.

**SYMPTOMS.**—Those who are the subjects of myxœdema present a peculiar and characteristic appearance, due to the mucoid œdema. The face is universally swollen, all the features being equally and uniformly involved, without reference to gravitation. Thus the eyelids and lips are enlarged; the alæ nasi are thick and broad; the ridges of expression are blurred and coarsened, or the lines obliterated. The skin is peculiarly waxy-looking and anæmic; but the cheeks are overspread with a dull pink flush, in vivid contrast, and abruptly limited towards the orbits. The body generally is similarly swollen; and the hands lose all shapeliness, being described by Sir W. Gull as “spade-like.” The œdema is resilient, and does not pit on pressure. The skin throughout is thickened, translucent, dry, and rough, there being little or no perspiration. As myxœdema advances, ordinary anasarca often supervenes. The temperature is almost always lower than normal, and may fall to  $94^{\circ}$  or even lower. Generally the patients complain of constant chilliness. The mucous membranes which can be seen and felt present similar characters to the skin. A peculiar appearance results from a diminution in the size, or almost complete disappearance of the thyroid body; and a correlated tumefaction, with marked resilience of the skin, in the lower triangle of the neck, above the clavicle. The hair is often scanty; and the teeth decay early.

Another characteristic group of symptoms in myxœdema are those connected with the nervous system. They are indicative of progressive hebetude involving the intellectual faculties, sensation, and voluntary motion; and ultimately mental disorder often supervenes, terminating in coma. The expression of the face becomes fixed, heavy, and very sad. Thought and volition are slow, but lead to correct results. Patients are markedly or even painfully conscious of a want of normal activity in performing the ordinary actions of daily life. They have a difficulty in collecting their thoughts, ideas come slowly and deliberately, and are tardily expressed in conversation, while to write a letter takes a long time. The language is, however, correct, and the writing is unchanged. There is a condition of lethargic good temper. Later on memory becomes defective. Articulation is slow and laboured; and the voice monotonous and of leathery quality. The movements of the body are slow and languid; and the maintenance of fixed attitudes requires much effort. There is no loss of muscular power, but a tardiness of co-ordination, and a torpidity of muscular sense; and it appears as if the muscles were toneless and excessively relaxed during rest, so that a considerable initial contraction is necessary before they bear on their attachments. While the patient is at rest, laxity of the muscles in some instances gives rise to drooping of the head on the chest. In walking the balance of the body is maintained with difficulty, its weight being thrown on each leg in succession; and a quiver often runs through the body when the foot is raised from the ground. Moreover, sudden falls are not uncommon; and this has led to fracture of the patella. Sensation is also slow, but ultimately sure. The special senses are similarly affected; and two particular symptoms are often noticed, namely, a persistent unpleasant taste, sweet, bitter, or of other characters; and a persistent unpleasant smell.

In the early stage of myxœdema there are no evidences of organic disease of either of the viscera, but these are ultimately set up. The

urine is at first usually increased in quantity; of low specific gravity; but contains no abnormal ingredients. In the last stage it generally becomes albuminous.

The *course* of myxœdema is very chronic, but the termination generally fatal, the duration ranging from six years upwards. Ultimately there is great general debility; while mental symptoms supervene, such as fretfulness, irritability, and moroseness, often delusions and hallucinations, and speedy coma at the close. Death may result from coma, uræmia, or inanition. Some cases have improved under treatment, and Sir Andrew Clark looks upon myxœdema as fairly curable.

**TREATMENT.**—The chief indications in the treatment of myxœdema are to give good food, at the same time carefully regulating the diet; to protect the patient from cold; to employ *tonics*, especially iron and arsenic; and to use assiduous frictions, as well as baths, especially vapour-baths. Jaborandi and nitro-glycerine have been found beneficial by Dr. Ord.

## CHAPTER LXVIII.

### DISEASES OF THE NERVOUS SYSTEM.

#### I. CLINICAL CHARACTERS.

THE clinical investigation of nervous affections is frequently very difficult, and there is no class of diseases in connection with which a definite and systematic mode of examination is more requisite. Further, it is highly important to have a clear notion as to the anatomy and physiology of the nervous system, particularly as to the functions of the different parts of the central organs, and the distribution and functions of the nerves. The clinical phenomena which may be associated with the nervous system will now only be enumerated, but some of the most important of these phenomena will be hereafter discussed in detail. They may be indicated as follows:—

1. **Morbid sensations in the head**, including headache, tenderness, sense of weight or heaviness, throbbing, heat, vertigo or dizziness.

2. **Morbid sensations connected with the spine**, chiefly pain, tenderness, burning, or a peculiar sense of tightness round the body, as if it were encircled by a tight cord extending from the spine—*girdle-pain*. As regards spinal pain, it is important to notice whether it is felt all along the spine or is localized; if it is constant or paroxysmal; if it shoots in any direction; and in some cases how it is influenced by walking, by movements of the spinal column, by percussion or kneading along the spine, by concussion of the heels, and by the passage of ice or of a hot sponge along the spine.

3. **Mental disturbance.**—It is impossible to indicate here all the numerous derangements coming under this head, especially as observed



in cases of insanity, but their general character may be gathered from the following summary :—*a.* Impaired consciousness, from mere stupor to absolute coma. *b.* Disorder of the intellectual faculties, including perception and apprehension, thought, reasoning, judgment, and memory. Under this would come the various forms of delirium, mental confusion, delusions, illusions, and hallucinations; or there may be mere failure or complete loss of one or all of the mental powers. In some cases the mental faculties are unnaturally acute. *c.* Alterations in the moral feelings and actions, manner of behaviour, disposition, affections, spirits, and temper. *d.* Emotional disorder, as evidenced by the emotions being unduly excitable or the reverse. *e.* Disturbance of speech as an intellectual act—*aphasia*. *f.* Disorders affecting sleep, including somnolence, insomnia, uneasy sleep with unpleasant dreams, somnambulism, and somniloquism:

**4. Subjective disturbances of the special senses.**—*a. Vision.* The chief derangements of vision are photophobia or undue sensibility to light; photopsia or subjective sensations of flashes of light, of iridic colours, sparks, *muscæ volitantes* or spectra; defective sight, either dimness of vision to complete blindness or amaurosis, double vision or diplopia, hemiopia, part of the field of vision being lost, or altered perception of colours. *b. Hearing* may be affected, as shown by intolerance of sound; different degrees of deafness; or tinnitus aurium. *c. Smell* or *taste* may also be impaired or lost; unduly sensitive; or perverted.

**5. Alterations in general sensation and tactile sensibility.**—These comprehend :—*a.* Hyperæsthesia, or undue sensibility to touch; and dysæsthesia or hyperalgesia, or undue sensibility to pain. *b.* The opposite condition of hypæsthesia, anæsthesia, or analgesia. There may be a sensation of something intervening between the skin and any object touched; or inability to distinguish the characters of the surface, or the form of an object. *c.* Various kinds of pain or tenderness felt in different parts of the body. *d.* Paræsthesiæ or perverted sensations, such as numbness, tickling, itching, formication or creeping of ants, heat or cold, pricking, tingling, aura epileptica. *e.* Thermo-anæsthesia and thermo-hyperæsthesia. *f.* Diminished rapidity of conduction to the brain, and of consequent perception of tactile or painful sensations.

**6. Impairment of the muscular sense.**—This deviation exists in certain forms of disease, and is evidenced by a difficulty in realizing weight and resistance, or in performing certain muscular acts without looking and paying attention to them; or by a want of consciousness as to whether different muscles are contracted or not, or whether joints are being passively moved.

**7. Alterations affecting motility.**—These are of the greatest importance, and comprise :—*a.* General restlessness and jactitation. *b.* Abnormal attitudes or movements while lying, sitting, standing, or moving, such as being coiled up; boring the head into the pillow; a disposition to stagger or fall, to advance irresistibly or run, to rotate on an axis, or to proceed in a circle. *c.* Evidences of muscular weakness, in the way of general trembling; local tremors; or unsteadiness of a limb when raised. *d.* Signs of undue muscular irritability, namely, fibrillar movements; twitchings, subsultus tendinum; rigidity; clonic or tonic spasms; convulsive movements; and cramps or painful spasms. As varieties of symptoms due to spasmodic movements should be specially

mentioned strabismus; rolling about of the eyes, or the condition termed nystagmus, in which the eyeball presents continuous oscillatory and rotatory movements, which cannot be voluntarily controlled, or only occurring on voluntary movement; champing of the jaws; grinding of the teeth; and trismus or lock-jaw. *e.* Paralysis, either general, hemiplegic, paraplegic, or local. *f.* Loss of power in co-ordinating muscles for the performance of various acts. *g.* Automatic involuntary movements, such as those observed in chorea. *h.* Deficient or excessive reflex irritability. *i.* Cataleptic fixity of a limb; or slow purposeless movements of flexion and extension.

8. **Changes in vascular supply, nutrition, and secretion.**—The influence of the nervous system on these processes is well known, and it will suffice to mention here, as illustrations, the change in temperature and the wasting, with tendency to bed-sores, often observed in paralyzed parts; the disorders of nutrition and secretion which frequently accompany neuralgia; and the influence of nervous affections on the secretion of tears, saliva, or urine.

9. There are some important **extrinsic** symptoms often associated with nervous diseases, namely, those referable to the stomach, bladder, bowels, and sexual organs, many of them being due to paralysis. They include nausea and vomiting; obstinate constipation, accumulation of feces in the rectum, involuntary or unconscious defecation; irritability of the bladder, retention or incontinence of urine, involuntary micturition; diminution or loss of sexual inclination or power, undue sexual excitability, or constant priapism.

## II. PHYSICAL EXAMINATION.

Objective modes of examination are highly important in the investigation of nervous diseases, and the following outline may serve to indicate the nature and scope of the information to be thus obtained:—

**A. Examination of the head** as to size and shape; state of the fontanelles; or signs of tumour.

**B. Examination of the spinal column** as to shape, or evidences of a tumour; or as to sensations on palpation, percussion, or the application of heat or cold.

**C. Tests of sensation.**—In testing sensation it must be remembered that there are really several kinds of sensation, each of which it may be necessary to investigate. These include (1) *Common cutaneous sensibility*. (2) *Painful sensations*. (3) *Tactile sensations*, or those of touch, which comprehend (*a*) pressure; (*b*) temperature; (*c*) locality. (4) *Muscular sense*. In many cases much care and tact are necessary in testing these different kinds of sensation, and in discriminating between them. Without entering into details, it must suffice to give the following summary. *Cutaneous sensibility* is tested by noting the effects of a slight touch or pressure, or of tickling, pinching, or pricking the skin; or more accurately by the faradic current. The sense of pressure is best tested by applying different weights over the part to be examined. *Temperature* may be determined by applying to the skin two test-tubes, one containing cold, the other hot water. *Locality* can be tested by

pinching or pricking the skin while the patient is not looking, or has his eyes shut, and making him state the seat of the irritation. Special instruments are also employed, such as Weber's, Jaccoud's, or Sieveking's æsthesiometer. Weber's instrument consists essentially of a pair of compasses, the points of which are covered with sealing-wax, and it is noted what is the shortest distance apart at which these can be recognized as separate points of touch, an approximate idea being thus gained as to the degree of sensibility of a particular part. The *muscular sense* is tested by making the patient lift weights suspended in a towel or handkerchief, and noticing the smallest weight which can be recognized as such, or the smallest difference between two weights which can be appreciated; by directing him, his eyes being closed, to perform certain acts, such as to point to or touch the nose or ear, or the great toe, or to place his legs in different postures; or by placing the limbs in different positions, and ascertaining whether the patient can, without looking, state in what positions they are.

**D. Tests applied to muscles.**—*a.* When any muscles are supposed to be paralyzed, the patient should be desired to attempt to perform different actions which would bring the affected muscles into play, and thus it can be observed whether they are really paralyzed, and to what degree; it being noted whether these actions can be performed at all, or if they are merely slowly produced and deficient in power. An instrument named the *dynamometer* has been invented by Duchenne, for the purpose of measuring and recording accurately the strength of the hand-grasp; and also of measuring the force of traction which can be exerted by other groups of muscles. *b.* When there is impairment or loss of co-ordinating power, the involved muscles should also be tested in an appropriate manner; thus the patient may be made to balance the body in an erect position, or to walk along a straight line without looking. *c.* A matter of frequent importance is to determine whether muscular irritability is retained or lost, as well as the degree of facility with which it is excited, and the force with which the muscles act when thus irritated. This is accomplished by employing some direct mechanical irritation, such as pressure, percussion, or drawing the finger along the muscles; but especially by electricity. In many cases also the reflex excitability of muscles has to be tested, but this subject must be separately discussed.

**E. Examination of reflexes.**—During the last few years certain phenomena have come to assume an important position in relation to nervous diseases, which are known by the general term *spinal reflexes*. These are tested as to their presence; their absence or abolition; or their exaggeration or impairment. They are divided into two primary groups, namely:—1. *Superficial* or *skin-reflexes*. 2. *Deep* or *tendon-reflexes*.

*Superficial reflexes* depend on impressions conveyed by the ordinary afferent or sensory nerves, when certain parts of the skin are irritated. *Tendon-reflexes* are produced by a sudden impulse or blow applied to the tendon or aponeurosis belonging to a muscle.

The modes of production and indications of these reflexes are conveniently arranged by Dr. Bastian, according to the following tables: \*—

\* See article on "Spinal Cord, Diseases of," in Quain's Dictionary of Medicine, pp. 145 and 1458.



## 1. SUPERFICIAL REFLEXES.

Name of Reflex.	Mode of Excitation.	Nature of Result.	Level of Cord upon which Reflex depends.
<i>Plantar reflex.</i>	Tickling sole of foot.	Movements of toes; of these and foot; or of these and leg.	First, second, and third sacral nerves (lower part of lumbar enlargement).
<i>Gluteal reflex.</i>	Irritation of skin of buttock.	Contraction of glutæi.	Fourth and fifth lumbar nerves.
<i>Cremasteric reflex.</i>	Irritation of skin of upper and inner part of thigh.	Drawing up of testicle.	First and second lumbar nerves.
<i>Abdominal reflex.</i>	Irritation of skin of abdomen along edge of ribs, and above Poupart's ligament.	Contraction of upper or of lower part of abdominal muscles.	Eighth to twelfth dorsal nerves.
<i>Epigastric reflex.</i>	Stroking side of chest over sixth and fifth intercostal spaces.	A dimpling of corresponding side of epigastric region (contraction of highest fibres of rectus abdominis).	Fourth to sixth or seventh dorsal nerves.
<i>Scapular reflex.</i>	Irritation of skin in interscapular region.	Contraction of posterior axillary fold (teres), or of several of scapular muscles.	Sixth or seventh cervical to second or third dorsal nerves.

## 2. DEEP REFLEXES.

Name of Reflex.	Mode of Excitation.	Nature of Result.	Level of Cord upon which Reflex depends.
<i>Knee jerk.</i>	By striking patella tendon with edge of hand or with percussion hammer, whilst leg hangs loosely over fellow, or over forearm of operator. Also by striking quadriceps tendon above patella.	A single upward jerk of the leg and foot, slight or distinct.	Second and third lumbar nerves.
<i>Ankle clonus.</i>	With knee extended or very slightly flexed, by pressing quickly and firmly against anterior part of sole of foot (so as to stretch calf-muscles) and then keeping up the pressure.	A series of clonic contractions at the ankle-joint, continuing as long as the pressure is maintained, and instantly ceasing when it is relaxed. If the condition is very highly marked it may spread to the whole limb, or even to that of the opposite side.	First to third sacral nerves (lower part of lumbar enlargement).

A modification of the ankle-clonus has also been described under the term *front-tap contraction*. It is obtained by tapping on the muscles of the front of the leg during passive flexion of the ankle. Deep reflexes can also occasionally be produced in the upper limb, though not present in health, and they are said to indicate secondary descending degeneration after brain-lesions. Eulenberg indicates the movements of tendon-reflexes by *graphic representation*.

*F.\* Uses of Electricity.*—Electricity is of use for the diagnosis,

\* The following observations on the "Uses of Electricity" have been written by my friend Dr. Vivian Poore.

prognosis, and treatment of diseases, especially of the nervous and muscular systems. It is used in three forms, namely :—

1. Electricity produced by *friction*, otherwise known as *static* or *Franklinic electricity*, requiring for its application rotating cylinders or discs of glass, Leyden jars, insulating stools, &c. This form is so difficult to manage, and the apparatus is so cumbersome, that it is now rarely used, and may be disregarded in the present volume.

2. Electricity induced by *magnets* or neighbouring currents of electricity. This is called the *induced current*, the *Faradic current*, the *magneto-electric current*, or the *interrupted current*. Its most common form is that in which a coil is made to rotate between the poles of a horseshoe magnet. The best form is that in which a coil of coarse wire having a core of soft iron in its middle is connected with the two plates of a galvanic element. Over this primary coil another coil of finer wire is made to slide. The current of the primary coil being automatically made and broken with very great rapidity by means of a spring hammer, currents of electricity are induced in the coil which slides over the primary coil, and the strength of such induced currents can be graduated with very great delicacy by the extent to which the outer coil covers the inner.

3. Electricity produced by *chemical action* in cells, such cells being technically known as “elements.” This is called *Galvanic* or *Voltaic electricity*, and is sometimes spoken of as the *constant current*. The elements must be “constant” in their action, and none are better than those which are known as the Leclanché elements (of which there are many imitations), and which are now in common use for a variety of purposes. A medical galvanic battery should be composed of *small* cells, not less than twenty in number and united in *series*, that is, the *zinc* plate of one element joined to the *carbon* plate of the next, and so on. In such a battery the current flows from the last carbon plate to the first zinc plate. The wire connected with the carbon is spoken of as the *positive* pole, and that with the zinc as the *negative* pole, and it must be remembered that the current always flows from positive to negative. The negative pole is more stimulating and irritating than the positive.

In *diagnosis* electricity is used to test the *irritability* of nerves (motor and sensory) and muscles. Great care is required in the employment of electricity for such a purpose, and we shall best explain the method to be used by giving directions for testing a particular group of muscles, say those of the hand. This is most readily done by comparing the irritability of a muscle on the right side with the corresponding muscle on the left. We must be sure in the first instance that the muscles are in perfect repose, and it is often impossible to get this condition fulfilled without the intelligent help of the patient. Let the patient, stripped to the waist, sit in a comfortable chair with both hands arranged symmetrically in front of him and resting in perfect repose on a small table of suitable height, or on the back of a chair. Next we must be sure that the current passes through equal lengths of the body to the muscles on either side; this is most certainly effected by fastening one pole to the middle line of the body (say the nape of the neck). If telegraph wire conductors be used (and they are the best) it will be found a good plan to place a small toilet sponge moistened with salt and water over the lower cervical vertebrae, and over this to fasten the bright copper wire of the conductor by means of a strip of bandage tied round the neck. Next we must be sure that the skin over the muscles to be tested is equally pervious (if we may use the expression) to electricity, and this

is best effected by thoroughly moistening the skin with *hot salt and water*. Having fastened a sponge-holder or rheophore of suitable shape to the other conductor of the battery, sit facing the patient, and proceed to test the muscles and compare the irritability of one side with the other. It is well to begin with the side which is presumably healthy. Let your current be at zero to begin with, and gradually increase its strength until a distinct movement of the muscle is obtained. This done, apply the same current in a precisely similar way to the opposite side and compare the results. We may find that the irritability is lost, lessened, or heightened, or that a muscle which gives no response to faradism contracts more or less readily to galvanism.

Irritability to both forms of current is *lost* if the muscle be so extremely wasted that no appreciable amount of fibre remains. It is *lessened* in all cases in which muscles have been disused for some time, as when limbs have long been kept in splints or immovable apparatus, and in old cases of hemiplegia and hysterical paralysis. Irritability when lessened from these causes is usually quickly recovered after a few applications of the current. It is often lessened in diphtheritic paralysis, in moderately severe cases of lead-paralysis, and in cases of nerve-injury which are recovering. Irritability is lessened to both forms of current when muscles have been over-used, as in writers' cramp, and in some cases of chorea. In cases of locomotor ataxy it is sometimes found that the muscles of the affected limb vary in their irritability, some showing excess and others diminution. After an injury to a nerve which completely stops its conducting power, it is common to find that the irritability to faradism and galvanism of muscles supplied by the injured nerve undergoes a gradual diminution, until at the end of seven or eight days the irritability to faradism is completely lost, while the irritability to galvanism revives and ultimately becomes excessive.

Irritability to both currents may be apparently lessened in cases where the skin is very dry and harsh, and the current finds great resistance to its transit through it.

Irritability of muscles to faradism or galvanism may be *heightened* in some cases of *slight* nerve-injuries, in which the nerve is bruised or tender. It is apparently heightened in emaciated and anæmic subjects.

It is noteworthy that in some forms of progressive muscular atrophy the irritability to both forms of current remains normal as long as there is any muscle remaining.

Sometimes in the early stages of hemiplegia muscular irritability is heightened; and in cases of paraplegia from a limited lesion leading to degeneration of the lateral columns of the cord a similar condition of things is found.

*Degenerative Re-actions.*—In the condition thus named it is found that muscles give no response to faradism, but respond readily, or too readily, to galvanism. When a motor nerve is divided or obliterated by pressure, or when the motor cells in the cord or brain from which it emanates are destroyed, degeneration travels down the nerve, and ultimately reaches the terminal fibres and end-plates distributed among the muscular fibrillæ, leaving the muscle practically without any nerve at all. Muscles in such a state give no response to faradism (for this current acts mainly on the intra-muscular nerves), but as long as any muscular fibres remain they can be made to contract, by virtue of their



own inherent irritability, by the galvanic current slowly interrupted. Sometimes muscles in this condition respond too readily to galvanism; but why this should be so it is difficult to say.

Degenerative re-actions are found in cases of paralysis due to damage to a nerve trunk (as in facial palsy and other forms of nerve injury); as well as in cases of paralysis due to destruction of the motor cells in the spinal cord (anterior polio-myelitis, infantile paralysis, spinal paralysis of adults), and of course in those more extensive lesions of the cord, such as acute myelitis, which involve the motor cells in a common ruin. Degenerative re-actions are also found in extreme forms of lead-palsy. In cases of paraplegia from a local injury, in which the motor cells below the injury retain their functional vitality, the degenerative re-actions are not found, so that the rules may be laid down that:—

1. As long as a muscle is connected by a healthy nerve with healthy motor cells in its nerve-centre, it will not give degenerative re-actions, even though it be absolutely paralyzed to the will. Neither will a muscle in such a condition undergo rapid or extensive wasting.

2. When a muscle is cut off from the influence of its motor cells, by damage to the cells themselves, or to the motor nerve which emanates from them, it gives degenerative re-actions as soon as the degeneration of the nerve to its end is complete; and a muscle in such a state wastes very rapidly.

It must be borne in mind that a muscle may be hopelessly paralyzed, and yet may respond to electricity quite normally.

The irritability of motor nerves may be tested by applying the rheophore exactly over the nerve we wish to test; and all that we have said with regard to the muscles may be equally applied to the nerves.

Electricity is of some use also in testing the sensibility of sensory nerves.

Electricity is of use in the *prognosis* of disease, and it will be convenient to offer here a few remarks on this subject. 1. A correct prognosis must depend upon an exact diagnosis. Thus a muscle or limb is paralyzed, and by the aid of electricity (combined with other indications) we are helped to a knowledge of the exact seat of the paralyzing lesion, whether psychical, cerebral, spinal, or nervous; and on the exactness of this knowledge depends the accuracy of prognosis. 2. Electrical re-actions, when considered in conjunction with the element of time, are often a great aid to prognosis. Thus, let us take an ordinary case of facial paralysis, from a (rheumatic?) thickening of the sheath of the facial nerve after it leaves the stylo-mastoid foramen. The tendency of such a case is to recover completely in ten days or a fortnight, and the degenerative re-actions will be scarcely appreciable. If the degenerative re-actions are well-marked, we may infer that the degeneration of the motor nerve is complete to its very end, and we may feel sure that recovery will be delayed for some weeks. If the degenerative re-actions persist after many months, the chances of ultimate recovery lessen. Directly, however, a muscle which has given degenerative re-action begins to respond to faradism, our prognosis becomes more hopeful. Prognosis must depend upon the exactness of the diagnosis, and our knowledge of the natural history of the disease with which we have to deal. Electricity is a valuable aid to prognosis,

but it is not an infallible touchstone, and if it be used in combination with ignorance, it is almost certain to mislead.

The uses of electricity in *treatment* will be subsequently considered.

G. When a limb is paralyzed, the **state of nutrition** of its tissues must be noted, especially that of the muscles, by feeling them, and by making circular measurements, for which a special apparatus has been invented by Dr. Russell Reynolds. It should also be observed whether there is any local change in **temperature**; or in the characters of the pulse.

H. **Examination directed to the special senses.**—It may be requisite to test the sense of hearing, taste, or smell; but the most important matter coming under this head is examination directed to the eye and to vision. This comprehends:—(i.) *Examination of the position and movements of the eye-balls.* (ii.) *Examination of the pupils*, observing whether both are contracted or dilated, or rapidly alternate from one to the other condition; if they are equal or unequal; and if they act properly under light, and to accommodation. (iii.) *Testing the sight* in various ways, particular attention being paid to the *field of vision* in all directions; and also to the perception of colours. (iv.) *Examination with the ophthalmoscope.*—This instrument has now come to occupy a most important position as a mode of investigation of diseases of the nervous system, and Dr. Hughlings Jackson has specially insisted upon the importance of ophthalmoscopic examination as a routine measure in these affections. For a full account of this subject, and of the various kinds of apparatus employed, with the methods of using them, reference must be made to special treatises, including the valuable work on “Medical Ophthalmoscopy,” by Dr. Gowers. It is, however, by practical demonstration that the use of the ophthalmoscope is best learnt, and considerable personal practice with the instrument is required before it can be satisfactorily employed. In the succeeding remarks a brief description will be given of the morbid appearances which may be presented, the structures to which attention must be directed in the examination being the optic disc, blood-vessels, retina, and choroid. At the outset it must be remarked that the normal amount of vascularity is subject to great variations, and therefore but little importance should be attached to slight alterations in this respect, unless they are changing or unilateral.

*α. Hyperæmia.*—This may be limited to the vessels of the disc or retina, or may involve both sets. It is characterized by more or less increased redness, with enlargement of the vessels and apparent increase in their number, many radiating from the disc, and some appearing to be tortuous or varicose, there being in some cases minute dark red spots, due to little “kinks,” in the vessels. Pulsation in the arteries is often unusually distinct, especially on lightly pressing the eye-balls. Slight cedema of the disc may follow, dimming the edge, and veiling its surface. There may be subjective symptoms of dimness of vision, heaviness about the eyes, flashes of light, or iridic colours. The encephalic conditions with which hyperæmia may be associated are congestion; acute or chronic inflammation, especially meningeal; and tumours. It may remain as such; but is more frequently the first stage of an acute inflammation, especially when due to a tumour. *β. Anæmia.*—This condition may be persistent, as in general anæmia; or transitory, as in vascular spasm. As a rule the disc, retina, and choroid are affected. There is pallor, with emptiness and shrinking of the vessels. It may be attended



with temporary blindness, flashes of light or *muscæ volitantes*, and general weakness of vision. The local causes are vascular spasm and embolism. Anæmia has been noticed in epilepsy and acute uræmia.

γ. *Œdema of the disc*.—Most frequently accompanying other conditions, namely, hyperæmia, ischæmia, but especially neuritis, in rare instances œdema of the disc exists alone. δ. *Ischæmia of the disc*.—*Choked disc* (Allbutt). There is still much doubt and discussion as to the real significance of the condition thus named. By one set of authorities it is regarded as originating in congestion, in consequence of some increased intra-cranial pressure, which obstructs and prevents the return of the blood from the eye through the ophthalmic vein to the cavernous sinus. Von Graefe advanced the view that ischæmia is due to "obstruction at the cavernous sinus, with concurrent action of the sclerotic ring." More recently it has been attributed to the pressure of fluid driven down into the subvaginal space around the optic nerve, and compressing the latter at the terminal *cul-de-sac*, so that the return of venous blood is impeded. This space is continuous with the subarachnoid space around the brain, and any increase of intra-cranial pressure or of subarachnoid fluid causes distension of the sheath around the nerve. Manz has endeavoured to prove this by experiments on animals. On the other hand, several of the highest authorities in ophthalmoscopy deny that this is the mode of origin of ischæmia, and consider that it is really a form of neuritis. Dr. Hughlings Jackson and others maintain that this is set up in a reflex manner, as the result of some irritation in the brain, and this reflex influence has been supposed to be conveyed through the vaso-motor nerves. Schmidt found experimentally that the liquid in the sheath around the optic nerve passes into lymph-spaces in the nerve at the lamina cribrosa, and he suggested that neuritis is set up by the influence, perhaps irritation, of fluid driven in by intra-cranial pressure. Others hold that the so-called ischæmia is always a descending neuritis, the inflammation being propagated directly from the brain along the trunk of the optic nerve. When advanced, the appearances in ischæmia are those of intense congestion and inflammation, with hæmorrhages. The disc is much swollen and prominent, generally rising steeply on one side and sinking gradually on the other, while the margin is obscured by infiltration and excessive vascularity, the latter giving it a mossy look, owing to the great increase in the number of capillaries. The colour may be deep-red, but is often a mixture of dirty grey and red, from the mixing of exudation with distended capillaries and minute extravasations. The nerve-fibres are somewhat swollen, less transparent than in health, so that the papillary region looks more coarsely fibrous. Cell and nuclear proliferation takes place in the connective tissue between the bundles of nerve-fibres and around the vessels. The retina is only altered immediately around the disc, being opaque and its veins being enlarged, sometimes with streaks of exudation along the larger of them. Some nerve-fibres are disintegrated. The trunk of the optic nerve is unaffected. This state frequently exists to a marked degree without any disturbance of central vision. The causes of ischæmia are meningitis, tumours, hydrocephalus, and caries of the sphenoid bone. ε. *Descending optic neuritis*.—This signifies inflammation extending along the optic nerve from within the cranium, the extension probably taking place chiefly along its connective tissue. Hence the optic trunk itself is involved, and the morbid ophthalmoscopic appearances are chiefly confined to the disc, occasionally involving the adjacent retina. The



ophthalmoscopic distinctions from ischæmia are thus described:—The disc is less swollen, and does not present the steep, one-sided elevation; the main trunks of the vessels are chiefly enlarged and tortuous, and there is not the great increase in number of the minute branches and capillaries observed in ischæmia; the colour is less intense and more uniform, with more opacity, and these appearances extend further into the retina; there is often a “woolly” aspect, probably due to œdema. Numerous small hæmorrhages frequently occur, which leave white spots. The intimate changes chiefly affect the connective tissue, which undergoes proliferation, the nerve-fibres being subsequently disintegrated and wasted. A variety has been described as *perineuritis*, in which the outer neurilemma is most affected, the appearances being visible mainly in the margin of the papilla, and extending more widely into the retina.

9. *Chronic optic neuritis*.—Here there is an early stage of redness of the disc, with in some cases hæmorrhages and slight effusions, followed by consecutive atrophy, the vessels gradually contracting and disappearing.

10. *Retinitis*.—Very rarely resulting from cerebral disease, this is characterized at first by hyperæmia of the disc and retina, followed by silvery patches of exudation upon the latter. The entire retina is probably never affected from cerebral disease. The intracranial causes of all the varieties of optic neuritis are meningitis and cerebritis, in whatever way these may have been set up. The inflammation must be contiguous to the nerve, and the latter is more likely to be affected if the morbid process is severe or prolonged. Tumours and other morbid conditions may give rise to neuritis, but only indirectly, by first exciting inflammation of the cerebral structures. Chronic neuritis is said to be connected with abuse of tobacco, general paralysis, and locomotor ataxy. 11. *Atrophy of the disc*.—Two forms of atrophy are recognized, the *simple*, *progressive*, or *primary*; and the *consecutive*, which is secondary to ischæmia or neuritis. Dr. Hughlings Jackson distinguishes between them by the raggedness of the edges, and blurring of the outline in the consecutive form; by the clean-cut even rim, and more brilliant appearance in the primary form. Dr. Allbutt, however, considers that the condition described as simple atrophy often succeeds chronic neuritis, and that the ragged and irregular form is only transitional, gradual changes taking place in the products of inflammation, which are finally entirely removed. True primary atrophy may result from destruction of the fibres in the course of the optic nerve, so as to sever their distal ends from their central attachment, as by pressure of a tumour or inflammatory exudation; disease at the root of the nerve in the centres of vision; progressive sclerosis extending along its trunk; or failure of nutrition from degeneration of arteries or embolism. The ultimate appearances observed in atrophy are that the disc becomes white, glistening, and more or less cupped; the smaller vessels fade away; the connective-tissue is increased; and the nerve elements disappear.

1. There is a peculiar tendency among **malingerers** to sham nervous affections, and it sometimes requires considerable ingenuity to detect the imposture. In any case where anomalous nervous symptoms are complained of, without any objective signs, malingering should be suspected, and the patient should be closely watched, without letting it appear that this is being done. The tests to be applied will, of course, vary in different cases, but as illustrations of such tests may be mentioned the use of chloroform; various methods of detecting shammed fits, as by

putting snuff under the nose, applying heat or cold suddenly, or pressing with the nail under the matrix of the thumb-nail; supporting a supposed paralyzed limb in an extended position, and letting it fall suddenly; pricking unexpectedly a part stated to be anæsthetic, while the patient is not looking; and the use of a strong electric current.

*J.* In all cases of nervous disease, especially cerebral, it is of great importance to examine carefully the **heart** and **vessels**; and also to **test the urine**. It may also be requisite to investigate certain acts, especially micturition. In some cases local temperatures need to be taken and compared. Attention may also be directed here to the red blotches of cutaneous congestion, named *tâches cérébrales* by Trousseau. These may be observed scattered over the skin in certain conditions without any irritation; but in other cases the skin has to be irritated in order to bring them out, by drawing a pencil or the finger-nail along it.

---

## CHAPTER LXIX.

### ON CERTAIN HEAD-SYMPOMS.

#### I. HEADACHE OR CEPHALALGIA.

**ÆTIOLOGY.**—The causes of headache are very numerous, and terms are often prefixed indicating its mode of origin, such as congestive, plethoric, anæmic, organic, nervous or idiopathic, neuralgic, dyspeptic, or bilious. The *pathological conditions* which may give rise to this symptom are :—

1. Disturbance in connection with the *cerebral circulation*, including congestion, especially that due to general plethora, increased cardiac action, vaso-motor paralysis of the cerebral vessels (by inducing which many remote causes excite headache), or venous obstruction; deficiency of blood; or an abnormal condition of this fluid, particularly when it is hydræmic, imperfectly aerated, or impregnated with various deleterious ingredients.
2. *Injuries or organic diseases of the brain or its membranes*, such as meningitis, cerebritis, abscess, tumour, softening.
3. *Disease of the cranial bones or their sinuses*; or of the structures forming the *scalp*.
4. *Neuralgia*, affecting the nerves either within or outside the skull.

The chief *remote* causes which may excite headache, by giving rise to certain of the conditions mentioned above, include all those which induce general plethora or anæmia; cardiac or pulmonary diseases, or severe fits of coughing; affections of the stomach, bowels, and liver; renal and cutaneous diseases; fevers and acute inflammations; ague or mere malarial influence; gout and rheumatism; uterine disorders; hysteria; various causes which exhaust or depress the nervous and vital energy, such as sedentary habits, deficient ventilation, over-work in confined rooms, undue mental exertion, depressing emotions, exposure to the hot sun, particularly when fatigued, loss of sleep, over-lactation, venereal excesses and masturbation; and abuse of coffee, tea, alcohol, tobacco, opium and various other drugs which affect the brain. Some individuals are much more subject to headache than others, and especially delicate females of a nervous temperament.

**CHARACTERS.**—The points concerning which it may be necessary to inquire with reference to headache are:—*a.* Its mode of onset; and if it is brought on by any obvious cause. *b.* Whether it is constant, or only felt at intervals. *c.* Its exact situation, whether general, unilateral, frontal, occipital, over the vertex, or localized to a particular spot; and also if it seems to be superficial or deep. *d.* Its characters, the chief varieties being heavy, dull, aching; throbbing; shooting or darting; boring; oppressive; accompanied with a sense of fulness, as if the head were going to burst; or with a feeling of great heat. *e.* Its intensity, and if this is variable or not. *f.* The effects of movements and change of posture, especially of moving or hanging down the head; of muscular exertion; of coughing; of light or sound; of firm pressure over the whole head or any part of it; of taking food or stimulants; or of pressure on the carotid vessels. *g.* If it is accompanied with soreness and tenderness, either over the scalp generally, or over any particular spot.

## II. VERTIGO—GIDDINESS—MENIÈRE'S DISEASE.

**SYMPTOMS.**—There are two distinct forms of giddiness, so far as the sensations of the patient are concerned. In the one the feeling is that of confusion and instability, or of motion of the body, and as if it were impelled in different directions, accompanied with a tendency to fall and unsteadiness of gait; in the other extraneous objects appear to move and to assume abnormal positions; both may, however, be combined. The sensation is often described as “dizziness” or “swimmings.” It varies much in intensity, from an uncomfortable feeling of oscillation, to a condition in which the patient reels or staggers and has to grasp some object to prevent him from falling, or in which he actually falls. The feeling may be momentary, constant, or paroxysmal. In many cases it is only or chiefly felt on movement or in certain positions, especially on bending the head downwards. It may be worse in the sitting, standing, or recumbent posture in different cases; while closing the eyes, or staring fixedly for a time at an object, has often a marked influence on vertigo, either aggravating or relieving the sensation. Occasionally an attack comes on during sleep, awakening the patient. Commonly other head-symptoms are present; as well as disturbances of the special senses.

**ÆTIOLOGY AND PATHOLOGY.**—The experimental researches and clinical observations which have been made of late years show that vertigo is not such a simple phenomenon as was formerly supposed. The normal equilibrium of the body is maintained by a somewhat complicated mechanism, consisting of (1) an afferent or sensory apparatus, tactile, visual, and auditory, which conducts impressions to (2) a co-ordinating centre, believed to be the cerebellum, and this governs (3) the motor apparatus, efferent impulses being transmitted to the muscles, especially those of the head, neck, and spine. Any portion of this mechanism may be deranged, so as to lead to vertigo, the power of adjustment being disturbed. The term should, strictly speaking, be confined to the sensation which is felt, and should not include the movements which may be associated with it. It has been defined as “the consciousness of disturbed locomotor co-ordination—a rudimentary disorder of co-ordination of locomotive movements” (Hughlings Jackson). The causes of vertigo are very numerous, and most of them act through the circulation, by influencing the quantity or quality of the blood. In many cases the central



circulation is affected. Some causes act in a reflex manner. They may be divided into *centric* and *eccentric*, and among the most important are injury to, or organic disease of the brain or its membranes; degenerative changes in the cerebral vessels; certain functional nervous disorders, as epilepsy and migraine; certain movements, such as swinging or waltzing, or the movements of a ship; febrile conditions; exposure to paludal and other emanations; tobacco-smoking; abuse of alcohol or narcotics, as well as excess of certain drugs, such as quinine, salicine, or salicylic acid; renal disease; gout; suppression of chronic cutaneous diseases, hæmorrhages, or discharges; anæmia; nervous exhaustion and depression from excessive mental and bodily work, especially if combined with close confinement, anxiety and worry, excitement, and poor or irregular living; digestive derangements; organic or functional diseases of the heart affecting the circulation, especially a weak or fatty heart; disease of nerve-trunks or of the spinal cord, interfering with conduction from the periphery to the brain; and disorders of the special senses. With regard to the *special senses*, these have an important causative relation to giddiness. *Auditory vertigo* will be separately discussed. With reference to *vision*, vertigo may be associated with strabismus, nystagmus, the effect of a sudden strong light, disorders of sight, or actual disease of the eye. The *tactile apparatus* may also be diseased; or the nerve-trunks or spinal cord may be so affected that they do not conduct impressions properly. Giddiness has, moreover, been attributed to unpleasant and powerful *odours*. The disorders of the special senses are supposed to cause vertigo, by giving to the co-ordinating centre either no information at all, or wrong information. Ferrier states that loss or perversion of visual or tactile sensations may be compensated for, if the two remaining sensory processes continue intact, but nothing compensates for entire loss of labyrinthine impressions.

VARIETIES.—Numerous varieties of vertigo have been named, and different classifications have been made by different writers, mainly founded on an ætiological basis. It will only be practicable here to offer a few remarks about two particular forms; and to consider in some detail auditory vertigo.

*Gastric vertigo* is described as occurring either in severe acute paroxysms, coming on quite suddenly, being often due to an undigested meal, and sometimes assuming a grave character, accompanied almost with loss of consciousness; or as a milder chronic complaint, either constant or occurring in frequent attacks. Dyspeptic symptoms are not prominent in most of these cases. The vertigo is of both kinds, but consists chiefly of apparent movement of external objects. The chronic form is rendered worse by fasting, and is often relieved by a moderate meal or by a little stimulant, as well as by shutting the eyes, or gazing fixedly at some object. Gastric vertigo is often associated with slight dyspeptic complaints, but there may be well-marked organic disease. It is not uncommonly accompanied with various other symptoms; and an attack may be brought on by very slight causes, or without any obvious cause. In many instances relief is afforded by the recumbent posture. Dr. Ramskill describes what he terms *essential vertigo*, which is observed mostly in persons about 30 years of age, who do not complain of any other symptoms, but in whom there are signs of a weak heart, and of a dilated right ventricle. He states that it is not materially improved by remedies, unless these are accompanied by rest and freedom from anxieties of every kind.

**Auditory Vertigo—Menière's disease.**—The relation of the semicircular canals to equilibration has been proved to be of great importance; and it has been shown that definite locomotive disturbance follows injury to each canal. The sensory impressions produced in these canals are associated with varying tension of the endolymph, which affects the vestibular branch of the auditory nerve. This nerve is closely related with the pneumogastric at its origin in the medulla oblongata; and filaments also pass from the inferior cervical ganglion along the vertebral artery, from which the labyrinth receives its blood-supply. Hence the frequent association of vertigo with disorders of the stomach, heart, and other organs.

Some of the causes of vertigo already mentioned produce their effects partly by influencing the labyrinthine tension. This may be modified by changes in the position of the head; variations in the tension in the labyrinthine vessels; differences of pressure in the tympanic cavity; or actual disease of the ear. Certain cases in which vertigo is associated with perversion or abeyance of the labyrinthine function are grouped under the term *Menière's disease*; and, according to the strict definition of this class of cases, there is always a simultaneous affection of the semicircular canal and cochlea, as indicated by deafness, tinnitus aurium, and vertigo. Auditory vertigo may be induced by syringing the ears, especially when the membrana tympani is perforated. It may also be caused by diseases of the labyrinth itself; or by conditions of other parts of the ear which affect this portion indirectly, such as accumulation of wax or a foreign body in the auditory meatus, tympanic disease, obstruction of the eustachian tube, or spasm or paralysis of the small muscles. The labyrinthine disease may be either irritative or destructive, and the effects as regards the tendency to movement on the part of the patient will be exactly opposite in the two cases.

Menière's disease is characterized by attacks of giddiness associated with other symptoms, of variable duration and intensity. The attack begins with a loud noise in one ear, or an exaggeration of habitual tinnitus, variable in its character. The feeling of giddiness quickly follows, and it may be almost or quite simultaneous. It is usually very marked, and accompanied with a tendency to certain movements, or such movements actually take place; sometimes the patient suddenly falls, or is thrown more or less violently to the ground, generally either forwards or to one side. In rare instances there is loss of consciousness, or there may be more or less mental obscurity, but in the great majority of cases the mind is unaffected. Frequently nausea or vomiting and faintness occur, the face being pale, and the skin cold and clammy. In some instances oscillatory movements of the eyes are observed. The attack gradually passes off, but vomiting and giddiness may persist for several hours or days, brought on or aggravated by rising from the recumbent posture. More or less deafness and tinnitus are often persistent; and there may be a feeling of constant vertigo, easily increased by digestive disorders. In severe cases the attacks are liable to become more and more frequent, and ultimately permanent distressing vertigo is established, with paroxysmal exacerbations. Even in such cases, however, ultimate recovery may take place, either when complete deafness supervenes, or from the effects of appropriate treatment. On the other hand, it is said that death has occurred, no cause having been found except disease of the semicircular canals. Menière's disease may simulate epilepsy or an apoplectic attack.

## TREATMENT.

In order to relieve either of the head-symptoms just considered, a point of primary and essential importance is to find out its *cause*, as treatment has in most cases to be directed against this, and must be varied accordingly. In persons subject to headache or vertigo, attention to their diet, occupation, habits, and mode of life is frequently most essential. The various organs of the body must also be looked to, particularly the special senses, digestive apparatus, vascular system, and kidneys. In many cases a course of *vegetable* or *mineral tonics* is highly serviceable; and arsenic proves very beneficial sometimes, as well as quinine in full doses. When headache is merely temporary, associated with depressed nervous energy, *stimulants* will often relieve it, such as a little weak brandy and water, spirits of ammonia or chloroform, or a cup of strong coffee. Among *local* remedies which may be useful under various circumstances may be mentioned the use of cold, warm, or anodyne applications to the head; cold or warm affusion; sustained pressure around the head; the application of sinapisms or blisters to the nape of the neck or to some other part; and local removal of blood. Attention to posture may also be of importance in relieving headache or giddiness.

---

## CHAPTER LXX.

## DISORDERS OF CONSCIOUSNESS.

CONSCIOUSNESS may be more or less exalted, the mental faculties being unusually keen and active; impaired in various degrees to absolute insensibility; or perverted in different ways. It will be impossible to discuss all the phenomena which may result from these disorders, but some of the most striking will now be considered.

## I. DELIRIUM.

This symptom implies an acute and temporary disorder of the mental faculties, which generally reveals itself in the language or actions of the patient. It varies in degree, from slight wandering and incoherence, to the most complete and thorough derangement of the mental faculties. Frequently the patient has a fixed delusion. When the delirium is but slight, the patient can often be roused temporarily, so as to become tolerably coherent. It may be constant, but commonly tends to be worse by night, or may only come on at this time. In character the delirium may be mild and quiet; more or less wild and violent, the patient shouting furiously, or attempting to get out of bed, or to injure those around; talkative and cheerful; surly; suspicious; or low and muttering, when it is often attended with picking at the bed-clothes or *carphology*. In many cases delirium is associated with more or less stupor.



**ÆTIOLOGY.**—Delirium may arise either from excitement or depression of the cerebral functions, being accordingly either *active* or *passive*. The grey matter covering the cerebral hemispheres is specially affected when this symptom occurs. The causes of delirium are:—1. *Organic diseases* of the brain or its membranes, especially meningitis. 2. *Reflex disturbance* in connection with remote organs, such as the stomach, bowels, or uterus, particularly if attended with severe pain. 3. A poisoned condition of the *blood*, as in delirium tremens; acute febrile and inflammatory diseases; imperfect aëration of the blood; poisoning by belladonna and other substances. 4. *Nervous exhaustion*, as in delirium tremens partly; after excessive venery; or from undue mental exertion. 5. *Acute mania*. Some individuals are much more liable to delirium than others, particularly children and nervous persons.

**TREATMENT.**—In the treatment of delirium, if it is of the active kind and attended with much vascular excitement, the measures which may be needed are to shave the head; to apply cold assiduously; to use cold affusion; or to remove blood. In other cases the aim of treatment should be to endeavour to procure sleep, by means of some *narcotic*. Opium, which is sometimes usefully combined with tartar emetic or with some stimulant; hydrate of chloral; or full doses of bromide of potassium, often prove most serviceable. When delirium is of the low type, it is commonly an indication for the free use of *stimulants*. Warm affusion is frequently of much value in these cases. An important object to be always borne in mind is the removal from the system of any deleterious materials which may be causing delirium. Of course due precautions must be taken, if necessary, to prevent the patient from injuring himself or others. All external sources of disturbance must be removed, and the patient kept as quiet as possible.

## II. INSENSIBILITY—STUPOR—COMA.

These terms imply various degrees of suspension of consciousness, depending immediately upon some condition of the brain, complete coma being attended with absolute loss of sensation and perception, of the power of expression, and of voluntary motion; in short, with total abolition of all the cerebral functions. In investigating this symptom it is important to take into consideration:—1. Its mode of onset, whether sudden or gradual, and if it is due to any obvious cause. 2. Its degree, noting if any signs of sensibility can be elicited, as by touching the conjunctiva; and also whether the patient can be roused temporarily or permanently. 3. Whether the insensibility is transitory or persistent.

**ÆTIOLOGY.**—Pathologically loss of consciousness may result from injury to, or compression of the brain-substance; from extreme cerebral congestion or anæmia; or from the circulation through the brain of poisoned blood, or of blood which is inadequate to maintain its functions. The causes of insensibility are very numerous, and it will be expedient to give a complete list of them here, it being borne in mind, however, that coma implies loss of sensibility directly due to some cerebral disorder, and must be distinguished from asphyxia, syncope, and shock. They may be arranged thus:—1. *Local injury* to the head and its consequences, such as cerebral concussion; fracture of the skull; or compression of the brain. 2. *General shock to the system*, from injury; rupture of an internal organ; severe mental emotion; or any other cause.

3. Certain *functional nervous disorders*, namely, epilepsy, hysteria, convulsions. 4. *Morbid conditions of the brain or its membranes*, especially congestion; hæmorrhage; effusion in connection with the membranes or ventricles; cerebritis and abscess; embolism or thrombosis; chronic softening; and some cases of tumour. 5. *Blood-poisoning* from morbid conditions within the system, as uræmia, diabetes, certain cases of jaundice, low fevers. 6. *Introduction of poisons from without*, especially alcohol, opium and other narcotics, or prussic acid; and also the inhalation of certain gases and vapours, such as carbonic oxide or anhydride, hydric sulphide, chloroform, or ether. 7. *Syncope* from any cause. 8. Conditions inducing *asphyxia*. 9. As special forms of unconsciousness may be mentioned that which follows prolonged *exposure to cold*; *sunstroke*; a stroke of *lightning*; or *starvation*. 10. It must not be forgotten that sudden insensibility is a favourite form of *malingering*.

It will be convenient in this connection to make a few observations with regard to the term *apoplexy*. Originally this word merely implied an attack of sudden coma without convulsions, corresponding to what is now called an *apoplectic seizure*, *fit*, or *stroke*; such a seizure, however, was found to be most commonly due to cerebral hæmorrhage, and hence apoplexy came to be employed as indicative of this particular pathological condition. Subsequently the meaning of the word was extended so as to denote hæmorrhage into any organ, for example, pulmonary apoplexy. Strictly this use of the term is quite incorrect, and it is highly important to bear in mind that apoplexy and cerebral hæmorrhage are not synonymous, for the former may be due to other causes, and the latter does not always give rise to an apoplectic seizure. The comatose state characteristic of apoplexy is usually accompanied by other phenomena, such as an alteration in the colour of the face; slow, laboured, or stertorous breathing; abnormal states of the pupils; changes in the pulse; or paralysis. These are extremely variable and inconstant, however, and therefore cannot properly enter into its clinical definition.

The ordinary causes of an apoplectic seizure are:—1. *Cerebral congestion—Congestive apoplexy*. 2. *Cerebral or meningeal hæmorrhage—Sanguineous apoplexy*. 3. Sudden *anæmia of the brain*, due to embolism or thrombosis of a main vessel; cardiac failure, especially from fatty disease; or probably vaso-motor disturbance, leading to spasmodic contraction of the arteries. Rarely an apoplectiform attack is associated with:—4. *Uræmia* and other forms of *blood-poisoning*. 5. *Sun-stroke*. 6. *Organic affections of the brain or its membranes*, such as meningitis, abscess, chronic softening, tumours. 7. It is said, sudden *serous effusion into the ventricles—Serous apoplexy*. The last-mentioned cause is, however, very doubtful, and the cases in which it is supposed to have occurred were probably those either of uræmic poisoning or of cerebral atrophy, though it must be added that some authorities believe that uræmia may lead to cerebral symptoms by causing rapid effusion of serum. 8. In extremely rare instances a fatal apoplectic attack has occurred where no morbid condition whatever could be detected at the *post-mortem* examination—*Simple apoplexy*. The immediate condition of the brain upon which an apoplectic seizure depends is a matter of dispute. Probably it may be due to a want of a proper supply of arterial blood, whether the result of interference with its entrance, of venous engorgement, or of a poisoned condition of the blood; of compression or actual destruction of the nerve-elements of the brain; or of shock.

**TREATMENT.**—The measures to be adopted when a person is insensible differ so materially according to the cause of this condition, that no uniform plan of treatment can be laid down. A few general hints may, however, be given regarding the management of the comatose state. The patient should be placed comfortably in the recumbent posture, with the head a little raised, all articles of clothing about the neck and chest being loosened, and plenty of fresh air being admitted. If it is known or suspected that the coma is due to poison, or even if there is much doubt as to the cause of this condition, there ought to be no hesitation about using the stomach-pump, as this instrument does no harm if properly employed, and may prove most serviceable. If the insensibility depends upon blood-poisoning, as from uræmia, means for promoting elimination of the deleterious agent, particularly by acting upon the skin, are highly valuable. In cases due to cerebral lesion it is well not to interfere too actively at the outset. The chief measures which it may be necessary to have recourse to, in order to rouse the patient, are shaking and calling loudly; dashing cold water over the face and chest, or cold affusion: the application of sinapisms to the nape of the neck, and to various other parts of the body; the use of electricity; the administration of *stimulants*, especially by enemata; and artificial respiration. In certain cases it may be requisite to remove blood locally or by venesection. It is important in cases of prolonged unconsciousness to see that the limbs are kept warm; that the bladder and bowels are properly evacuated; and that the system is maintained by adequate nourishment, which may be administered by enemata.

---

## CHAPTER LXXI.

### DISORDERS AFFECTING SLEEP.

THE disorders in connection with sleep which may be met with are chiefly of three kinds, namely:—1. *Somnolence* or undue sleepiness. 2. *Insomnia* or sleeplessness; or where the sleep is restless and disturbed. 3. *Somnambulism* and *somniloquism*, or sleep-walking and sleep-talking, with allied states. Each of these requires brief consideration.

1. **Somnolence.**—This implies either that there is an increased disposition to sleep; or a condition of profound sleep, which may last for considerable periods, and from which it is very difficult or even impossible to rouse the individual, this condition culminating in a state of *trance*. Abnormal sleepiness or drowsiness is mainly observed under the following circumstances:—*a.* In certain subjects who are naturally of a *lethargic temperament*, and who will fall asleep at any time if allowed to remain quiet. *b.* As a result of the effects of considerable *external heat* or *cold* upon the general system. *c.* In consequence of *over-eating* and in some cases of *dyspepsia*. *d.* From *blood-poisoning*, in connection with renal disease; the advanced stage of fevers; some cases of jaundice; indulgence in excess of alcohol; or the introduction of narcotizing agents into the system. *e.* Owing to *imperfect aëration of the blood*, as, for instance, from being in over-crowded and badly ven-



tilated rooms; or as the result of diseases interfering with the respiratory process. *f.* In connection either with a *plethoric* or an *anæmic* state of the system. *g.* From *imperfect nutrition of the brain-substance*, such as that due to disease of its vessels, when the drowsy condition may be premonitory of apoplexy. *h.* In some cases of *disease of the brain or its membranes*. *i.* As the result of *starvation*.

Remarkable cases of prolonged sleep have been occasionally observed, having no evident cause. Others are associated with hysteria, or with marked anæmia. Some individuals are able, after a long period of mental labour with deficient sleep, to indulge in sleep of considerable duration, and thus to make up for that which they have lost.

**2. Insomnia.**—This is often a serious condition, and one which gives much trouble to the practitioner. It may be that the patient feels no inclination to sleep; or that the desire for repose is experienced, and may even be urgent, but there is a dread of going to sleep; or slumber is very restless and much disturbed, perhaps only uneasy dozes of short duration being obtained, from which the patient wakes up in a state of agitation or terror. Rest is often interfered with in consequence of unpleasant dreams; or it may be prevented by bodily or mental suffering, cough, or other causes. The effects of prolonged want of sleep are very grave; it is a prominent cause of insanity, while it often gives rise to great distress in cases which come under observation in ordinary practice. In times past forcible prevention of sleep was resorted to as a means of torture. At the same time it may be remarked that under certain circumstances many individuals can do with very little sleep for a considerable period.

The most important conditions with which insomnia may be associated are as follows:—*a.* *Insanity*, of which sleeplessness is also often a marked premonitory symptom. *b.* A state of *cerebral excitement* or *exhaustion*, or of *mental disquietude*, resulting from undue intellectual effort or excessive study, especially if sleep has been neglected; mental anxiety or worry in connection with business or other matters; exciting passions; or other causes. *c.* *Acute febrile diseases*, particularly at their early stage. *d.* *Dyspepsia* in a considerable number of cases. *e.* *Chronic alcoholism* and *delirium tremens*. *f.* After taking strong tea or coffee. *g.* Conditions accompanied with great bodily pain, or other forms of suffering. *h.* Some cases of *disease of the brain or its membranes*, especially meningitis in its early stage. *i.* Certain peculiar affections of the *nervous system*, such as tetanus or hydrophobia. *j.* *Disease of the heart*, in which want of sleep is often a serious symptom; and also *disease of the great vessels* occasionally. *k.* *Abnormal blood-conditions*, such as *æmia* in some cases, gout, or sometimes the presence of bile in the blood. *l.* *Pregnancy*, and the condition following parturition, especially in nervous and excitable women.

**3. Somnambulism—Somniloquism.**—These conditions have been regarded as being due to an incomplete sleep or partial waking, but it is more probable that they are associated with a state of abnormally profound and heavy sleep. In this state dreams exercise an unusual influence, and excite motor acts of various kinds, of which somnambulism is the most remarkable. When an individual is in either of these conditions, he is perfectly unconscious of his actions, knowing nothing about them on waking from sleep, and it is usually difficult to rouse him fully. Somnambulists will go to the most dangerous places, and perform strange and complicated acts; they may also sleep for very

prolonged periods. Their general health is often quite satisfactory. These disorders of sleep occasionally assume a periodic character.

Somnambulism and allied states usually commence during youth or at puberty. They generally originate from some definite cause, such as overloading the stomach, violent mental emotion, or over-study, but once established they may continue independently of any such exciting cause. Occasionally hereditary influence has been traced. Sleeping on soft luxurious beds, and with the head low, may act as predisposing causes of these conditions.

TREATMENT.—In the management of cases in which any disorder affecting sleep occurs, the first object in treatment must be to endeavour to find out its *cause*, and, if possible, to remove or counteract this. Regulation of the diet, especially of meals taken late in the day, and of the general habits; avoidance of an undue quantity of tea, coffee, alcohol, &c.; the taking of a proper amount of exercise daily; avoidance of excessive mental labour, excitement, or worry; and attention to the conditions of the bed-room and bed, will often prove of much service. The apartment must be properly ventilated, and the bed should have a firm mattress and pillows, without too many bed-clothes, the head being well-raised. Then the condition of the blood must be improved, if necessary, and any organ attended to a diseased condition of which may be the cause of disturbed sleep, as well as any special disease upon which this may depend. Treatment directed to the alimentary canal is often of the greatest service. In cases of insomnia due to mental causes, entire cessation from occupation, and a change of air and scene, are frequently of the highest value. Pain and other causes which may prevent sleep must be treated by appropriate remedies. The direct measures employed for procuring sleep are the administration of *sedatives*, *anodynes*, or *narcotics*, either internally, by enema or suppository, or by subcutaneous injection, such as opium or morphia, hydrate of chloral, bromide of potassium, cannabis indica, hyoscyamus, conium, hop, belladonna, or nepenthe; the use of *local applications* to the head, for instance, a wet bandage, cold or warm douching, or the ice-bag; and the employment of mesmerism, braidism, and similar agencies. In some cases a glass of stout, or wine-negus, or of some spirit-and-water, taken just before going to bed, is decidedly useful for procuring sleep. Fixing the eyes steadily upon some point, counting to a hundred, being read to, and various other devices are resorted to with the view of obviating sleeplessness, and they sometimes succeed.

In cases of somnambulism and similar conditions, it may be desirable to try to break off the habit by waking the patient once or twice during the night. Somnambulists must, however, on no account be suddenly awakened when they are in the act of walking, even apart from their being in dangerous places, as this may cause a fright which may lead to very serious consequences.

---

## CHAPTER LXXII.

## MOTOR DISORDERS.

A SUMMARY of the various disorders affecting motion has already been given, when indicating the clinical characters of nervous diseases. In the present chapter it is intended mainly to discuss the chief phenomena indicating undue muscular irritability; and the principal forms of paralysis. It must be mentioned, however, that these two classes of phenomena are not uncommonly associated in various degrees and forms in the same case, though they more frequently occur separately. A few remarks will also be offered concerning superficial and deep reflexes.

## I. SPASMS—CONVULSIONS—ECLAMPSIA.

Spasms may be defined as involuntary contractions of the muscles, varying widely as regards their intensity and extent, and being either intermittent and interrupted, with intervals of relaxation, the movements being often of a jerky character—*clonic spasms*; or more or less continuous and persistent—*tonic spasms*, these in their extreme form culminating in permanent *rigidity*. If spasms are accompanied with severe pain, they constitute the condition known as *cramp*. The violent tonic contractions observed in lock-jaw and strychnine-poisoning are named *tetanic*. There is not, however, any marked line of demarcation between these different forms of motor disorder.

Spasm of muscles may be a local phenomenon, affecting, for instance, those supplied by a particular nerve, or even only a single muscle, and the spasm may then be either clonic or tonic. Thus it may be met with in connection with the elevator of the upper eyelid, causing this part to be persistently drawn up; or occasionally it affects the internal or external rectus of the eyes, leading to forms of strabismus. *Nystagmus* has already been mentioned as a symptom due to chronic spasm or to inco-ordination of the eyeball muscles. One form of *wry-neck* is also of spasmodic origin. In *histrionic spasm* the face is affected. In *writer's cramp* and allied affections muscles which are much used and over-taxed become the seat of spasmodic movements when brought into action. Internal muscular organs are also liable to spasm and cramp. Local spasms may sometimes be excited or arrested by pressing certain parts.

The spasmodic movements implied by the term *convulsions* vary considerably in their severity and extent; and also as to the parts of the body which they involve. Thus they may be slight and localized; unilateral; or more or less general. Some writers include under convulsions all forms of motor disorder in which there are unusual involuntary movements, such as fibrillar trembling of muscles, muscular flickerings, various kinds of tremor, and choreic movements. Ordinarily, however, the term implies more or less marked spasmodic movements, and these may be so violent as actually to rupture the muscles. *Eclampsia* is a word which is now often used to characterize all forms



of powerful convulsions of a more or less epileptiform type, whatever their cause may be. According to the extent and localization of the convulsions, we are frequently able to refer their origin to some special part of the nervous system, as will be hereafter pointed out. Not uncommonly convulsions are accompanied or followed by partial or complete loss of consciousness. They are of much importance in children, in whom a series of *convulsive fits* are liable to occur from very slight causes—*infantile convulsions*. They are frequently preceded by premonitory indications of nervous disturbance, such as twitchings, grinding of the teeth, restlessness or peevishness, which in children should always be looked upon as warnings. It is unnecessary to describe the distortion of the features, and the very varied movements of the limbs and body which may result from convulsive spasms, these being usually a combination of the clonic and tonic varieties, the former predominating. The chief dangers in connection with convulsions arise from implication of the respiratory muscles or glottis, leading to grave interference with breathing; from obstruction to the return of blood from the brain; and from the exhaustion which the extreme violence or frequent repetition of the fits may cause, especially if they prevent sleep for a long period. Serious sequelæ may follow as direct consequences of convulsions, such as hemiplegia; strabismus; loss of sight, smell, or hearing; defect of speech; or impairment of the mental faculties.

**ÆTIOLOGY.**—All forms of motor disorder now under consideration are referable to some kind of irritation, acting upon some portion or other of the nervous system. Convulsions have been immediately attributed to “an abnormal discharge of unstable grey matter” (Hughlings Jackson). They originate in some irritation or *discharging lesion*, either direct or indirect, affecting this grey matter. The main causes may be arranged thus:—1. **Centric.** *a. Injuries* to the head, especially fracture of the skull, with irritation of the grey matter by spicula of bone. *b. Various organic diseases* of the brain and cord or their membranes, namely, all forms of meningitis, hydrocephalus, cerebral hæmorrhage, rupture of an aneurism, embolism, softening, tumour. *c. Idiopathic, dynamic, or essential.* Here the convulsions are independent of any obvious organic mischief, but are supposed to result from some vascular or nutritive disturbance in the brain, as in some cases of epilepsy, hysteria, or the convulsions induced by strong emotions. *d. Circulation of abnormal blood* through the central nervous system, as exemplified by the convulsions which in children sometimes usher in, or occur during the course of acute specific fevers or inflammatory diseases; uræmic convulsions; and those which may be associated with imperfect aëration of the blood, or, it is said, with rheumatic fever, jaundice, syphilis, tuberculosis, and rickets. In the two conditions last-mentioned, however, the nervous system is probably highly susceptible, and convulsive movements may be excited by very slight reflex disturbance. 2. **Eccentric, reflex, or sympathetic.** In this class of cases the convulsions are due to some *reflex irritation*, particularly in connection with dentition; digestive disorders; intestinal worms; or the passage of a gall-stone or a renal calculus. Occasionally they result from direct irritation of some local nerve; the pricking of a pin in the clothes of a child; the application of a blister; or a burn of the skin. *Puerperal* convulsions are either uræmic or reflex in their origin.

The most favourable periods of life for the occurrence of general convulsions, independent of organic disease, are childhood, especially during

the period of dentition; puberty; when cutting the wisdom-teeth; and at the change of life. In children the ordinary causes are reflex irritation; the onset of some acute fever or inflammation; tubercular meningitis; or the presence of some chronic constitutional illness. Later in life they are most frequently associated with epilepsy; with organic affections of the nerve-centres; or with uræmia.

TREATMENT.—In treating spasmodic movements, if they should call for special interference, and especially if they are of the nature of general convulsions, the indications are:—1. To look for any *reflex irritation*, and remove this if possible, particular attention being paid in the case of children to the teeth and alimentary canal, the gums being lanced, or an *aperient* or *emetic* given, if required; at the same time regulating the feeding. It is also well to examine the clothes for any source of irritation. 2. To treat any *disease* with which the convulsions may be associated, such as rickets, tuberculosis, epilepsy, central organic disease, or blood-poisoning. 3. To mitigate or check the *spasmodic movements*. During a paroxysm of convulsions the recumbent posture; freedom from every disturbance; relaxation of the clothing about the neck and chest; and a free current of cool fresh air, are needed. It is not advisable to restrain the movements, except in so far as to prevent injury to the patient. Water may be sprinkled over the face and chest. If the convulsions continue, a warm bath containing mustard; the application of ice to the head; warm pediluvia; cold or warm affusion; and the application of sinapisms to the nape of the neck, epigastrium, or extremities, are the chief remedial measures which may be employed. Many practitioners resort at once to the application of leeches to the temples or back of the neck, or to venesection, especially in the case of robust children; but in most cases this is needless or injurious, and as a rule removal of blood is only indicated when there are signs of serious interference with the respiratory functions. The principal medicinal remedies available are *narcotics* and *antispasmodics*, especially bromide of potassium; hyoscyamus in full doses; opium; hydrate of chloral; chloroform by inhalation; and assafoetida by enema. Of course most of these drugs need due caution in their administration. It is of the greatest importance to endeavour to procure sleep if this is seriously interfered with, particularly should there be much exhaustion. The milder forms of spasm and cramp may often be considerably mitigated by friction, dry heat, judicious restraint, and other measures. 4. To treat the *consequences* of convulsions. The chief dangers are from suffocation and exhaustion. To obviate the former, removal of blood and artificial respiration are indicated. To prevent or counteract exhaustion, it is extremely important to administer abundant liquid nourishment, especially in the case of weakly or badly-fed children, and if it cannot be taken by the mouth, nutrient enemata must be employed. *Alcoholic stimulants* are also most useful in many cases, being sometimes required in considerable quantities, along with *medicinal stimulants*, such as ammonia, ether, camphor, or musk. The administration of food and stimulants often promotes sleep most efficiently.

## II. MOTOR PARALYSIS OR PALSY—PARESIS.

Motor paralysis is a symptom of the greatest importance in nervous diseases. Some of the main points to be noted with regard to it have already been indicated when discussing the physical examination of the nervous system, and it is only needful further to remark, that particular attention must be paid to its mode of onset, whether sudden or gradual; its exact extent and distribution; its degree; whether it is permanent or temporary, constant or variable, or influenced materially by volition, emotion, or other causes; as well as to its subsequent progress, observing whether the paralysis tends to become worse, to improve, or to invade other muscles; and if any additional phenomena arise in the affected part, especially involuntary reflex movements, clonic or tonic spasms, rigidity, or permanent flexion of joints. The tendency in many forms of persistent paralysis is towards imperfect nutrition of the tissues from mere want of exercise, as evidenced by softness and flabbiness of the muscles and other structures, wasting and diminution in the circumference of the limb, with dryness and scurfiness of the skin; and to feebleness of the circulation, the local pulse becoming small and weak, the skin pale or blue and congested, and the temperature lowered, while the affected part is much more influenced by the temperature of the surrounding medium than in health, and œdema supervenes in some instances. Under certain conditions serious trophic lesions occur with great rapidity, such as acute bed-sores, as will be hereafter pointed out. Occasionally an extraordinary growth of hair is observed over a paralyzed part.

There are certain important varieties of paralysis, designated according to its mode of distribution in the body, some of which it will now be requisite to consider briefly. They include:—1. **General paralysis**, which does not necessarily imply that every muscle in the body is affected, but the term is applied to that condition in which both arms and legs are paralyzed, along with more or less of the trunk. 2. **Hemiplegia** or unilateral paralysis. 3. **Paraplegia** or paralysis of the lower extremities; the lower part of the trunk, with the bladder and rectum, being usually involved at the same time. 4. **Disseminated or irregular paralysis**. 5. **Local**, where the palsy is limited to one limb or a part of it; to certain muscles which are supplied by a special nerve, as the facial, or which are associated in their action for a particular function; or even to a single muscle.

1. **General Paralysis** is met with:—(i.) Rarely in cerebral diseases, namely, temporarily in congestion; in hæmorrhage into certain parts, as into the pons, both ventricles, or the meninges; and in some cases of tumour, extensive softening, or meningitis. (ii.) In connection with disease or injury of the upper part of the spinal cord. In this case of course the face is not affected, and the condition has been termed *cervical paraplegia*. Usually both motion and sensation are then involved, and the upper limbs are more affected than the lower. (iii.) In the early stage of essential paralysis of children and allied conditions. (iv.) In extreme cases of diphtheritic paralysis. (v.) In certain cases of rapid and almost universal paralysis, of doubtful pathology, but which Dr. Buzzard\* thinks is dependent upon syphilis. (vi.) In extreme progres-

\* See "Diseases of the Nervous System," page 301.



sive muscular atrophy. (vii.) In *general paralysis of the insane*. In the disease last-mentioned the paralysis begins in the tongue, as shown by impaired articulation, with tremulous movements of the organ, and a difficulty in its protrusion. Next the muscles of the face quiver, especially those of the lips; while the pupils are often unequal. Then follows weakness of the limbs, with unsteadiness of gait, the patient stumbling and staggering on turning round suddenly, the ability to perform various ordinary actions being also impaired. When the muscles are put into action they are tremulous. More or less speedily, and usually by interrupted grades, the paralysis extends and increases until the patient becomes utterly helpless, and is unable to swallow, food passing into the larynx; while the pupils are unequally dilated; and urine and fæces escape involuntarily. Automatic and reflex movements also cease. The muscles do not waste much as a rule, and they retain their electric irritability. During the progress of the paralysis twitchings and spasms are common. The muscular sense is much affected. Cutaneous sensibility is generally impaired and finally lost. Mental derangement usually precedes the paralysis; it may assume various types, but in most cases there is a brief period of melancholia, followed by a marked change in character, then incoherence, with delusions as to personal importance and greatness, the patient imagining himself to be extremely strong, wealthy, of high birth, or possessed of wonderful sexual powers. The ultimate condition is one of absolute dementia, the mind becoming a complete wreck.

**2. Hemiplegia.**—In the majority of cases of one-sided paralysis only the muscles of the arm, leg, lower part of the face, and tongue are involved in various degrees. In some cases there is a difficulty in wrinkling the forehead or closing the eye; or, on the other hand, the upper eyelid may drop slightly. Speech is often affected, but usually only in cases of right hemiplegia. Deglutition is rarely interfered with. The 3rd, 4th, and 6th nerves almost always escape, while the motor branch of the 5th is also usually but little, if at all, affected. The signs of paralysis of the several cranial nerves will be pointed out when they are individually discussed. It may be noticed that they are more liable to be involved according to their proximity to the posterior extremity of the brain. This may be due to their anatomical arrangement, the fibres of the unaffected nerves lying outside the track of the lesion, and being, therefore, beyond its influence; but it has also been attributed by Broadbent and others to the more intimate connection of the nuclei of the nerves which escape with the corresponding nuclei on the opposite side of the medulla and pons, so that they are more influenced by impulses which start from these healthy nuclei. The arm and leg are most affected in hemiplegia, and if the paralysis is complete the limbs are quite helpless, in the recumbent posture the leg tending to rest on its outer side, with the toes everted. If it is partial, but still well-marked, the gait is usually very characteristic. The patient leans towards the sound side, lifting up the opposite shoulder, and while the arm often hangs helplessly, the leg during progression is carried forward by describing a kind of outward swing or sweep, while the toes are directed downwards towards the ground. In less-marked instances the leg merely drags, the toes, however, pointing downwards, while the arm cannot be moved well, and the power of squeezing is diminished. The leg may be less affected than the arm, and paralysis may occur later in this limb. As regards the muscles of the neck and body, these are as a rule but

little affected, and if they are implicated at first, they generally speedily recover their power.

Not uncommonly partial restoration is effected after hemiplegia, which almost always commences in the leg, beginning above and extending downwards, the muscles on the front of the leg being last restored. The arm may remain for a long time, or even permanently, disabled; if it improves, recovery takes place from the proximal towards the distal part of the limb, as in the leg. The most complicated movements are the last to return. Generally there is no limitation of the reflex movements of respiration on the affected side, at least for some time; further, the superficial reflexes may be readily excited in some cases, sometimes even more easily than in health, but in most cases they are impaired; while the paralyzed muscles of expression can in some instances be brought into play under the influence of strong emotion. The knee-jerk generally becomes exalted; and ankle-clonus is developed. The muscles do not in the large majority of cases show any tendency to waste, except to such a degree as can be accounted for by mere disuse and inactivity. Moreover, electric irritability is not impaired, and may even be increased at first; after prolonged disuse of the muscles it may become somewhat diminished, but can speedily be restored. Under certain circumstances, however, rapid wasting and loss of contractility occur. The temperature is at the outset generally raised on the paralyzed side, but afterwards falls below the normal, it may be as much as  $1^{\circ}$  or more. "Late rigidity" or "contracture" not uncommonly sets in in the affected limbs, especially in the arm. It involves the flexors more particularly; is variable in degree; and is at first remittent and capable of being overcome, being mainly observed during voluntary efforts or under excitement, but by degrees becomes permanent and more and more marked, until at last the limb is completely flexed and rigid. The cause of this "late rigidity" has been much disputed. Probably it is due to descending sclerosis involving the motor tracts of the crus, pons, medulla, and spinal cord, as has been maintained by Charcot, Bastian, Ferrier, and others. Hughlings Jackson regards the condition as a species of tonic distortion, caused by the cessation of cerebral influence over the muscles which in health the cerebrum chiefly innervates, and consequent unantagonized action of the cerebellar centres, and unimpeded cerebellar influx. Duret is inclined to attribute late rigidity exclusively to reflex irritation, owing to the irritation extending to sensory tracts. Ferrier thinks it is possible that in some cases reflex contracture may be superadded to that due to sclerosis. The condition has also been attributed to cirrhosis of the affected muscles; or to the unrestrained action of one set of muscles, their opponents being paralyzed.

**ÆTIOLOGY.**—(i.) Hemiplegia is in the large majority of cases a sign of *organic cerebral disease*, the paralysis being almost always on the side opposite to that of the lesion. By far most commonly it results from some lesion affecting the corpus striatum or the neighbouring white substance (the internal capsule) either directly or indirectly, and causing actual destruction, compression, hyperæmia, or anæmia; it may, however, be immediately associated with injury or disease of a certain portion of the cerebral convolutions, or of other parts of the brain, as will be pointed out in a subsequent chapter. The morbid conditions which may thus give rise to hemiplegia are:—*a.* Rarely congestion, it being then merely temporary. *b.* Hæmorrhage most commonly. *c.* Embolism or thrombosis of a considerable artery. *d.* Acute cerebritis or softening



and abscess. *e.* Chronic softening from any cause. *f.* Cerebral tumour. *g.* Unilateral meningitis. (ii.) In very exceptional instance, hemiplegia results from *unilateral disease of the spinal cord*. Of course there is then no facial paralysis; and the tongue is unaffected. (iii.) Occasionally this form of paralysis is observed in connection with certain *functional nervous diseases*, apart from any evident organic lesion, namely chorea, epilepsy, and hysteria. It may also be associated with pregnancy or parturition.

**3. Paraplegia.**—This form of paralysis varies much in degree, and comes on either gradually, rapidly, or suddenly. When it is complete, the utter helplessness of the legs, as the patient lies in the recumbent posture, or attempts to stand supported on each side, is very striking; in less advanced cases there is more or less weakness and difficulty in movement, with unsteadiness of gait, dragging of the feet, and stumbling while walking. Reflex movements are usually very easily excited. The condition of electric irritability varies in different cases. The height to which the paralysis extends up the trunk will depend upon the seat of the lesion in the spinal cord; and when it involves the upper part all the limbs are affected, constituting the condition termed *cervical paraplegia*, which has already been alluded to.

**ÆTIOLOGY.**—(i.) Paraplegia is in the large majority of cases the result *of injury to, or disease of, the spinal cord*. Thus it may be due to:—*a.* Fracture or dislocation of the spinal column; or a wound or violent concussion of the cord. *b.* Compression of the cord from without by a tumour. *c.* Caries of the spine and its consequences. *d.* Spinal congestion, when the paralysis is usually partial; or spinal anæmia. *e.* Spinal meningitis. *f.* Acute myelitis. *g.* Chronic softening or sclerosis. *h.* Hæmorrhage in connection with the cord. *i.* Morbid growths or parasites. (ii.) Sometimes paraplegia is a purely *functional disorder*, and the varieties belonging to this category which have been specially recognized are:—*a.* *Hysterical*. *b.* Paraplegia depending upon *idea*. *c.* *Emotional*. *d.* *Reflex*, in connection especially with uterine affections, or those of the bladder or urethra; but also associated with pregnancy, dentition, worms, or exposure to cold and wet. Brown-Séquard thinks that reflex paralysis is due to anæmia of the cord, produced by reflex spasmodic contraction of the vessels, through vaso-motor influence. *e.* *Alcoholic*, which is described as a temporary form of paraplegia, induced by alcoholic excess, and also supposed to be due to some vaso-motor effect. *f.* *Malarial*.

**4. Disseminated or Irregular.**—In this form the paralysis is distributed in various parts of the body, for instance, in the arm or leg on opposite sides, or in the limbs on one side, and the face or eye on the other. It either depends upon corresponding irregular distribution of some lesion of the nerve-centres or nerves; or upon disease of a particular portion of these centres, namely, the pons varolii and medulla oblongata, then constituting what is termed *bulbar paralysis*. It is in connection with diseases of this part of the nerve-centre that so-called *cross paralysis* is observed, in which the limbs are paralysed on the side opposite to the disease, and the face on the same side.

**5. Local and Special Paralyses.**—It is not intended here to describe the numerous varieties of local paralysis which may come under observation, but merely to point out their general causes, and to consider the chief facts relating to paralysis of certain special motor nerves.



Local palsy may be an indication of slight or commencing central disease; but in the majority of cases the cause is *peripheral*, either directly affecting one or more nerves, or certain muscles. This *peripheral* paralysis may be due to:—(i.) Destruction of a nerve from injury. (ii.) Pressure upon it by a tumour, aneurism, or inflammatory thickening; or mere temporary compression, as from prolonged sitting, or lying on the arm. (iii.) Changes induced in the nerve itself, probably mostly inflammatory, from neighbouring irritation, such as that set up by necrosed bone or ulceration; exposure to cold; syphilis; rheumatism or gout. (iv.) The entrance of certain poisons into the system, especially lead; or, it is said, malarial poison. (v.) Changes in the muscles, either atrophic or degenerative, as in progressive muscular atrophy. Local paralysis may be a sequela of diphtheria, or more rarely of other febrile affections. It may also result from local embolism. When paralysis results from disease of a nerve or of its nucleus of origin, it is limited to the muscles supplied by that particular nerve; tends speedily to become complete; and it is very liable to be followed by rapid wasting, with loss of electric irritability.

a. **Facial Paralysis—Bell's Palsy.**—Paralysis of either facial nerve and consequently of one side of the face, is the most important local variety that comes under observation in practice, the entire nerve being then usually involved. The signs are as follows:—There is complete absence of expression on the affected side of the face, which appears flattened and smooth, the features being blank and meaningless. The corresponding half of the mouth seems broader than the opposite half, while the angle falls. Sometimes saliva flows from the mouth. The ala of the nose falls in, and consequently the nasal aperture is diminished in size. The healthy side of the face appears to be, or is actually drawn away, and the angle of the mouth seems to be raised. The eyelids on the paralyzed side are unusually apart, the lower one dropping down, and as they cannot be closed the tears tend to trickle down the cheek, the corresponding nostril is dry, and the constant exposure of the eyeball soon leads to irritation of the conjunctiva, which is liable to be followed by serious injury to the deeper structures. It is however, on attempting to bring the affected muscles into play that the most evident signs of facial paralysis are observed. The patient cannot smile, weep, wrinkle the forehead, elevate the eyebrow, frown, close the eyelids, knit the brows, or expose the teeth on the paralyzed side. Articulation of labial sounds is impaired, as well as the ability to whistle; while if the patient is directed to blow out the cheeks, the affected one flaps loosely. During mastication the food tends to collect between the cheek and gums, while fluids often run out of the mouth; the power of spitting is also impaired. If the facial nerve is implicated in a certain part of its course, other less obvious signs are said to be observed, dependent upon some of its branches being distributed to the tongue, salivary glands, and palate, namely, perversion of taste on the anterior part of one side, and occasionally slight drawing of the tongue towards the same side; deficient secretion of saliva; relaxation and imperfect action of the velum palati on, and pointing of the uvula towards, the affected side; with a somewhat nasal character of the voice.

**ÆTIOLOGY.**—It is of considerable importance to recognize in what part of its course the facial nerve is implicated, and to determine the cause of the mischief. The causes of facial paralysis may be summarized thus:—(i.) Organic mischief in the brain, involving the

root of the nerve. (ii.) Pressure upon the nerve within the skull after it has emerged from the brain, especially by various kinds of tumour, or by meningeal exudation. (iii.) Injury or disease involving the nerve in its course through the temporal bone, chiefly from necrosis of the petrous portion of this bone, or from gunshot injury. (iv.) Causes affecting the trunk or branches of the nerve after its exit from the stylo-mastoid foramen, namely, injury, as from a cut or contusion; pressure by parotid and other tumours or enlarged glands; direct exposure of the side of the face to a cold draught of air, as in travelling by train with the window open; general exposure to cold and wet; gout, rheumatism, or syphilis; or, it is said, malarial influence.

DIAGNOSIS.—The diagnosis of the origin of facial paralysis rests on:—

1. The *history* of the case, as revealing some of the causes just enumerated; and also the *mode of onset* of the paralysis, whether sudden or gradual. 2. The *accompanying symptoms*. Thus, when the paralysis is due to cerebral mischief, there are generally evident signs of this, such as hemiplegia and mental disturbance; if there is some intra-cranial pressure outside the brain, headache and other local symptoms are commonly complained of, while other cranial nerves are frequently involved, and sometimes paralysis of the limbs is observed on the opposite side. If the temporal bone is diseased, deafness and otorrhœa are usually present. If the nerve is affected outside the skull, some cause of pressure may be obvious; there may be no symptoms whatever except the paralysis; or the sensory nerves of the face are sometimes implicated as well, in the direction of neuralgia or anæsthesia. 3. The *extent of the nerve* involved. When facial paralysis arises from cerebral causes, it is only the lower part of the face which is in most cases prominently affected, the muscles of the eyelids and forehead either acting normally, or being only slightly weakened. In all the other forms the whole side of the face is paralyzed. It is only when the nerve is implicated in its course through the temporal bone that the palate and tongue are affected. 4. The *degree of electric irritability*. In cerebral paralysis electric irritability is retained, unless the disease lies at the origin of the nerve from its nucleus; in all other forms it is impaired or lost temporarily or permanently to both faradic and constant currents, whilst the muscles react too rapidly to a slowly interrupted constant current, but not to the faradic. 5. The *progress* of the case, and the *effects of treatment*. For instance, when due to tumour, injury, or bone disease, the paralysis is generally persistent; when originated by cold, rheumatism, or syphilis, it may often be cured by proper treatment.

In very rare instances *double facial paralysis* is observed, but it is difficult to recognize. It may be due to centric disease, especially hæmorrhage into the pons; or, it is said, to disease of the nerves from exposure to cold, rheumatism, or syphilis.

*b. Paralysis in connection with the eye.*—The nerves to be considered here are the *third*, *fourth*, and *sixth*. When either of these is involved, this is indicated by some variety of strabismus, with double vision, the relative position of the images seen by the two eyes varying in each case. Complete paralysis of the *third* nerve is characterized by ptosis or dropping of the upper eyelid, with inability to raise it; permanent external strabismus; dilatation and immobility of the pupil, which is usually directed a little downwards; and a difficulty in adapting the eye to vision at different distances, owing to paralysis of the ciliary muscle. In some cases only ptosis is observed, when the paralysis

is peripheral in its origin. The cause may be centric disease; pressure upon the nerve in its course; exposure to cold; or rheumatism. When the *fourth* nerve is paralyzed, the superior oblique muscle cannot act. This is shown by upward strabismus; displacement of the false image downwards; and when the eye-ball is depressed, the pupil is seen to move in a curved line directed downwards and towards the opposite side, the false image being tilted towards this side, when the pupil is below the horizontal line. Paralysis of the *sixth* nerve is evidenced by persistent internal strabismus; and displacement of the false image towards the side opposite to the paralysis. These forms of paralysis are generally associated with some pressure in the course of the nerves, especially by a tumour or meningeal exudation. Sometimes all the nerves of the eye are simultaneously affected. Ocular paralysis is often observed in locomotor ataxy, syphilitic disease, and after diphtheria. There are also certain peculiar cases described by Mr. Hutchinson under the terms *ophthalmoplegia interna* and *externa*, in the former there being progressive and more or less symmetrical paralysis of the internal muscles of the eyes; in the latter, progressive and more or less symmetrical paralysis of the muscles which move the eyeballs and raise the eyelids. Besides this there may be paralysis of the parallel movements of both eyes, either upwards, downwards, or to the right or left, due to lesion of the centre governing these movements.

**c. Paralysis of the tongue.**—As a rule unilateral paralysis of the tongue, dependent upon implication of the *hypoglossal* nerve, is a part of hemiplegia. The signs are a widening of the tongue on the affected side; difficulty in its movement and protrusion; deviation of the organ to the sound side when in the mouth, to the paralyzed side when protruded; and impaired articulation. The entire tongue may be paralyzed, so that it cannot be protruded or moved, rendering articulation impossible, and deglutition very difficult.

**d. Paralysis of the pharynx** is mainly indicated by great difficulty or impossibility of swallowing; and more or less thickness of speech, which assumes a guttural quality, or may be quite unintelligible. It usually results either from some centric disease, affecting the nuclei of the nerves supplying the pharynx, as in glosso-pharyngeal paralysis; or as a sequela of diphtheria. Other parts are also generally implicated, either simultaneously or in succession.

**e. Paralysis of the inferior maxillary nerve** is evidenced by impaired power of mastication on the affected side, and certain derangements of the muscular movements concerned in this act. When the jaws are firmly closed, the temporal and masseter on the paralyzed side remain flaccid, and do not harden. On moving the lower jaw forwards or backwards it assumes an oblique position, in the former case the inclination being towards the paralyzed side, especially noticeable when the patient opens his mouth widely; in the latter case towards the unaffected side. Usually motor paralysis of this branch of the fifth nerve is accompanied with sensory derangement of the face, and the other branches are in most cases involved. The condition generally depends upon some local disease.

**TREATMENT.**—The objects to be aimed at in treating paralysis of any part are to restore the muscles to their normal activity as speedily as possible, if this is practicable; and to counteract the tendency to the atrophic and other changes to which the structures are liable. Of course the measures to be adopted must first of all have reference to



the *cause* of the paralysis, by removing which restoration is often rapidly and completely effected. As illustrations may be mentioned the use of iodide of potassium in the treatment of paralysis due to syphilis or lead. In many forms of paralysis *time* is a most important element in treatment, and much harm may be done in not a few instances by interfering too actively or too soon. It is requisite to see that a paralyzed part is properly covered with warm clothing, and that it is kept clean. The chief local measures employed to counteract palsy are systematic passive motion of joints, which may be combined with efforts at voluntary movements; various baths and douches, either hot or cold; friction, either with the hand alone, with flesh-brushes or gloves, or with some stimulant liniment; shampooing; and electricity, which may also be usefully combined with voluntary attempts to move the affected muscles.

The employment of *electricity* in the treatment of paralysis demands special notice, and it will be convenient here to give a brief summary of the main facts pertaining to this subject, derived chiefly from Dr. Russell Reynolds's work. Much discrimination and caution are needed in resorting to this therapeutic agent, as it is very powerful for evil as well as for good. The beneficial results which electricity is capable of effecting in paralysis are as follows:—1. Restoration of the functions of a muscle or nerve when its activity is impaired, and thus possibly restoration of voluntary movement. 2. Prevention of wasting of the muscles, and consequent arrest of the progress of the disease. 3. Increase in the vascularity of a part, thus removing coldness, blueness, and other signs of feeble circulation. 4. Improvement in the nutrition of the muscles, nerves, and other structures, should they be atrophied or ill-nourished. 5. Prevention, retardation, or removal of spasmodic contractions and rigidity. 6. Probably the long-continued use of electricity may improve the nutrition of the part of the nerve-centre from which the nerve or nerves which supply the affected muscles originate. The kind of electricity required varies in different cases, but it may be stated generally that for promoting the action of muscles faradization and the interrupted galvanic current are most useful, though franklinic electricity is occasionally more beneficial than either; that for improving the circulation and nutrition the continuous galvanic current, or faradization by means of a metallic brush answers best; whilst to oppose the excessive action involved in spasmodic movements and rigidity (and this applies to these conditions under all circumstances), a weak constant galvanic current, or very rapidly-interrupted faradization may be applied to the affected muscles; or in certain conditions of rigidity the use of faradization or interrupted galvanism to the antagonistic muscles is most efficacious.

Some general hints as to the employment of electricity in treating paralysis will now be given. Care must be taken not to frighten the patient at the outset. The current used must not be so strong as to cause pain; or, on the other hand, so weak as to be useless; and the applications should be brief, so as not to tire the patient or the muscles. It may be repeated twice a day, daily, or every other day, according to circumstances. In employing galvanism, one handle, containing a sponge of sufficient size and well-wetted, must be kept fixed on one spot, such as over the shoulder or in the bend of the elbow in the case of the upper extremity, and the other drawn slowly along the muscles in succession. With faradization the two poles must be kept near to-

gether, and it is almost always best to hold both in one hand and draw them along each muscle. More action is excited at certain spots, which generally correspond to the points where the nerves entering the muscles are most superficial. In treating paralysis of a special nerve, one handle must be placed over the trunk of the nerve, and the other moved about or not, according as galvanism or faradization is employed.

A few observations will now be offered on the uses of electricity in the chief varieties of paralysis.

(1.) *Cerebral*.—In cases of sudden cerebral paralysis electricity must on no account be used for some time, even for purposes of diagnosis or prognosis, and the greatest care is necessary in its employment for a long period. Even if the paralysis has been gradual in its onset, caution is needful should there be head-symptoms, such as headache, a sense of weight, or giddiness. Much improvement may be effected in other cases in the various directions already indicated; but, so far as the paralysis itself is concerned, the value of electricity will depend on the degree of contractility shown by the muscles on its first application. If this is normal or nearly so, the power of voluntary movement can be but slightly, if at all, increased by its application. If it is much diminished from want of use, much good may be done by reviving the contractility of the muscles, but once this has become normal, no further improvement as regards voluntary motion can be effected.

(2.) *Spinal*.—If the muscles act readily under electricity in cases of complete spinal paralysis, the power of voluntary movement in the limbs cannot be increased by its use, but sometimes the functions of the bladder, rectum, or sexual organs may be much improved by its local application to the anus or perinæum. If the paralysis is partial and contractility is impaired, much good may be effected up to the point of restoring the contractility; electricity must not, however, be employed in acute cases of this kind, but it is of the greatest value in those which have set in slowly. If there is atrophy of the limbs, galvanism is most efficacious; if none, faradization. Should there be the complete "spinal paralysis" of Marshall Hall, electricity cannot improve the power of movement, and if no sign of contractility is observed after a few applications, it is useless to proceed with its use; if the contractility is merely impaired, much improvement may often be brought about. In cases of infantile paralysis due to spinal disease, the use of the slowly-interrupted galvanic current frequently proves very serviceable for a time when other forms of electricity fail, but as the muscles improve in their action faradization becomes most efficacious.

(3.) *Local*.—Should a nerve be completely destroyed in its course, and electric contractility be quite extinguished, no improvement can be effected by electricity. In some cases, however, the morbid changes in the nerve disappear, but more or less paralysis persists from want of use. Here electricity is of great service, and it will be well in such cases to continue its application for some time, provided any contraction can be excited. In certain cases of local paralysis from lead, cold, and other causes, the use of a slow galvanic current has most effect upon the muscles. Under such circumstances, therefore, this current should be used at first, and a gradual change made to faradization as improvement is perceived.

A few special remarks are needed with reference to the treatment of *facial paralysis* from exposure to cold. The local application of heat and moisture constantly, leeching, and steaming, are the measures which



are most useful at first ; followed by blistering, friction with stimulating liniments, and the employment of the slow galvanic current. Iodide of potassium, quinine, or strychnine act beneficially in some cases, when given internally.

### III. SUPERFICIAL AND DEEP REFLEXES.

The several *superficial reflexes* correspond to certain definite portions of the spinal cord, the stimulus being conducted by the afferent or posterior root of the nerve, traversing the grey matter, and passing out by the efferent or anterior root, thus giving rise to muscular contractions. As reflex actions can be produced which have their respective centres in almost every part of the cord, their absence, presence, or exaggeration gives important information as to the conditions of particular portions of this nerve centre, and of the corresponding nerves.

The *tendon-reflexes* have been much studied by Erb, Westphal, Gowers, Buzzard, De Wattville, Waller, and others. Erb, who originally investigated them, regarded them as ordinary reflex phenomena. Westphal attributed them to the immediate contraction of the suddenly-stretched muscles. They can only be evoked in muscles which are in a state of passive tension ; and Gowers suggests "that the tension excites, by a reflex influence, a state of extreme irritability to local stimulation." Buzzard has been compelled to agree with those who hold the view that the phenomenon is not a spinal reflex, though dependent upon the integrity of the reflex arc. The question, however, is still "*sub judice*."

With regard to the *pathological relations* of the tendon-reflexes, it will only be practicable to allude here to the *patellar-reflex* and *ankle-clonus*. The patellar reflex is almost invariably present in health, and it is stated that it is only absent in about one per cent. of individuals examined. Its persistence "almost always signifies that the nervous arc is not seriously interrupted in that part of the spinal cord which gives origin to the lumbar plexus." (Buzzard.) It may be *lost* either from a "fault" in the muscle itself (as in pseudo-hypertrophic paralysis) ; from a lesion of the anterior root of the spinal nerve ; atrophy of the large ganglion cells in the anterior horn, as in infantile paralysis or adult spinal paralysis ; or a lesion in the sensory tract, as in locomotor ataxy, which is the most important disease in which the knee-reflex is lost, and this may be observed before the more striking symptoms of the disease have become developed. There are exceptional cases of locomotor ataxy associated with lateral sclerosis in which it persists, and still more rarely it is exaggerated. The patellar-reflex is also absent whenever the lumbar portion of the spinal cord is greatly disorganized, as by acute myelitis or softening. Eulenberg has shown that tendon-reflexes are diminished by several anodynes and sedatives, sometimes after a preceding augmentation.

Attention will now be directed to *increase* of deep-reflexes, or their development in disease. *Ankle-clonus* cannot be produced in health, and its occurrence is a morbid sign. Gowers considers the *front-tap contraction* a very delicate test of morbid irritability, and states that it can often be obtained when the ankle-clonus cannot. The *knee-reflex* is also exaggerated in some forms of disease, a blow that in health would cause only a very slight movement of the foot producing a jerk upwards of



several inches; or a much lighter blow than will cause any effect in health may even produce a very decided kick. This may be associated with the development of ankle-clonus, and the exaggeration of these reflexes in anything like a marked form "signifies that from some cause the inhibitory influence of the higher centres is no longer being normally excited" (Buzzard). This writer considers it probable that the restraining influence which is continually and unconsciously exerted over certain spinal reflexes in a state of health, is conveyed to the ganglion-cells of the anterior horns by the antero-lateral columns of the cord. It is when these columns are most extensively invaded by sclerosis that the most marked ankle-clonus is obtained, and this sign is considered valuable evidence of sclerosis of the lateral column. It may also follow hemiplegia from cerebral disease; and may be observed to a moderate degree in hysterical paraplegia, as well as in spastic paraplegia. There seems to be some difference of opinion as to the diagnostic value of exaggerated knee-jerk, and the development of ankle-clonus, and Bastian states that the latter may exist to a well-marked extent when the antero-lateral columns of the cord are pressed upon at a certain level, even though no lateral sclerosis has been developed. The production of deep reflexes in the upper limb needs no special comment. Eulenberg has found that subcutaneous injection of strychnia increases tendon-reflexes, and may under certain circumstances be used to render the completely absent tendon-reflex temporarily perceptible and graphically demonstrable. The tendon-reflexes are usually increased, and ankle-clonus can be obtained for a few minutes after an epileptic fit.

---

## CHAPTER LXXIII.

### DISORDERS OF SENSATION.

ALL forms of sensation may be more or less impaired or lost; exalted; or perverted. In the present chapter it is only intended to consider the most important deviations noticed in connection with ordinary sensation.

#### I. SENSORY PARALYSIS—HYPÆSTHESIA—ANÆSTHESIA.

Sensation may be more or less impaired—*hypæsthesia*; or completely lost—*anæsthesia*. Generally it involves the whole thickness of the tissues of the part affected, but may be confined either to the skin or to the muscles. Anæsthesia may be gradually established, sensation becoming more and more impaired; or it may occur suddenly. When this condition exists, the patient is wholly insensible as regards tactile sensations, and may be pinched, pricked, cut, or injured in any other way without being aware of it. In *hypæsthesia* the sense of touch is more or less indistinct, and the patient feels as if a thick layer of some soft and yielding material, such as cotton-wool or flannel, intervened between

the skin and anything brought into contact with it. This is especially noticed in connection with the hands and feet, when the patient grasps anything or stands. In this condition also, as well as during the development of anæsthesia, various unusual sensations or *paræsthesiæ* are often experienced, such as numbness, formication, tingling, or pins and needles. In some cases even of complete anæsthesia to objective impressions, neuralgic pains of a subjective character are complained of in the affected part. It is a curious fact that in exceptional cases, although tactile sensation is lost, the power is retained of distinguishing differences in temperature, or of feeling painful impressions. Occasionally, in connection with marked hypæsthesia, the impression of anything brought into contact with the affected surface seems to be delayed in its passage to the nerve-centre, so that it may be some seconds before the patient is conscious of it. There is often in this condition great difficulty in distinguishing different sensations from each other. With regard to muscular sensibility, when this is lost there is almost always loss of muscular contractility and sensibility of the skin, but in exceptional instances these are unimpaired. In cases of cutaneous anæsthesia reflex irritability may be destroyed, normal, or increased, according to the cause of the loss of sensibility. The distribution of sensory paralysis presents the same variations as in the case of motor paralysis. Thus it may be:—1. **General.** 2. **Unilateral—Hemianæsthesia.** 3. **Bilateral,** but affecting only the legs and the lower part of the body—**Sensory paraplegia.** 4. **Disseminated.** 5. **Local.** The ætiology of the two kinds of paralysis is also very similar, and it will only be necessary to allude briefly to the more frequent varieties of sensory paralysis, but it may be remarked that all forms are not uncommonly associated with functional diseases, especially hysteria.

1. **Hemi-anæsthesia**, when present, is usually the result of some cerebral lesion, but in a large number of cases of hemiplegia from this cause sensation is intact; or it may be impaired at first, but is speedily restored. Moreover, in cerebral hemi-anæsthesia sensation is not as a rule completely lost, or it may be retained in certain parts, or the anæsthesia may be irregularly distributed. The lesion which causes it generally implicates the optic thalamus or the posterior part of the internal capsule, but may be situated in the posterior portion of the cerebral convolutions. In rare instances hemi-anæsthesia results from disease of one lateral half of the spinal cord, the loss of sensation being on the side opposite the lesion.

2. **Bilateral anæsthesia**, involving the legs and lower part of the body, is almost invariably associated with paraplegia, and is due to disease of, or injury to, the spinal cord. Sensation is, however, more or less retained in many cases where the power of motion is completely lost.

3. **Local anæsthesia** is generally due to disease of some special nerve, or of its nucleus of origin, its seat varying accordingly. It might possibly be associated with localized disease affecting certain of the posterior convolutions of the brain. When a particular nerve is paralyzed, if it is a compound one sensation and motion will be equally impaired. One of the best illustrations of paralysis of a purely sensory nerve is that of the *superior maxillary*, or of its continuation the *infra-orbital*; sensation is then lost in the parts to which this nerve is distributed, and when the patient attempts to drink out of a glass or cup, a very curious feeling is experienced, as if the vessel were broken

opposite the middle of the upper lip. Nutrition and secretion are frequently seriously interfered with when sensory nerves are paralyzed.

**TREATMENT.**—The general remarks made as to the treatment of paralysis of motion apply equally to that of sensation. Local warmth, friction, and electricity are often useful. The latter must not be resorted to for some time in cases of anæsthesia or hypæsthesia from cerebral causes, and even then only very cautiously ; it does not lead to much improvement in most of these cases. Faradization with a brush acts best. Electricity is often very beneficial in various forms of sensory paralysis met with in hysteria, either faradization or franklinic electricity being employed, the latter by directing sparks on to the affected part; charging this part and then drawing sparks from it; or applying a small charge from a Leyden-phial. If sensibility is lost locally from destruction of a nerve, no benefit can be anticipated from electricity. When motor and sensory paralysis are combined, electrical treatment directed to the former may improve the latter at the same time. Particular care is necessary in cases of sensory paralysis as regards cleanliness and avoidance of pressure.

## II. HYPERÆSTHESIA—DYSÆSTHESIA.

*Hyperæsthesia* signifies increased or exalted sensibility, *dysæsthesia* increased sensibility to painful impressions, but they are almost always associated. These deviations from the normal state are usually local, but occasionally unilateral or disseminated. They are of course frequently met with in diseases of the skin and other affections, but as regards nervous diseases, they are generally associated either with hysteria or general nervousness, or with acute inflammation involving the nerve-centres or special nerves. When one leg is paralyzed from spinal disease, its sensibility frequently becomes painfully acute.

## III. NEURALGIA.

*Neuralgia* or nervous pain is a comprehensive term applied to certain painful affections occurring in different parts of the body, the pain appearing to follow the distribution of particular nerves, and having special characters. It will be convenient first to consider the general ætiology, pathology, and clinical features of the complaint; and then to allude briefly to its principal varieties.

**ÆTIOLOGY AND PATHOLOGY.**—In a large proportion of cases neuralgia is distinctly dependent upon some *general* or *constitutional* condition. The causes which may give rise to such a condition are:—1. Exposure to malaria. 2. The presence of certain metallic poisons in the system, as lead, mercury, or copper. 3. Whatever tends to induce anæmia, or general mal-nutrition and debility. 4. Various causes which lead to depression and weakness of the nervous system, such as prolonged worry and anxiety; undue mental exertion; strong emotion; general concussion of the nervous system, as from a railway collision; hysteria; excessive fatigue; exposure to heat; ennui and luxurious habits; or excessive venery. 5. Degenerative changes associated with the decay of life. 6. Rheumatism, gout, syphilis, or exposure to cold and wet. In the case last-mentioned, however, neuralgia is probably mostly the result of inflammatory and other changes in the nerve itself; or of



pressure by surrounding thickening of fibrous membranes, or by morbid deposits.

An important group of causes of neuralgia are *local* in their action, including:—1. *Injury* to a nerve in some part of its course, as from contusion; wound by a needle; partial section; the lodgment of a foreign body, such as a piece of glass, when the pain may be felt in some distant part. When a nerve is completely cut across, either itself or some other nerve related to it now and then becomes subsequently the seat of neuralgia. 2. *Pressure* upon a nerve, for example, by foreign bodies, such as a bullet; cicatricial thickening or old adhesions; neuro-mata; tumours, aneurisms, or enlarged glands; callus uniting fractured bones; congested veins; or as the result of prolonged sitting, tight boots, or hanging the arm over a chair. 3. *Irritation* of a nerve by necrosed bone, especially when it passes through a foramen or canal in this condition; by carious teeth; by surrounding inflammation or ulceration: or by direct exposure to a cold draught. Even when neuralgia is local in its origin, its occurrence is greatly influenced by the general state of the system, and Anstie went so far as to affirm, "it is universally the case that the existing condition of the patient at the time of the first onset of the disease is one of debility, either general or special." Among the chief *predisposing causes* of neuralgic affections may be mentioned the female sex; certain periods of life, especially that of sexual development, and about or beyond middle age; hereditary tendency to nervous affections; and a nervous temperament. An acute attack is predisposed to or intensified by fatigue or any other lowering influence. It may come on quite spontaneously; or be brought about by mental disturbances, pressure, cold, heat, over-exercise, and numerous other influences.

Pathologically neuralgia may be connected with some evident morbid change in the affected nerve or in the nerve-centre, but as a rule no such obvious change can be detected. Probably in some cases the nerve is more or less congested or inflamed. Marked atrophy with degeneration of a nerve has been found in connection with neuralgia from pressure, occasionally so advanced as to render sensation almost extinct. Anstie remarked "I think it most probable that in *all* cases of neuralgia there is either atrophy, or a tendency to it, in the posterior or sensory root of the painful nerve, or in the central grey matter with which it comes in closest connection."

**SYMPTOMS.**—*Pain* is the essential symptom of neuralgia. The important characters of this pain are as follows:—1. It is almost invariably unilateral. 2. In recent cases it is distinctly intermittent, coming on in more or less sudden paroxysms, usually at irregular intervals, but occasionally at regular periods, especially in malarial cases; later on it is only more or less remittent. 3. The pain during the paroxysms is generally severe, in some cases most excruciating, being described by such terms as stabbing, piercing, boring, burning, or screwing, at the same time shooting out from a point along some of the branches of the nerve affected, but rarely along all of them; the darts, twinges, or "ties" in some instances come on with the suddenness of an electric shock, giving rise to intolerable agony. The pain may extend to contiguous or even to distant nerves. Frequently strong pressure over the chief point affords relief; in other cases gentle friction has the same effect; in others, however, there is exquisite tenderness. The paroxysmal pain often ends as abruptly as it commenced, with a sense of ex-

treme relief and comfort. The pain in the intervals is much less severe, of dull or aching character, and in the superficial neuralgias presents circumscribed points of tenderness—*points douloureux*, corresponding to the exit of branches of the nerve through bony foramina, or through openings in fibrous membranes, though they appear to the patient to be diffused beyond these spots, in some cases giving the sensation of tolerably extensive contusions. It is not practicable in a treatise like this to indicate the seat of all the tender spots noticed in the different local neuralgias, but a knowledge of the distribution of the nerve affected, and of the points at which its branches become superficial, will enable their situations to be recognized.

There are certain general facts relating to neuralgia to which it is desirable to allude. When the complaint is *local* in its origin, the pain usually sets in more gradually; is more constant; and is less capable of relief. In advanced age neuralgia is commonly very severe and intractable, the points of tenderness being often intensely painful. Gouty neuralgia is also sometimes extremely intense. Once an attack has happened, there is always a liability to its recurrence, and the paroxysms may be repeated at regular periods. An individual may have had neuralgia when young, and then be quite free from it for many years, but be again subject to the complaint at a later period of life. Different nerves may be implicated in different attacks, or even during the same attack.

Some interesting *complications* are often associated with neuralgia, affecting either sensation or motion; the state of the vessels; or nutrition and secretion. The chief of these which have been noticed include local hyperæsthesia, hypæsthesia, or paræsthesiæ, such as numbness, tingling, or formication; disturbances of the special senses, especially that of sight; spasmodic twitchings, tonic spasms, convulsive movements, or even local paralysis; pallor, followed by redness of the skin, pulsation of the arteries, increase in temperature, and swelling of the affected part, with subcutaneous œdema; hypertrophy or atrophy of the tissues in prolonged cases, or increase of adipose tissue; increased firmness, falling-off, or whitening of the hair; the breaking out of skin-eruptions, such as herpes zoster or acne; increased vascularity of the conjunctiva, conjunctivitis, iritis, and other morbid conditions of the eye; periostitis; swelling or unilateral furring of the tongue; erysipelatoid inflammation of the tissues to which the affected nerve is distributed; impaired gastric secretion; increased flow of saliva or tears; and local increase of perspiration.

**VARIETIES.**—Neuralgias are primarily divided into:—I. **Visceral**, including:—1. *Cardiac*. 2. *Hepatic*. 3. *Gastric or gastralgia*. 4. *Intestinal or enteralgia*. 5. *Peri-uterine and Ovarian*. 6. *Testicular*. 7. *Renal*. II. **Superficial**, namely:—1. *Tic-douloureux*. 2. *Cervico-occipital*. 3. *Cervico-brachial*. 4. *Intercostal*. 5. *Mastodynia or Irritable breast*. 6. *Lumbo-abdominal*. 7. *Sciatica*. 8. *Crural*. The visceral group will not be further alluded to, the most important of these affections having been already considered under their respective organs. The names applied to the several forms of *superficial* neuralgia will indicate their respective localities, but a few need special comment.

1. **Tic-douloureux—Brow-ague—Prosopalgia.**—This is one of the most common forms of neuralgia, the *fifth* or *trigeminal* nerve being involved. Rarely are all the divisions implicated, and it is the ophthalmic branch which is most frequently affected, the pain, therefore, being

chiefly felt above the orbit and about the temple. Numerous points of tenderness are described, but the most important are the *supra-orbital* and *parietal*, the latter being situated just above the parietal eminence, and corresponding to the inosculation of several branches. A variety of this neuralgia is named *clavus hystericus*, in which there is extreme pain, in character as if a nail were being driven into one or more spots, usually corresponding to the supra-orbital or parietal points.

**2. Intercostal Neuralgia.**—In this variety the pain is felt along the course of one or more intercostal nerves. Those on the left side, especially from the 6th to the 9th, are most frequently affected. There is a constant pain, mostly corresponding to the point of exit of a lateral cutaneous nerve, and increased by a deep inspiration or cough, or sometimes by moving the arm. Shooting pains are also experienced at intervals, extending from the spine along the intercostal spaces, or from the lateral point backwards and forwards. Three very distinct "*points douloureux*" can generally be detected, namely:—*a. Vertebral.* *b. Lateral*, opposite the lateral cutaneous branch. *c. Sternal* or *epigastric*, where the anterior cutaneous nerve perforates. This variety of neuralgia is very common in anæmic and chlorotic females. It also frequently precedes herpes zoster, and a very severe and obstinate form is liable to follow this affection in old people. For the diagnosis of the pain of intercostal neuralgia from that of pleurodynia or pleurisy, the condition of the patient; the want of connection of the pain with any excessive or prolonged exercise of the local muscles, of any marked exacerbation from their use, or of relief from rest; the characters of the pain, with the points of tenderness; and the results of physical examination, are generally quite satisfactory. The appearance of an eruption of herpes is pathognomonic.

**3. Sciatica or Hip-gout** are the names applied to neuralgia in the course of the branches of the sciatic and other nerves about the hip. Generally the pain is mainly seated in the buttock and posterior and outer part of the thigh, but it may affect various parts of the lower extremity, even down to the leg or foot. There is generally a persistent and deep pain near the tuberosity of the ischium, which is increased paroxysmally, shooting upwards or downwards, either without any cause, or as the consequence of pressure, movement, especially a sudden jerk, or even the act of coughing. The patient is often obliged to walk very carefully, or may be unable to move at all. Local anomalies of sensation; spasmodic movements or cramps; and partial paralysis are very common in sciatica. Many cases of this affection are exceedingly severe, and will not yield to treatment. The limb may waste from want of use.

The *local* causes which most frequently give rise to sciatica are long-continued sitting; direct exposure of the buttock to a cold draught, as in using windy privies; and sitting on a cold or damp surface. Not unfrequently this complaint is associated with gout or rheumatism.

**TREATMENT.**—The general principles applicable to, and the chief remedies employed in the treatment of all forms of superficial neuralgia, will now be briefly considered. 1. Any *local cause* of irritation must be removed. In regard to this point a word of caution is necessary respecting tic-douloureux. This complaint is often attributed to decayed teeth, and not unfrequently these are extracted one after another without any improvement resulting, for the simple reason that the neuralgia is not dependent upon this cause at all. 2. It is highly important that those who are subject to neuralgia should adopt measures to *prevent*



attacks, by attending to diet and hygiene; wearing warm clothing; regulating the state of the alimentary canal; and, in short, promoting a state of good general health in every possible way, while at the same time they avoid every cause which is likely to bring on a paroxysm.

3. Treatment directed to the *general state* of the system, or to some *constitutional diathesis*, is in a large proportion of cases of the utmost consequence. Radcliffe and Anstie have shown the great advantage to be derived from the use of fatty elements in neuralgia, when nutrition is impaired, especially cod-liver oil or Devonshire cream. Iron in anæmic subjects; quinine in full doses, especially in malarial neuralgias; arsenic in the form of Fowler's solution; strychnine or nux vomica, are among the most valuable remedies for neuralgia. In some instances valerianate and other salts of zinc, or nitrate of silver prove serviceable. Phosphorus has been found highly beneficial in many cases. Should the neuralgia be associated with gout, rheumatism, syphilis, or the presence of some metallic poison in the system, treatment directed against such a condition is essential.

4. An important class of remedies in the treatment of neuralgia are those which have a direct *sedative* effect on the nervous system, including mainly opium or morphia; belladonna; cannabis indica; hydrate of chloral; croton-chloral; bromide of potassium; conium; atropine; tincture of aconite; veratria; and ammonium chloride in full doses. Other drugs stated to be very efficacious are eucalyptol, the essential oil derived from the *Eucalyptus globulus*; tincture of gelsemium sempervirens; nitro-glycerine; and tonga. These medicines are either given by the mouth; or applied to the affected part in the form of plasters, liniments, ointments, oleates, or tinctures; or, above all, some of them are introduced by subcutaneous injection, particularly morphia and atropine, which may be used in combination. They are not merely to be used for the temporary relief of pain, but are in many instances most important agents in bringing about a cure, if employed systematically and regularly every day for such a period as each case may require. In using subcutaneous injections, it is best to begin with a very small dose—gr.  $\frac{1}{10}$  to  $\frac{1}{6}$  th of morphia, increasing it as occasion requires, some cases needing large quantities after a time. As a rule the injection need not be made at the seat of pain, but Anstie recommends that this should be done in advanced cases, where there is much hyperæsthesia, and where there is reason to believe that much thickening and hypertrophy exist about the nerve. If necessary, the sensibility may be first blunted by the ether spray. The use of *alcohol* demands brief notice. There can be no doubt but that the pain of neuralgia may often be temporarily lulled by the use of alcoholic stimulants, but experience has convinced me that we should hesitate in recommending them, as there is in this complaint a strong tendency on the part of the patient to be taking stimulants at frequent intervals and in increasing quantities, so that the foundation may be laid for confirmed habits of intemperance. It must also be remarked that due caution should be observed in the use of narcotics, lest the patient should become so habituated to them that he cannot do without them.

5. Certain *anodyne local applications* have already been alluded to above, the most useful being liniment or plaster of belladonna or opium; tincture of aconite; ointment of aconite or veratria; and a liniment containing eucalyptol. Among other local remedies which may be serviceable are dry heat, or heat with moisture; chloroform liniment; sinapisms; flying blisters; and light linear cauterization. In

obstinate cases blistering and even stronger forms of counter-irritation may be required. Cold is useful in some cases, in the form of ice or evaporating lotions, and I have in more than one instance found much benefit follow the application of the ether spray over the seat of pain for a few minutes three or four times daily. Massage has been much commended for neuralgia. A most valuable local method of treatment is that by electricity. The constant galvanic current is decidedly the best as a rule, but sometimes faradization acts beneficially, or merely charging the patient from a friction-machine, and afterwards drawing a spark from the seat of pain. In employing galvanism, it is necessary to use only a very weak current, especially about the head, carefully guarding against giving rise to unpleasant head-symptoms; to apply it by well-wetted sponges in the direction of the nerve, the positive pole being placed over the seat of pain; and not to make the application for too long a time, but with frequent repetitions. Surgical interference has been had recourse to in very obstinate cases of neuralgia. The method of procedure now most in favour is to expose the affected nerve, and stretch it forcibly; the other modes are to divide the nerve, or even cut a piece out. Another plan is to forcibly flex a limb, and thus stretch a nerve. Temporary improvement generally follows these methods of treatment, and sometimes a cure is thus effected.

---

## CHAPTER LXXIV.

## APHASIA—APHEMIA—AMNESIA.

DISORDERS relating to speech and the use of articulate language, as well as to reading and writing, constitute important clinical phenomena, which at the present day have been much studied in nervous diseases, and the terms given above are used to express the chief deviations from the normal state which are observed with respect to spoken and written language as an intellectual act. Exclusive of stammering, there are two classes of causes which interfere with the proper employment of articulate language, which are not recognized under these terms, and which must be excluded at the outset:—1. There may be complete mental incapacity and loss of intellectual power, so that no ideas are originated in the mind which the individual wishes to convey, as in the case of idiots. 2. There may be merely a difficulty or even an impossibility of performing the mechanical act of articulation, owing to more or less paralysis of the parts necessary for this act, namely, the tongue, lips, and palate; the power of thought, and also of expression, as evidenced by the ability to write sensibly, being perfectly natural. This is observed in certain cases of hemiplegia, general paralysis of the insane, glosso-labio-laryngeal paralysis, some cases of locomotor ataxy, chorea, and other affections. The exact form of the deviation differs in the several conditions, and it must be remembered that paralysis of articulation may accompany true aphasia.

Coming now to the consideration of the various phenomena presented by cases grouped under the term *aphasia*, it may be remarked that, although as a rule the mental condition is more or less impaired, it is not affected to such a degree as to prevent the formation of ideas, but the patient cannot recollect words or their meaning, and thus is unable to express his thoughts; or has lost the power of co-ordinating and arranging them in a proper manner, for purposes of articulate or written language. In its strict signification aphasia merely refers to disorders of *speech*, but it is at present usually employed in a general sense, to include all the different forms of derangement which come under the group now under consideration, and the special use of the other terms mentioned will be presently indicated. It must be remarked that in all forms of aphasia, phonation or the power of producing vocal sounds is retained more or less.

**PATHOLOGY.**—The conditions recognized as aphasia are in the large majority of cases associated with *right* hemiplegia, and are due to some lesion in the left cerebral hemisphere, involving the region which the left middle cerebral artery supplies. It is more particularly with *embolism* of this artery that they are connected, as was first pointed out by Dr. Hughlings Jackson; but they may depend upon hæmorrhage, softening, cerebral tumour, and other lesions, and I have known them arise temporarily, apparently from mere vaso-motor disturbance. With regard to the exact localization of the mischief, some writers maintain that there must be an injury to some part of the corpus striatum, or of certain motor nuclei or inter-communicating fibres in its neighbourhood. Niemeyer partly attributed the frequency of aphasia in connection with diseases in the region above indicated, to the fact that pressure acting on one side in this region is readily propagated to the opposite one, so that the brain becomes bilaterally disordered. Most authorities now, however, agree with Broca, in regarding the third left frontal convolution as being the seat of the faculty of articulate language, and in referring the disorders met with in most cases of aphasia to some lesion affecting specially the posterior third of this convolution. When both sides are involved, paralysis of articulation may be observed along with aphasia, as in a case reported by Dr. Barlow. There is one form of aphasia, named *aphemia*, in which the patient is quite speechless, which is supposed to depend upon disorder of a special co-ordinating centre, situated somewhere in or below the corpus striatum, whose assumed function is to regulate or combine the groups of movements necessary for the production of elementary articulate sounds, it being so placed as to receive the communications from the intellectual centre of language above, and to transmit them to the nuclei of origin of the motor nerves below, by which they are conducted to the muscles of articulation. Any lesion affecting this centre, or cutting off its communication either above or below, may, it is presumed, lead to speechlessness. No special morbid condition, however, has been definitely associated with this form of aphasia.

**SYMPTOMS.**—The phenomena included under the term *aphasia* are somewhat variable, and it is necessary to point out briefly the chief diversities observed in different cases in actual practice.

In one rare group, to which Bastian would limit the term *aphemia*, the patient is completely speechless, even after having regained the use of every other faculty which has any relation to speech. He is able to write, and retains all his mental faculties; while there is no paralysis of



the muscles of articulation, for these can be used perfectly for all other movements. Aphemia has been noticed after epileptic or apoplectic fits. Several years ago I had under my observation a case which was probably of this kind. The patient was a young girl who, after a sudden fit of insensibility, was found on recovering consciousness to be suffering from right hemiplegia and complete mutism. She recovered the use of the leg entirely, but the arm remained permanently paralyzed, and even at the end of some years there was no sign of returning speech. The case was supposed to be one of embolism.

In another class of cases there is a loss of memory of words or even of letters, this condition being termed *amnesia*. It is almost always accompanied with impaired power of recalling facts, and of conducting mental operations; but there is no necessary relation between these defects, and amnesic patients are often quick in perception and intelligence, obviously understanding everything said to them. The amnesic condition is evidenced in the speech, reading, and writing; and presents the widest differences in the degree of its manifestations, though the power of articulation and of writing are perfectly retained. With regard to speech, as examples of the disorders to which this act is liable may be mentioned the use of only one or two inarticulate sounds, words, or phrases, or of a few words or expressions, which the patient tends to repeat; the use of wrong words in sentences, and loss of memory as regards the names of things and persons, which are wrongly applied; forgetfulness of the names of letters; the occasional misuse or omission of certain words or letters; the employment of wrong endings or beginnings of words, or the transposition of syllables or letters. The patient may repeat words which have been uttered immediately before, but speedily forgets them. Individuals who are almost completely amnesic may utter words or expressions under the influence of powerful emotions. Some of these disorders are often recognized when the patient attempts to read, though some patients can read distinctly enough, but are unable to compose sentences for themselves, even sufficient to answer the simplest question. With respect to writing, though this act can be done from a copy, provided the patient is not suffering from right hemiplegia, he generally cannot write correctly out of his own head or from dictation. Those who can write, occasionally write sense, frequently nonsense, but more frequently either unintelligible characters, or distinct but unconnected words (Reynolds). They may copy from printed matter, and yet not have the slightest idea of the names or meaning of the letters or words. Occasionally they can write down figures from dictation when they cannot put down words, and may even be able to do simple arithmetical sums.

In some cases of aphasia there is a combination of amnesia and aphemia. These conditions may also be associated with actual paralysis of articulation.

TREATMENT.—There is no special treatment for the various aphasic conditions, and this must be directed to the disease with which they are associated. In cases of aphemia, it may be possible to teach lip-speech, according to the manner in which deaf-mutes are instructed.

---

## CHAPTER LXXV.

## ON SECONDARY AND TROPHIC LESIONS IN NERVOUS DISEASES.

OF late years much attention has been paid to the study of the lesions which arise in the course of organic cerebro-spinal nervous diseases, both in the nervous structures themselves, and also in various other tissues of the body. This subject will now be briefly considered, although it will be hereafter further illustrated in connection with particular diseases. For our knowledge respecting these lesions we are greatly indebted to Charcot.\* The conclusions arrived at have been determined by experiments on animals; as well as by clinical and pathological investigation.

1. With regard to the *nervous system* itself, secondary degenerative changes are liable to follow various localized lesions affecting either the centres or the nerves, and extending upwards, downwards, or horizontally, hence named *ascending*, *descending*, and *collateral* lesions. Various additional symptoms are consequently developed in the course of nervous diseases. When hæmorrhage takes place into one of the cerebral hemispheres, especially involving the corpus striatum, the motor tract proceeding therefrom is liable to undergo gradual degeneration in a descending direction, involving in succession the crus cerebri, anterior pyramid, thence passing to the opposite side of the spinal cord, and finally proceeding downwards chiefly along the posterior part of the lateral white column. The same result may follow other lesions. As has been already mentioned, this probably accounts for the "late rigidity" observed in certain cases of hemiplegia. In the spinal cord, if a lesion occurs in some part of its length, it may extend both upwards and downwards, the ascending degeneration being confined to the posterior column, the descending to the lateral column. When the lower end is involved, the changes are liable to extend in an upward direction along the posterior columns, especially those portions which lie contiguous to the posterior median fissure. The degeneration may also proceed horizontally, either from the original seat of disease or from the secondary lesions, and then the anterior cornua and their motor nuclei are particularly implicated.

With reference to the influence of affections of the nerves, more or less serious phenomena are attributed to irritation of the nerve-centres excited by lesions of the sensory nerves. Thus, it is believed by many authorities that centric irritation induced in this manner may account for certain grave diseases, such as tetanus. If the anterior root of a spinal nerve is in any way divided, degeneration extends along its peripheral or distal portion; while if the posterior root is cut, the change proceeds towards the centre, along the portion still in connection with the cord.

2. The effects of diseases of various parts of the nervous system upon the nutrition of *other structures* are often very striking. They may be

\* See "Diseases of the Nervous System," Vol. LXXII. of "New Sydenham Society's Works."

observed in connection with lesions of nerves or of nerve-centres, and have been found more especially associated with the skin and subcutaneous tissues, the muscles, the joints and bones, and certain internal viscera, especially the kidneys and bladder. It has long been known that muscles may waste in paralyzed parts from mere prolonged inaction and disuse, and may undergo more or less fatty or other degenerative changes; also that inflammation, ulceration, or gangrene may arise in structures deprived of sensation, due merely to the fact that in consequence of the loss of sensibility the patient is unconscious of pressure, of the contact of mechanical irritants or irritating excretions, and of other deleterious influences, and thus is unable to prevent them from injuring the tissues. This is well illustrated by the bed-sores which are liable to form in persons suffering from spinal disease, with complete paralysis of the lower extremities. The trophic lesions now to be considered are not, however, thus explicable, but are due to irritative or inflammatory lesions of different parts of the nervous system, the nature of which will be presently pointed out.

As regards the character of the consecutive changes in the several structures, in the skin and subcutaneous tissues they are either of an inflammatory or an atrophic nature, and are evidenced mainly by erythematous redness, which may be combined with tumefaction of the skin and subcutaneous tissue, simulating phlegmon; vesicular and bullous, or sometimes pustular skin-eruptions, such as herpes, pemphigus, ecthyma, &c.; glossy skin; and acute gangrene, ending in bed-sores. The muscles undergo acute wasting, more or less complete loss of electric contractility occurring with equal rapidity. These effects are due to an inflammatory process, which is evidenced by hyperæmia and hyperplasia of the interstitial connective tissue, with multiplication of the nuclei of the sarcolemma, the muscular fibres being involved as a consequence of these changes, becoming gradually more and more attenuated, but rarely if ever losing their striated appearance or undergoing fatty degeneration. In connection with the joints, the morbid conditions which have been noticed are acute or subacute inflammation or synovitis, which often ends in ankylosis; or sudden attacks attended with more or less diffused swelling of the limb, but with little or no pain, and ending in the rapid destruction and erosion of the cartilages and articular ends of the bones, which may lead to dislocation. Occasionally periostitis occurs, which often terminates in necrosis. Trophic lesions in the viscera resulting from nervous diseases are mainly exemplified by rapidly-developed inflammation of the kidneys and bladder, accompanied with the discharge of ammoniacal and fœtid urine, containing blood or pus.

It is necessary now to point out the relation of the different portions of the nervous system to these consecutive lesions.

*a. Nerves.* Brown-Séquard attributes to reflex influence through centripetal nerves, the occurrence of cutaneous eruptions and muscular wasting in some cases; and many pathologists regard various forms of internal inflammation as being due to a similar influence. Certain lesions of motor nerves are followed by rapid reduction of electric contractility, and corresponding atrophy in the muscles which they supply. In connection with diseases of sensory nerves, the different forms of skin-eruption are liable to arise, which is well exemplified by herpes zoster; and also gangrene or atrophic lesions. In anæsthetic lepra atrophy of the muscles occurs; and erythematous patches appear on the skin, followed by the development of vesicles or bullæ, or by atrophy of



the cutaneous tissues, and in some cases leading to gangrene of the skin, of the deeper soft structures, or even of the bones.

*b. Spinal cord.* Numerous consecutive lesions are liable to follow various diseases of the spinal cord. Thus, in affections involving the posterior columns, such as locomotor ataxy, several skin-eruptions are liable to occur, owing, it is supposed by Charcot, to the implication of the nerve-fibres passing through the outer part of these columns before they emerge from the cord. These may also be observed in connection with a form of inflammation of the meninges of the cord, which leads to compression and irritation of its structures, as well as of the roots of the sensory nerves. Bullæ are sometimes developed in various parts in cases of vertebral caries. Bed-sores form at a very early period and with great rapidity in some cases of paraplegia, appearing only on the paralyzed parts, and especially over the sacral region, hence usually occupying a somewhat central and symmetrical site. They are also sometimes seen on the heels, the hips, and inside the knees. They occur more especially where pressure is exerted, but may be independent of this cause, or of any irritation by the excretions. At first patches of erythema are observed, with more or less infiltration and congestion of the subjacent tissues, which may involve the muscles and bones. Soon vesicles or bullæ form, and superficial sloughing takes place, gradually extending in area and depth, until a large surface may be destroyed, muscles and bones being sometimes involved, and even cavities opened up. These lesions are necessarily very grave, having been mainly observed in connection with hæmorrhage or inflammation occupying a considerable extent of the central portion of the spinal cord, and they may thus be associated with injuries to the spinal column, or with acute exacerbations and complications of chronic diseases of the cord. The lesions of the cutaneous tissues just considered are supposed to be due to implication of the posterior cornua and central grey matter of the spinal cord.

More or less rapid loss of electric contractility in muscles, with corresponding wasting, may follow many injuries and diseases of the spinal cord, if they involve certain special parts of the anterior cornua, including those groups of multipolar nerve-cells from which the motor fibres arise which proceed to the affected muscles. Even diseases which begin in other columns, such as locomotor ataxy, may by lateral extension involve the anterior cornua at different points, and thus induce disseminated atrophic changes in the muscles. They are also seen in connection with diffuse inflammation or hæmorrhage into the cord, but are particularly associated with infantile paralysis and corresponding conditions in the adult (polio-myelitis). Joint-affections are also sometimes observed in cases of injury or disease of the spine, and that form in which rapid destruction takes place chiefly occurs in cases of locomotor ataxy. The exact cause of the joint-lesion is not determined. Some authorities consider that they depend on an affection of the anterior cornua of the cord; Buzzard thinks that they are probably due to a lesion of the medulla oblongata. These arthropathies are not uncommonly combined with rapid muscular wasting, and it is supposed that the two classes of morbid changes are connected with lesions affecting the same parts of the cord, but this is not always the case.

It is in certain cases of paraplegia from spinal disease that acute inflammation of the kidneys and bladder takes place, supposed to be of the nature of a trophic lesion.

*c. Brain.* In cases of hemiplegia, erythematous, vesicular, or pustular eruptions occasionally occur on the paralyzed limbs. Acute bed-sores, similar to those described in connection with the spinal cord, may also occur in these cases, more particularly if the hemiplegia is due to cerebral hæmorrhage. They are almost always observed about the centre of the buttock on the paralyzed side, and at a lower level than those associated with paraplegia. These lesions in cerebral diseases are supposed to be due to irritation of the ganglia at the base of the brain. Acute muscular wasting, with loss of electric contractility, is rare in connection with cerebral disease, and is probably always due to some secondary descending lesion of the spinal cord, involving the motor nuclei. Inflammation of joints is occasionally noticed in hemiplegia, especially if due to softening, usually only affecting the articulations of the upper limb.

**PATHOLOGY.**—Having now noticed the relation of the chief trophic lesions to the different parts of the cerebro-spinal nervous system, it remains only to offer a few observations as to the explanation of their occurrence. They are not due to mere abolition or suspension of the action of the parts of the nervous system with which they are severally associated, but they are always dependent upon some kind of irritation, which leads to inflammation. Thus, in the case of the nerves, it is not complete nerve-division which causes the lesions, but those injuries by which the nerve is contused, punctured, incompletely divided, or lacerated; or diseases in which a nerve is inflamed, compressed, or stretched. Again, as regards the brain and spinal cord, some irritation of an inflammatory nature, in connection with the nerve-cells which are related to the different parts involved in different cases, induces the trophic changes, and not the mere severance of the nervous connection between them. What the precise cause of the lesions may be is at present undecided, but they have been supposed to be associated with certain trophic nerves; or to be excited through the vaso-motor nerves, which influence the vessels, leading to neuromyolytic hyperæmia, or setting up an irritative affection. Charcot thinks that some of the consecutive changes may be due to the transmission of pathological irritations from their original seat in a centrifugal direction along the nerve-filaments, these irritations thus reaching various structures, in which they originate the trophic lesions.

---

## CHAPTER LXXVI.

### ON THE LOCALIZATION OF NERVOUS DISEASES.

THE localization of the phenomena presented in various nervous diseases, whether functional or organic, has of late years come to occupy a prominent position with reference to the diagnosis of this class of affections, and it is now recognized as one of the objects to be kept in view in making such a diagnosis, to associate these phenomena with particular portions of the nervous system. The knowledge which renders this practicable is partly founded upon anatomical investigations, by which

the structure and connections of the different parts of the nervous system have been determined. It has been mainly obtained, however, as the result of physiological experiments, which have greatly increased our information as to the functions of this system, by indicating the effects of irritation and destruction of its several parts. Some assistance has been derived from observing what portions of the nerve-centres are deficient in their development when a limb or part of it is either congenitally wanting, or has been amputated. Lastly, the observation of the actual phenomena associated with injuries and pathological changes affecting the nervous structures has contributed much to our knowledge, and the mass of material thus accumulated for our guidance is being daily added to by several workers in this field of pathology. Within certain limits the diagnosis of the localization of nervous diseases is not only important, but essential; at the same time it must be remarked that the extreme accuracy in this respect which is now aimed at by many of those who make these affections their special study, is more a matter of scientific than of practical interest, and there are several circumstances which render it very difficult to arrive at a positive opinion as to the precise localization of the lesion in a large number of cases. It will be expedient, before discussing the individual diseases of the nervous system, to consider the subject in some detail, and to point out the main facts at present known which bear upon it.

1. In the first place it is always essential to distinguish between affections of the **brain, spinal cord, and individual nerves**; or, in other words, to determine whether a nervous disease is *cerebral, spinal, or peripheral*. To localize the mischief thus far is usually not difficult, but it must be borne in mind that the brain and spinal cord may be involved simultaneously; and that special nerves may be implicated along with the nerve-centres.

*a.* When the **brain** is affected, the special clinical phenomena which, in different combinations, are to be looked for as pointing to this portion of the nervous system, are as follows:—*a. Morbid sensations* referred to the head; and occasionally evident objective changes, affecting its size or shape. *b.* Disturbance of the *mental faculties*. *c.* Subjective disorders of the *special senses*. *d.* Signs of derangement of the functions of the *cranial nerves*, either in the direction of irritation or paralysis. *e. Motor phenomena* referred to the limbs, and sometimes to the body, usually unilateral in distribution; sometimes localized, but then as a rule limited to one side; occasionally general, affecting more or less both the upper and lower limbs, as well as the trunk. Hemiplegia is very common in cerebral diseases. *f.* Sometimes disorders of sensation of corresponding distribution, though these are by far less common, as well as much less in degree. *g.* Changes in the *eyes*, as observed with the ophthalmoscope. *h.* Certain *extrinsic* symptoms, especially cerebral vomiting, and obstinate constipation. When certain parts of the brain are involved, respiration and circulation are much affected; and curious symptoms are observed in particular cases.

*b.* Diseases of the **spinal cord** present considerable variations in their symptoms, according to the seat and extent of the mischief, but the usual phenomena noticed are of the following nature:—*a. Morbid sensations*, and occasionally abnormal *objective signs* referred to some portion of the spinal region, the sensations sometimes shooting from this region in various directions. *b. Motor disorders*, generally bilateral, and usually involving both legs and the lower part of the trunk, paraplegia



being a frequent symptom in spinal diseases, the muscles of the legs presenting at the same time marked derangements as regards their reflex and electric excitability, or the limbs becoming rigidly flexed, while they often show a tendency to rapid failure of nutrition. In some forms of spinal mischief the prominent motor derangement is impairment or loss of the power of muscular co-ordination. *c. Sensory disorders*, of similar distribution to those affecting motion, sensation as well as motor power being often completely lost in the lower limbs and lower part of the body. *d. Derangements affecting the bladder and rectum*, indicated by retention of urine and its consequences, or incontinence; and inability to expel the fæces, which may accumulate in large quantity, or involuntary escape of fæces. *e. Sexual disorders*, in the direction of undue sexual appetite or constant priapism; or of impairment or loss of sexual power and desire.

*c. Peripheral* nervous disease is indicated by the localization of the phenomena to the region in which the particular nerve or nerves affected are distributed; these phenomena being either connected with motion, sensation, or both, according to the functions which the involved nerves possess. It must not be forgotten, however, that local nervous symptoms may be the result of limited or commencing central disease, implicating the roots of nerves, or the portions of grey matter to which these severally correspond. In motor paralysis due to actual disease of a nerve or of its nucleus or cells of origin, the muscles show a rapid tendency towards wasting and loss of electric irritability, as has been pointed out in the previous chapter.

2. Having thus far indicated the general distinctions between the effects of lesions of the brain, spinal cord, and nerves respectively, it now remains to consider how far the clinical phenomena observed in different cases can be relied upon for the localization of diseases in the principal parts of the nerve-centres.

*a. With regard to the brain*, it is a well-known fact, that when hemiplegia occurs from any lesion involving one of the cerebral hemispheres, the paralysis is almost invariably on the side opposite the lesion, which is accounted for by the decussation of the motor tracts in the medulla oblongata. Brown-Séquard disputes the validity of this law, on the authority of 200 cases, in which the paralysis was on the same side as the disease, but, presuming that these cases were free from fallacy, they are, as has been shown by Ferrier and others, explicable on anatomical grounds, for direct paralysis may occur, in consequence of the motor tracts not decussating according to their usual arrangement. Therefore, it may be definitely stated that opposite paralysis is the rule in cerebral diseases, and thus far their localization can usually be determined with certainty, to the extent of referring the mischief to either lateral half of the brain.

When, however, we come to attempt to localize a lesion in particular parts of the brain, the question becomes much more difficult, and many eminent authorities maintain that it is impossible to do so with any certainty. There are several reasons to account for this. In the first place, it has as yet been by no means satisfactorily settled what are the precise functions of the several parts of the brain. Again, lesions frequently give rise not only to direct, but also to indirect symptoms, through their effects upon surrounding parts, as well as upon the entire brain. Moreover, lesions are often very extensive, or there may be more than one, in some cases even several distinct seats of disease being

observed. It is believed, too, that certain centres exert a compensatory action with regard to each other, so that when one is destroyed its functions may still be carried on by other centres. Secondary lesions are also liable to be set up, and these tend to complicate the phenomena observed. Notwithstanding these and other difficulties, however, it is possible in a considerable number of cases to refer symptoms to definite portions of the brain, and this we are enabled to do mainly through the experimental investigations of Fritsch and Hitzig, Ferrier, Burdon-Sanderson, Duret, and other physiologists; and the pathological and clinical investigations of Hughlings Jackson, Ogle, Charcot, Pitres, Broadbent, Gowers, Dreschfeld, Dickinson, and numerous other workers in this field of inquiry. In the following remarks it is intended to point out the phenomena associated with lesions of the principal parts of the brain, so far as they have been determined by these investigations.

The *convolutions* were formerly regarded as being entirely connected with psychical functions, so that, if there were any signs of mental disturbance, it was concluded that the cortex of the brain was involved. That this portion of the brain is concerned with these functions is unquestionably true, but considerable lesions may be met with here, causing marked objective symptoms without any mental disorder, and this is attributed to the fact that when one hemisphere is destroyed, the mental processes may be carried on by the opposite one. On the other hand, lesions which cause mental derangement need not be accompanied with any objective phenomena. In cases of insanity it is presumed that the brain is always diseased, but often no distinct lesions can be discovered; and although various morbid changes have been observed in these cases, no definite relation has been found to exist between any particular lesion, as regards either its nature or locality, and any form of mental disorder. Of late years it has been shown that different regions of the convolutions have distinct functions, by which lesions affecting them can be localized. The *præ-frontal lobe* or *antero-frontal region*, roughly bounded by the coronal suture of the skull, may be the seat of extensive laceration or disease, either suddenly or gradually induced, without causing any objective phenomena, and indeed without any evident disturbance, either bodily or mental, especially if the lesion be unilateral; but in other cases, chiefly where the mischief is bilateral, psychical symptoms have been noticed, and marked mental deficiency has been found in connection with arrested development or atrophy of this lobe. By most authorities, as has been already stated, the faculty of articulate language is now regarded as being lodged in the posterior extremity of the third *left frontal convolution*, and hence any lesion involving this part is accompanied with aphasia, which is most frequently associated with right hemiplegia. If both sides are involved, there is also paralysis of articulation. The cerebral cortex presents a *motor zone*, destructive or irritative lesions of which are indicated respectively by muscular paralysis or spasm. This zone is said to include the bases of the three *frontal convolutions*, with those bounding the fissure of Rolando, namely, the *ascending frontal*, the *ascending parietal*, the *postero-parietal lobule*, and the internal surface of these convolutions, or so-called *paracentral lobule*. If a destructive lesion affects this motor zone extensively, it causes complete hemiplegia of the opposite side; it is, however, differentiated into centres for movements of the arm, leg, facial muscles, eyes, &c., and when the lesions are corre-



spondingly limited, various monoplegiæ are induced. The same remark applies to spasm, and if a convulsive seizure always begins in the same way, or if monospasms are frequently confined to one limb or a particular group of muscles, and especially if corresponding paralysis becomes subsequently developed, a lesion may be localized in a certain part of the opposite hemisphere with much precision. With respect to the particular centres for each part, the chief are indicated by Ferrier as follows:—for the arm, upper third of ascending frontal convolution; hand and wrist, upper parietal; leg and foot, postero-parietal lobule; facial muscles, middle third of ascending frontal and base of second frontal; mouth and tongue, lower third of ascending frontal and base of third frontal; lateral movement of head and eyes, posterior third of upper frontal convolution and corresponding part of second frontal.

How far it may be possible to diagnose between hemiplegia due to general destruction of the motor area of the cortex, or to lesions situated in the interior of the brain, is a matter of doubt, and in many cases such a diagnosis cannot be made, merely judging by the clinical phenomena observed. The main points bearing upon this subject, as given in Ferrier's work, are as follows:—It is said that there is less difference in temperature between the paralyzed and non-paralyzed sides when the paralysis depends upon cortical than on central disease, and that it subsides more rapidly. Cortical lesions are most frequently indicated by fractional or dissociated paralysis, or by a succession of dissociated paralyzes and monoplegiæ. A complete hemiplegia often resolves itself into a monoplegia; or a monoplegia becomes a hemiplegia by progressive advance of the disease to other motor centres, which is said to be very significant. Again, a monoplegia is very often associated with monospasm or early rigidity of the paralyzed limb, or of the muscles governed by the centres surrounding the lesion. Sometimes the paralyzed limb remains motionless, while convulsions occur in the others. Cortical paralysis is frequently erratic and transitory, more especially in connection with superficial or meningo-cerebritis. Lesions of the cortex are more frequently accompanied with localized pains in the head; and Ferrier has observed that even when pain is not spontaneously complained of, it may be brought out by percussion over the seat of lesion.

The cortex is also said to have a *sensory* zone, supposed to be localized in the *parieto-temporal lobe*, and to be differentiated for different sensations, as follows:—tactile sensation, hippocampal region; smell and taste, lower part of temporo-sphenoidal lobe; sight, angular gyrus and supramarginal lobule; hearing, superior temporo-sphenoidal convolution. From a pathological point of view there is thus far no direct evidence of this localization of sensory centres; but Ferrier holds that this must be looked for in connection with *bilateral* destructive lesions. He is also of opinion that sensory hallucinations in cases of insanity, as well as certain subjective sensations which usher in some epileptic attacks, are due to a morbid irritation of the cortical sensory centres.

No symptoms have as yet been definitely associated with lesions of the *occipital lobes*.

Coming now to the ganglia within the brain, and their surrounding medullary substance, the *corpus striatum* and *anterior two-thirds of the internal capsule* are concerned with motion, and a lesion affecting this part of the brain induces hemiplegia on the opposite side of the body, of the ordinary type. If the lesion is sudden, loss of consciousness and



impairment of sensation usually occur, of a temporary nature; but these symptoms are the result of its indirect effects. When it involves only the *nucleus caudatus* or the *nucleus lenticularis*, it is believed that the paralysis is merely temporary, and is comparatively slight in the former case. Hughlings Jackson is of opinion that the arm suffers less, and the leg more, the further back the lesion is situated. Should the anterior two-thirds of the internal capsule be ruptured, hemiplegia is marked and permanent, and it is only under such circumstances that secondary degeneration of the motor tracts, with consequent permanent rigidity, takes place.

The *optic thalamus* and *posterior third of the internal capsule* are concerned with sensation. The researches of Charcot and others have shown that destructive lesions of the internal capsule, external to the optic thalamus, cause hemi-anæsthesia on the opposite side of the body.

Lesions of the *corpora quadrigemina* are very rarely limited to these bodies, but they are liable to be involved along with neighbouring structures. If one of them is destroyed, vision is lost on the opposite side, this being due to destruction of the *anterior tubercle*; conversely, if the eye is destroyed on one side, the opposite tubercle becomes atrophied. The motions of the iris are also paralyzed if the lesion extends deeply. Equilibration and co-ordination are likewise disturbed; but these disorders have been attributed to lesions of the subjacent tracts, especially the *superficial cerebellar peduncle*. Irritation of both corpora quadrigemina on one side gives rise to dilatation of the pupil, and hemi-opisthotonos of the opposite side, which becomes general if the irritation is prolonged or bilateral, the head being retracted and the legs extended, trismus being also very marked. Unilateral destruction is said to cause incurvation of the trunk, and gyration to the side of lesion.

When either *crus cerebri* is destroyed, the consequences are paralysis of motion and sensation on the opposite side, sensation being especially affected, with marked vaso-motor paralysis, and a consequent rise of temperature of  $2^{\circ}$  or  $3^{\circ}$  in the paralyzed limbs; if the lower part of the crus is involved, the third nerve is implicated, there being hence oculomotor paralysis on the same side as the injury.

Before quitting the cerebrum, it may be well to notice the important aid which the ophthalmoscope may afford in localizing diseases in this part of the brain; and Dr. Gowers has also drawn my particular attention to the necessity of testing the field of vision for this purpose. For instance, partial double hemiopia is often present when unsuspected by the patient. This shows disease of the optic fibres on the side opposite to the hemiopia, behind the commissure; or of their terminations in the central ganglia, corpora geniculata, or optic thalamus; so that when the patient cannot see objects to his right with either eye the lesion is in the left optic tract. Hence, when other nervous symptoms are bilateral, this hemiopia may afford the only indication as to the side which is affected, and it may be the means of still further localizing the mischief to that part of the hemisphere which is contiguous to the optic tract and ganglia. If the lesion is situated in the commissure itself, which is very rare, the loss of vision is not on the same side in both eyes, but the two outer or two inner fields are lost.

It is not easy to determine definitely the symptoms directly due to disease of the *cerebellum*, so many of the phenomena which are observed being indirectly induced. The chief symptoms are due to a peculiar

disorder of equilibrium. There is no true motor paralysis, and although opposite hemiplegia is not unusual in cerebellar disease, this is probably the result of its indirect effects upon the subjacent motor tracts, which decussate at the pyramids. When any attempt at locomotion is made, the patient exhibits a reeling or staggering gait, with a constant tendency to stumble or fall over any obstacle or on moving hurriedly. The movements are not due to any actual want of co-ordination, but are such as would be made to preserve equilibrium, or to prevent the patient from falling. Sensation is not affected, unless it be indirectly. Nystagmus and strabismus have been noticed, more especially in connection with disease of the cerebellar peduncles. When the middle lobe of the cerebellum is the seat of hæmorrhage, vascular excitement of the sexual organs has been frequently observed, indicated in males by marked priapism, and it has been supposed that the cerebellum or, according to some writers, its middle lobe, is connected with the sexual instinct. This is not the case, however, according to the best authorities of the present day, who regard the sexual excitement as an indirect symptom, due to irritation of the posterior surface of the medulla and pons. Pain in the back of the head is often present in cerebellar disease; and vomiting is a very frequent symptom, probably on account of the indirect effect of the disease upon the medulla. In connection with lesions of the *superior* and *middle cerebellar peduncles*, the special phenomena observed are a rotatory distortion of the head and trunk, usually towards the side of the lesion; and a peculiar distortion of the optic axes, the eye on the side of the lesion being directed downwards and inwards, the opposite one upwards and outwards. Much, however, will depend upon the exact seat of the lesion, and whether it is of a destructive or irritative character.

A sudden lesion in connection with the *pons Varolii* and *medulla oblongata* usually proves speedily fatal, owing to the interference with the functions essential to life thus induced, namely, respiration and circulation. Not only do the motor and sensory tracts meet and blend in this portion of the nerve-centres, but several nerves have their nuclei of origin here. Hence the phenomena observed are liable to considerable variation in character and combination, according to the exact localization of the lesion, for not only may the different tracts be involved, but also either of the nerve-nuclei may be implicated, or the nerves after their emergence from these nuclei, whether their function be motor, sensory, or special. As before stated, it is in connection with lesions involving this region that so-called *cross-paralysis* occurs. In other cases there may be general paralysis of the limbs; or paralysis of one arm and both legs, or *vice-versâ*. The muscles of the eyes are variously affected; and one or both facial nerves may be involved, according to the seat of the disease. The fifth and other sensory nerves may also be implicated, and thus various degrees of impairment of sensation induced, usually irregular in distribution. Difficulties connected with mastication, deglutition, phonation, articulation, respiration, circulation, or the power of control over the bladder and rectum, are observed in different combinations; and it is to interference with certain of these functions that the great danger arising from diseases implicating the pons Varolii and medulla oblongata is due.

*b.* With respect to lesions of the *spinal cord*, if this is destroyed in its entire thickness, complete motor and sensory paralysis in the parts below the seat of lesion will ensue. Generally this involves only the legs and

the lower part of the trunk, but if the mischief is situated at or above the cervical enlargement, the arms will also be paralyzed; while if it is high up in the cervical region, the respiratory muscles and diaphragm become involved, and death will speedily ensue from asphyxia. With regard to micturition, a lesion in the cervical or upper dorsal region generally causes a difficulty in performing the act, or even retention of urine, owing to spasm of the sphincter; if it should occupy the lower dorsal or lumbar region, the sphincter is paralyzed, with consequent involuntary escape of urine. Constipation is usually marked; and defæcation may be involuntary. In some cases the destruction of the cord is not complete, and hence there is only impairment of the motor and sensory functions below the seat of disease. Again, it not uncommonly happens that motor power is entirely lost, while sensation remains. This is due to the fact that the sensory tracts are confined to the posterior cornua and the rest of the grey matter behind the central canal of the cord; therefore, in diseases of its periphery, or of the surrounding membranes, the sensory tracts are so situated that they may escape, while it also appears that a very narrow thread of grey matter is sufficient to keep up the connection between the peripheral parts and the sensory centres.

Coming now to the consideration of lesions involving special tracts in the spinal cord, if one *lateral half* is destroyed in its entire thickness, but to a limited extent longitudinally, this causes complete motor paralysis below the lesion *on the same side*; sensory paralysis *on the opposite side*, the upper limit of which is indicated by a more or less distinct line of demarcation. This precise limitation of a disease to one half of the spinal cord is, however, very rare, but the lesion may even be limited to a particular tract, and thus affect motion or sensation only. It only happens extremely rarely, if ever, that a lesion is limited to the *posterior part of the central region of the chord*, so as to induce bilateral sensory paralysis, motion being unaffected. One particular form of lesion tends to limit itself to special tracts, and consequently very striking phenomena are produced. If the mischief is confined to the *posterior columns*, or, according to Charcot, to the *outer bands* of these columns, which are contiguous to the inner and posterior aspects of the posterior cornua and the roots of the sensory nerves, there is loss of co-ordinating power over the muscles below the seat of disease, without any actual paralysis, as in locomotor ataxy. If the *lateral columns* are alone involved, more especially the white matter which lies behind a horizontal line drawn laterally through the median canal, motor paralysis occurs in the parts below, the muscles also tending to become at first tremulous, and ultimately more or less rigid and contracted. A lesion may be limited to the groups of large cells constituting the motor nuclei in the anterior cornua, and then paralysis only affects those muscles supplied by nerves arising from these diseased spots, this being frequently followed by rapid loss of electric contractility and wasting. The relations of other trophic lesions to particular parts of the spinal cord have been already pointed out. It will be readily understood that in the case of irritative lesions involving the cord, the distribution of the phenomena observed will be the same as in connection with destructive lesions, according to the part implicated, these phenomena only differing in their kind.

With regard to the localization of the motor roots of the spinal cord, mention must be made of the experiments of Ferrier and Yeo (Proc.



Roy. Soc., 1881), who stimulated by the faradic current the peripheral ends of the divided motor roots of the brachial and lumbo-sacral plexuses, and thereby found that distinct groups of muscles in the limbs were caused to contract; for instance, stimulation of the 4th cervical motor root put into action the deltoid, rhomboid, supra- and infra-spinatus, biceps, brachialis anticus, supinator longus, extensors of wrist and fingers, and so on with the other roots of the plexus; while in the lumbo-sacral plexus the second sacral nerve only caused contraction of the intrinsic muscles of the foot. Ferrier considers that these observations may explain the way in which groups of muscles are affected in diseases of the cells of the anterior cornua, such as occur in infantile paralysis, adult spinal paralysis, progressive muscular atrophy; the groups of muscles being affected, as the anterior cornua of different segments of the cord are attacked.

---

## CHAPTER LXXVII.

### HEMICRANIA—MIGRAINE OR MEGRIM— SICK-HEADACHE.

ÆTIOLOGY AND PATHOLOGY.—Most authorities regard the complaint known as *migraine* or *sick-headache* as being quite independent of any morbid state of the alimentary canal, and as essentially a nervous affection. Allbutt, however, considers that derangements of the abdominal viscera have an important influence in giving rise to migraine. The chief views as to the pathology of this malady are as follows:—1. That it is a form of neuralgia of the ophthalmic or occipital nerve; or of the filaments distributed to the dura mater. Some regard this neuralgia as being merely due to peripheral causes, but Anstie, who was strongly in favour of the theory that migraine is a form of *trigeminal neuralgia*, attributed it primarily to a morbid condition at the root of the fifth nerve in the medulla oblongata, its central nucleus in this part being the seat of atrophic molecular irritation, which has an unusually strong tendency to communicate itself to the neighbouring and closely connected nucleus of the vagus. 2. That the complaint is due to vaso-motor disturbance affecting the vessels of the head, produced through the sympathetic nerve. Latham considers that in the premonitory stage of sick-headache the small arteries are contracted, owing to excitement of the vaso-motor nerves, which depends upon a weakening of the controlling power exercised over them by the cerebro-spinal system, this probably originating in the medulla oblongata. During the stage of headache it is supposed that the nerves become paralyzed, and the vessels are consequently dilated; and Latham is of opinion that this paralysis is the result of depression following the previous excitement. 3. Liveing, in his admirable work on this subject, has advanced the hypothesis that the paroxysms of migraine are due to “nerve-storms, traversing more or less of the sensory tract from the optic thalami to the ganglia of the vagus, or else radiating in the same tract from a focus in the neighbourhood of the quadrigeminal bodies.”

The chief *predisposing causes* of migraine are the female sex, attacks being peculiarly liable to occur about the menstrual periods; hereditary tendency to the complaint, or to various other neuroses; anæmia and general want of tone; and a nervous, excitable temperament. A paroxysm often comes on without any obvious *exciting cause*, but it may follow errors in diet; exposure to the sun; breathing vitiated air; undue mental excitement or effort; fatigue, especially when combined with fasting; excessive sexual indulgence; and various other causes which lead to physical or nervous depression. Sometimes it results from some disturbance affecting the sight or hearing; and it may be brought on by straining the eyes, as after prolonged reading or sewing.

**SYMPTOMS.**—Sick-headache is characterized by periodic attacks, which usually commence during the period of bodily development, in persons from 15 to 25 years old; as a rule becoming more frequent and severe up to a certain time; but tending to diminish in frequency, or even to cease altogether in advanced age, particularly after the change of life in women.

An attack of migraine is generally ushered in by some *premonitory* symptoms, which are mostly observed when the patient awakens in the morning, such as a sense of depression, heaviness, or general uneasiness; vertigo; disturbed vision, especially a wavy glimmering; chilliness and shuddering; coldness of the hands and feet; tingling in the arm or tongue; irritability of temper; yawning, gaping, or sighing; disorder of speech or hearing; or disinclination for food, with a slimy taste. Soon the pain commences, and speedily becomes intense. Almost always it is unilateral, being felt chiefly in the supra-orbital region or sometimes within the orbit, but not uncommonly extending over the whole side of the head. The precise character of the pain varies much in different cases, but it is generally accompanied with a sensation of throbbing. Pressure on the carotid artery usually diminishes its intensity. There is increased local heat; and in many cases redness of the conjunctiva is observed, with an excessive flow of tears. During a severe paroxysm the patient usually takes to bed, feels extremely depressed and low, dreads every disturbance, begs to be left at rest, and is especially sensitive to light and noise. The pulse is frequently slow and soft. The pupils are contracted. When the suffering reaches its height, nausea and bilious vomiting generally set in, aggravating the pain, but afterwards this gradually diminishes, and the patient usually falls asleep. Anstie remarked that this vomiting is not ordinarily remedial, but that it “merely indicates the lowest point of nervous depression.” Vomiting may be directly beneficial, however, if there is much undigested food in the stomach. On awaking from sleep, the patient finds as a rule that the pain has ceased, but frequently complains of a little superficial tenderness for a day or two, and feels out of sorts. The *duration* of an attack is very variable in different cases, but it does not commonly last more than twenty-four hours, though it may go on for two or three days or more.

**TREATMENT.**—During the *premonitory* stage of an attack of migraine, if this is evident, certain measures may be adopted with the view of preventing or mitigating the subsequent symptoms. The patient should at once retire into a quiet darkened room, and lie down on the side which former experience has shown to be usually the seat of pain, with the head low, the extremities being kept warm. Very many remedies have been recommended for sick-headache, but their usefulness differs much

in different cases. The most important are *diffusible stimulants*, such as a little brandy or sherry and soda-water, champagne, or spirits of ammonia; a cup of simple strong tea or coffee; hydrate of chloral; croton-chloral; tincture of cannabis indica; bromide of potassium; nitrate of ammonium; caffeine, either internally or by subcutaneous injection; nitro-glycerine and guarana powder, which consists of the powdered seeds of the *Paulinia sorbilis*. The drug last mentioned is given in doses of gr. x-xv, but there is much contradiction in the statements of different observers as to its efficacy, and in my own experience it has had very different effects. The application of a weak galvanic current is sometimes useful. Anstie recommended a warm foot-bath containing mustard, and for the patient to breathe the steam from this at the same time. In some cases I have found the administration of a simple *emetic* such as sulphate of zinc, decidedly beneficial. Much relief often results from tightly binding the head with a wet bandage. Probably the steady application of ice, the cold douche, or the ether-spray might be serviceable in some cases. During the height of an attack it is best to leave the patient in perfect quiet, and not to give food or anything else. In the *intervals* many of the measures recommended for the treatment of neuralgia are indicated, and among the most useful medicines may be mentioned strychnine, arsenic, quinine, and bromide of potassium. Tincture of cannabis indica  $\mathfrak{m}$  v-x, thrice daily, has been found beneficial by several observers. Tincture of *actæa racemosa* has also been recommended. It is requisite to attend to the state of the alimentary canal; and to avoid all causes which are likely to give rise to an attack of migraine.

## CHAPTER LXXVIII.

## EPILEPSY—FALLING SICKNESS.

**ÆTIOLOGY AND PATHOLOGY.**—Epilepsy is the name given to a group of cases characterized by fits of loss of consciousness with convulsive seizures. It cannot be properly regarded as a distinct disease, as such fits may occur under a variety of conditions, including the following:—  
 1. In connection with various *organic diseases of the brain or its membranes*, for example, meningitis, hydrocephalus, tumour, embolism, softening, or syphilitic disease. 2. As the result of morbid conditions of the *skull*, which lead to pressure upon, or irritation of the brain, such as exostosis, a fracture with projecting spicula of bone, or necrosis. 3. From disorders of the *cerebral circulation*, leading to congestion or anæmia. 4. In certain forms of *blood-poisoning*, for example, uræmia and saturnism. 5. As a so-called *functional* affection, to which some writers specially apply the term *epilepsy*. In cases belonging to this group, although organic changes have been described in the brain and meninges, these are commonly absent, and when present they are probably the effects of repeated fits, rather than the cause of the epileptic phenomena. One view as to the nature of this functional variety of epilepsy is that it depends upon some nutritive change in the medulla oblongata, upper



part of the cord, and vaso-motor centres, which leads to excessive and perverted action in these parts, inducing sudden contraction of the vessels of the brain and cord, as well as of those supplying the muscles of the face, pharynx, larynx, respiratory apparatus, and limbs, to which all the subsequent phenomena of the fit may be traced. According to another theory a sudden discharge of nerve-force takes place from an immense number of nerve cells at the beginning of a fit, which leads to shock, and the convulsions, like other forms of this disorder, are the result of a "discharging lesion." (Hughlings Jackson.) The remote causes to which epilepsy, which is independent of some obvious local cause, has been attributed are:—1. *Mental disturbance*, especially emotional, for example, a sudden fright; prolonged grief or anxiety; and also excessive mental work, or undue forcing of the brain in childhood. 2. *Physical influences* affecting the brain, as a blow or fall on the head, or sunstroke. 3. Certain conditions affecting the state of the *blood and general system*, and thus influencing the nutrition of the brain, such as syphilis, rheumatism, gout, acute specific diseases, pneumonia, pregnancy. 4. *Reflex irritation*, as from dentition, worms, uterine and ovarian disturbances, sexual excesses or masturbation. Great prominence has been given by some writers to the sexual functions as a cause of epilepsy. 5. *Hereditary taint*. Undoubtedly this has an important influence in the causation of epilepsy, especially when it comes from the mother's side. In a considerable proportion of cases either epilepsy or some allied neurosis is prevalent in the family. Probably intemperance in the parents, syphilis, or a fright to the mother while the child is *in utero*, may be the means of inducing a congenital tendency to epilepsy. The complaint is developed at an earlier age in hereditary cases. 6. *Idiopathic*. This term applies to cases in which no obvious cause can be made out. Age requires special notice as a *predisposing cause* of epilepsy. In the great majority of cases the disease is developed between 10 and 20 years of age, and especially at or about the period of puberty. Sex does not seem to have any particular influence in young persons, but it is said that in persons of somewhat advanced age the proportion of cases of epilepsy is greater in women. It rarely happens that any immediate *exciting cause* of a fit can be made out.

**SYMPTOMS.**—Attacks of true epilepsy assume one of two forms, of each of which it will be necessary to describe the typical characters.

1. **Epilepsia mitior—Petit mal.**—This form is characterized by sudden and complete loss of consciousness, coming on without any warning, and lasting only for an instant, or at most for a few seconds; accompanied with slight pallor and subsequent duskiess of the face; loss of all expression; dilated pupils; and often, but not always, slight spasmodic movements affecting the face, respiratory muscles, or limbs. If the individual is speaking, he stops in the middle of a sentence, and generally appears to hold his breath. Voluntary movements cease, but automatic actions go on as a rule, such as those which are necessary for standing, sitting, or riding. In some cases there is not absolute unconsciousness, and there may be but a feeling of sudden vertigo—*vertige épileptique*—which causes the patient to cling to the nearest object. After the attack there is some degree of mental confusion, lasting but a few minutes, during which the patient often says and does things which he afterwards forgets and denies. Slight squinting may be noticed, or a feeling of choking may be experienced. On recovery there is no recollection of what has happened. These attacks may be preceded by

an *aura epileptica*, and they may be premonitory of severe epileptic seizures, or both forms may occur in the same subject. They are frequently followed by serious mental changes, ending in dementia or mania.

2. *Epilepsia gravior*.—*Haut mal*.—The advent of a fit of epilepsy is in a large proportion of cases indicated by *premonitory* symptoms, varying in duration from an instant to several hours or days. They present great variety, being either subjective or objective, and commonly of a nervous character, affecting the mental condition; general sensation or the special senses; the muscular system; or the vaso-motor nerves. Sometimes they are extrinsic, such as vomiting, obstinate constipation, sallowness of the skin, or foetid secretions. The so-called *aura epileptica* requires a few words of special comment. This is a peculiar sensation, well-known to the patient, which in many cases immediately precedes a fit, generally appearing to start from the distal end of a limb, especially the arm, and to run up towards the head, on reaching which part the seizure takes place. Sometimes it only extends from the elbow to the shoulder, or from the leg to the epigastrium, and has been stated to pass from the testicle or uterus to the throat. The sensation varies in its exact character, but has been compared to a stream of cold or hot air, and is frequently not unpleasant. It is curious that its ascent may sometimes be stopped, and the fit prevented, by pressure applied above the point from which the sensation starts, and this pressure need not be so powerful as to stop the circulation; occasionally a similar result will happen when the pressure is applied to the opposite arm.

*Actual attack*.—Three marked stages characterize an epileptic fit:—

*Stage I*.—The phenomena of this stage are a single, peculiarly disagreeable cry, yell, or moan in many cases, but not in all; immediately followed by absolute and instantaneous loss of consciousness, the patient falling anywhere, or often appearing to be thrown down; a violent tonic spasm of the muscles throughout the body, beginning generally about the face and neck of one side, the whole muscular system being in a state of extreme rigidity and strain, but not equally so, and hence there is a hideous distortion of the features, limbs, and body, the latter being drawn to one side, and the neck twisted so that the face looks over that shoulder, while the teeth are firmly clenched, the eyes wide open, and the eyeballs turned towards the same side; stoppage of respiration, usually complete, owing to the spasm of the muscles; change in colour of the face almost invariably, in many cases deadly pallor being observed at first, followed by duskiness or lividity, or this may be present from the commencement, or be preceded by florid or dull redness; marked dilatation of the pupils; and feebleness or cessation of the pulse at the wrist, due to the muscular spasm, for the heart acts forcibly, and the carotids throb violently. Practically these phenomena may be considered as simultaneous, the whole stage not lasting longer than from two or three to thirty or forty seconds.

*Stage II*.—The transition to this stage is abrupt, and is indicated by restoration of breathing, the respiratory muscles becoming relaxed, and the retained air being expelled. Unconsciousness continues, but severe clonic spasms take the place of the tonic rigidity, usually beginning with twitchings about the face or sometimes in the limbs, but soon extending more or less over the whole body, though often more violent on one side than the other. As a rule the side most affected in the first stage is convulsed more than the other; the eye, mouth, and head being drawn to that side, while the arm and leg of the same side are usually the more

convulsed. From these spasms originate the phenomena of this stage, namely, hideous distortion and convulsive movements of the features and eyeballs; forcible closure and champing of the jaws, causing grating of the teeth, foaming at the mouth, partly due to formation of excess of secretion, which is blown out of the mouth, and biting of the tongue or cheek, the froth being therefore often bloody; violent convulsive movements of the body and limbs, which are thrown about and twisted in all directions, the fingers being generally bent and the thumb pressed into the palm; alternate dilatation and contraction of the pupils; laboured, panting, and irregularly convulsive respiratory movements, often attended with gurgling sounds due to mucus in the trachea; increasing duskiess or lividity and turgidity of the face, tongue, and body generally, with distension of the veins, some of the smaller vessels sometimes giving way, thus giving rise to extensive petechiæ about the face or head; profuse perspiration, the sweat being sometimes peculiarly fœtid; tumultuous action of the heart, with throbbing of the large arteries, though the radial pulse is often weak; involuntary discharge of urine, fæces, or semen; and frequently rumbling noises in the intestines, vomiting, or hiccup. The average duration is said to be from  $4\frac{1}{2}$  to  $5\frac{1}{2}$  minutes, but it may vary from a few seconds to 10 minutes. The clonic spasms are believed to be the result of the stoppage of respiration in the previous stage, with consequent asphyxia; or more probably of the initial tonic spasm.

*Stage III.*—There is a gradual return to consciousness, with cessation of the spasmodic movements. The patient looks around with a bewildered, alarmed, or sad expression, and often attempts to get up or to speak, but some few minutes usually elapse before consciousness is completely restored. The heart still acts vigorously, and the skin is bathed in sweat. Vomiting often takes place. A large quantity of pale and watery urine may be passed, containing excess of urea and urates, or sometimes abundant phosphates; and it is said that a trace of sugar has been found after a severe epileptic fit. After return to consciousness the patient feels usually very exhausted and sleepy, as well as mentally confused, and complains of headache. In many cases, but by no means in all, he falls into a state of heavy sleep or stupor, almost amounting to coma, attended with a stertorous noise in breathing, from which it is difficult or impossible to rouse him, and which lasts for a variable time, sometimes passing into natural sleep. The muscles are relaxed, but present occasional twitchings or slight spasmodic movements. The face generally remains more or less dusky for some time, and the petechiæ continue visible. The patient is often languid and out of sorts for some days after a fit.

The *frequency* and *severity* of epileptic fits vary much in different cases. In a good many a tolerably marked periodicity is observed. In few instances does the interval extend beyond a month. The seizures are more frequent as a rule in severe cases, and they tend to increase in frequency and intensity as the disease advances. Not uncommonly two or more fits occur in succession, followed by a period of freedom from attacks. They are liable to come on by night as well as by day; and nocturnal fits of epilepsy may occur without the patient being in the least aware that they have taken place.

The *general* state of the patient also differs considerably. There is rarely perfect health, especially after epilepsy has existed for some time. Many epileptic patients suffer from headache or giddiness and



various other symptoms, the general system and digestive organs being also out of condition. The *mental* faculties become more or less weakened in most cases, and this may end in complete dementia or dangerous epileptic insanity. Sometimes partial and limited paralysis, twitchings, curious movements, disorders affecting sensation or the special senses, and other nervous phenomena are observed. As *complications* of epileptic fits, coma resulting from injury to the head, apoplexy, or meningitis may arise.

**Epileptiform seizures.**—This term has been applied by Dr. Hughlings Jackson to attacks of an epileptic character, distinct from those of epilepsy proper, and the following are some of the prominent points to which this observer has called attention:—In all cases the spasm begins unilaterally in the hand, face, or foot. In its range it varies widely, but arbitrarily. Dr. Jackson makes three ranges, namely, monospasm; hemispasm; or where the other side of the body, or part of it, is convulsed. The spasm progresses in a definite direction. When limited or nearly limited to an arm, it usually goes up. If in hemispasm the spasm begins in the hand, it goes up the arm and down the leg; if it begins in the foot, it goes up the leg, and, with many exceptions, down the arm. Dr. Jackson thinks that the more suddenly the spasm starts, and the more rapidly it begins to spread, the greater the range ultimately attained and the shorter the seizure. Consciousness is usually unaffected in limited convulsion of a limb, side of face, or even of one side of the body. Roughly speaking, consciousness usually ceases when the eyes and head begin to turn to the side first convulsed. The more sudden and rapid the spasm, the less is the range attained before consciousness is lost. In the severest epileptiform seizures consciousness ceases late in the paroxysm, while in severe epilepsy proper it ceases first thing or very early. Temporary paralysis or partial aphasia may follow the seizures; as well as temporary increased patellar tendon-reflex, and ankle-clonus on the affected side. When the fits are always of the same style, the inference is that there is persistent change in some cells in one locality, and that they occasionally attain high irritability and discharge excessively. They are due to different pathological causes, but when disease exists, it is usually in the so-called motor region.

**DIAGNOSIS.**—The chief conditions from which idiopathic epilepsy may have to be distinguished are hysteria; reflex convulsions; epileptiform attacks due to cerebral organic diseases, uræmia, or chronic alcoholism; syncope; Menière's disease; and feigned epilepsy. Some of these will be alluded to in future chapters. With regard to hysteria, a condition named *hystero-epilepsy* is now recognized, which presents a combination of the phenomena of both diseases. Attacks of *petit mal* have to be distinguished from fits of syncope; and from vertiginous attacks in cases of Menière's disease.

**PROGNOSIS.**—A cautious opinion should always be given in cases of epilepsy as to the final issue. Very rarely does a fit end fatally, but this might happen in consequence of some complication. As to the curability or improvement of the disease, the favourable prognostic circumstances are its being recent, or due to some definite cause which can be removed; the patient being very young and a male; absence of hereditary taint; the mind being unaffected. Inherited epilepsy is very rarely cured; and if the attacks have begun in early childhood from some reflex irritation, and have lasted many years, the prognosis is also

very unfavourable. The mental faculties are more liable to become affected in females; in persons who are strong and healthy; when the disease begins late in life; when the fits occur in rapid succession, with attacks of "petit mal"; and, it is said, when the spasms are not marked during the fit, and there is little or no subsequent coma.

**TREATMENT.**—1. **During a fit.**—It is best not to interfere actively during an epileptic seizure in most cases, merely attending to the matters mentioned when speaking of convulsions in general, preventing injury, but not holding the patient forcibly, and putting something between the teeth. If the fit does not soon cease, water may be dashed over the face and chest, and should it become dangerously prolonged, such measures might be had recourse to as the application of sinapisms to various parts; a warm bath, with cold affusion while the patient is in it; ice to the spine or head; stimulant enemata; electricity; local removal of blood from about the head; or even venesection, should there be great danger of asphyxia. After a fit the patient should be placed in a comfortable position, kept quiet, and allowed to sleep.

2. **In the intervals.**—There are certain well-defined principles to be followed in the management of an epileptic patient. *a.* It is requisite to look for and remove any obvious *cause* of epilepsy. Thus, should there be any local cause of irritation, such as a foreign body irritating a nerve, or worms, this must be got rid of. Further, as epilepsy may depend on some central organic mischief, careful investigation is required in order, if possible, to find out and treat any such disease, especially if due to syphilis, when iodide of potassium is of the greatest service. Any constitutional condition, as rickets or tuberculosis, must be attended to. *b.* The *general management* of an epileptic patient is highly important. He should have a nutritious, but light and digestible diet; take moderate daily exercise in the open air; be surrounded by proper hygienic conditions; avoid much mental work, especially in the case of children, who should be kept from school, though if the general health is good, older patients may follow some light occupation; have cold or tepid sponging daily, with friction afterwards; check any vicious habit, such as excessive venery, masturbation, or intemperance; and take a sufficient amount of sleep, the head being well-raised in bed. It is necessary to regulate the digestive functions, especially avoiding constipation, but only mild *aperients* should be used. Iron if there is anæmia, quinine, strychnine, arsenic, and other nervine or general *tonics* are often of service. Cod-liver oil is also frequently of much value. Many epileptics require constant watching, and all need more or less supervision; above all they must not be allowed to go into positions where they would be in danger from falling, or near a fire or water. Epileptic patients decidedly ought not to marry.

3. **Specific treatment.**—Innumerable specifics have been brought forward for the cure of epilepsy. Of these the only drugs that deserve special mention are bromides, especially bromide of potassium and ammonium; nitrite of sodium (introduced by Dr. Law); belladonna or atropine; stramonium; conium; extract or tincture of cannabis indica; preparations of zinc, especially the oxide, the sulphate in gradually increasing doses up to 10, 15, 20, or more grains thrice daily, the valerianate, the acetate, and the bromide; ammonio-sulphate of copper; nitrate of silver in minute doses; opium in small quantities; and chloroform by inhalation, not in sufficient quantity to induce complete insensibility,



either systematically employed at certain intervals daily, or only administered when there are signs of an impending fit. Nitrite of amyl and nitro-glycerine have also been recommended. Doubtless all these agents prove serviceable in different cases, and sometimes they may be usefully combined, as, for example, belladonna with sulphate of zinc. Bromide of potassium has been found eminently beneficial when given in doses of gr. v-xxx or more, thrice daily, on an empty stomach. It almost always lessens the number of fits; often keeps them off entirely, though the dose has generally to be gradually increased in order to accomplish this end; and sometimes a complete cure is effected by its use. The bromide is found to be particularly useful when the attacks are chiefly or entirely of the "haut mal" type; when they are very frequent; and when they occur mainly by day. Brown-Séquard recommends a combination of bromide of potassium and ammonium. The treatment of epilepsy by the constant application of ice to the spine seems to be useful in some instances. In obstinate and dangerous cases local removal of blood from the back of the neck, followed by counter-irritation by means of blisters, the actual cautery, setons, or issues, either over this region, or between the scapulæ, has been recommended. In very severe cases it has also been advocated to shave the head and apply croton oil liniment. When there is an aura starting from a limb, finger, or toe, a circular blister applied around the part may prove highly useful. The treatment of epilepsy by clitoridectomy, castration, circumcision, and such methods needs only be mentioned to be emphatically condemned. Trephining the skull has been resorted to in some dangerous cases with advantage.

4. **Prevention of fits.**—Some authorities attach considerable importance to the *prevention* of the fits in the curative treatment of epilepsy, by attending to warnings, and thus endeavouring to make the attacks abortive, and to prevent the changes in the nerve-centres which increase the tendency to other attacks. The measures to be adopted depend upon the nature of the premonitory symptoms. Thus, if a sensory aura is felt in a limb, a handkerchief or band should be applied tightly around this part rapidly, and several times in succession. A case was under my notice for a considerable time, in which the aura started from the thumb, and the patient used to prevent fits after a severe struggle by drawing a handkerchief tightly round the wrist. Brown-Séquard has shown that the fits may be averted by applying the ligature round another limb; as well as by pinching or striking the skin, or irritating its nerves by heat, cold, galvanism, or repeated pricks with a needle. If an involuntary muscular contraction precedes loss of consciousness, it is recommended to draw forcibly on the contracted limbs, so as to elongate them; or a blow, pressure, or friction upon parts where some muscles become rigid may have an equally good effect. In cases where disorders of breathing or laryngismus occur at the outset, the use of ether or chloroform as an anæsthetic is recommended. In connection with laryngismus, Brown-Séquard has found cauterization of the fauces with a strong solution of nitrate of silver very efficacious. Among other preventive measures available in different cases this authority mentions the administration of an *emetic*, *purgative*, or *stimulant*; a full dose of chloral hydrate; subcutaneous injection of atropine or morphia; the immersion of the hands in hot water; inhalation of nitrite of amyl; rapid and ample respiratory movements for five or six minutes; jumping or running; and reading very fast.



## CHAPTER LXXIX.

## HYSTERIA AND ALLIED AFFECTIONS.

**PATHOLOGY AND ÆTIOLOGY.**—Hysteria is a very complex morbid condition, of the nature of which it is impossible to speak definitely. It belongs to the nervous disorders, but its exact seat cannot be localized, though probably the brain is most disturbed. No characteristic pathological change has been discovered, but there is probably some nutritive derangement of the entire nervous system. The attempt to localize the primary disorder in the sympathetic ganglia, and to attribute the phenomena observed to vaso-motor disturbance, has no sufficient foundation.

Hysteria is infinitely more common among females, beginning usually from 15 to 18 or 20 years of age, but sometimes at a much earlier or later period, in exceptional cases only developing at the change of life. Young girls, old maids, widows, and childless married women are the most frequent subjects of the complaint, and its manifestations often cease after marriage. Hysterical fits are more common about the menstrual periods. These facts have led many to consider the hysterical condition as being primarily connected with some disturbance of the sexual organs or functions, which secondarily affects the nervous system. It has thus been attributed to malpositions of the uterus; undue sexual excitement and unsatisfied desire; venereal excess; and disordered menstruation, in the way of menorrhagia, amenorrhœa, or dysmenorrhœa. Charcot attributes great importance to ovarian hyperæsthesia as a cause of hysteria. That uterine and ovarian disturbances do help greatly in exciting hysterical attacks in a large number of instances cannot be doubted, but many eminent authorities deny that these constitute the essence of the complaint. Its frequency in women is probably due to the inherent conditions of their nervous system, often aggravated by their mode of existence. The general system may be disordered by many conditions, but the sexual functions assume an undue prominence in the mind, and thus any disturbance in connection with these functions produces an exaggerated effect. In many cases of hysteria there is nothing wrong about the generative organs or functions, while it occurs often enough in married women with families. The improvement which frequently takes place after marriage may be accounted for by the change in habits, thoughts, purposes, occupation, and general surroundings which this event usually involves.

Hysteria is in some instances distinctly traceable to digestive disturbances, especially long-continued constipation with accumulation of fæces. Causes referable to the mode in which girls are brought up, and to their general habits of life, aid materially in its production, such as want of useful employment; indolent and luxurious habits; over-petting and spoiling; subjection to the petty worries of fashionable life; keeping late hours at parties; or reading sentimental novels. Temperament and hereditary predisposition to nervous affections may have some influence, but the latter may often be explained by the patient imitating a hysterical mother. In not a few cases hysteria results from depressing

influences, such as long-continued anxiety or grief; disappointed affection; or overwork, with bad feeding and improper hygienic conditions. It may further depend upon some definite chronic disease, either local or general. In some instances the condition called hysterical can only be attributed to wickedness and perversity.

The hysterical state is now and then observed in males, but infinitely rarely an actual fit of hysteria. The subjects of this condition are usually from 35 to 50 years of age, and its causes are excessive venery or masturbation; over-work, with long-continued worry and anxiety; excessive and prolonged mental labour; some violent shock; senile degeneration; or commencing chronic cerebral disease.

The *exciting cause* of the first hysterical fit is generally some powerful and sudden emotional disturbance, such as a fright, but this may be very slight if the patient has previously been in a state of mental restraint, with pent-up feelings; or has been subject to depressing influences for a considerable period. Subsequent paroxysms also are liable to arise from a much slighter disturbance than that which brought on the first attack. Suppressed laughter may lead to very severe fits of hysteria. Occasionally they result from physical disturbance, such as injury; loss of blood; or some acute illness.

**SYMPTOMS.**—It is impossible to give even an outline of the varieties of clinical phenomena which may be presented in cases of so-called hysteria. There is scarcely a complaint which may not be simulated. In most cases, however, the prominent features are an undue excitability of the emotions, with defect in the power of the will and intellect; alterations in the general cutaneous sensibility, and in the special senses, usually in the direction of hyperæsthesia and dysæsthesia; and a tendency to involuntary muscular movements, or to some other disturbance of the motor functions. It will be necessary to describe first the characters of certain paroxysms or *hysterical fits*; and then to point out some of the principal phenomena which may be noticed in the *hysterical state*.

**The Hysterical fit.**—As a rule a fit of hysteria occurs when other persons are present, and never comes on during sleep. The attack is not sudden, but gradually worked up to, the patient generally having time to place herself in a comfortable position, and to adjust her dress; it is often preceded by sighing, sobbing, laughing, moaning, nonsensical talking, gesticulation, or a feeling of *globus hystericus*, but not by any peculiar cry. During the actual fit there may be apparent unconsciousness, but this is not complete, as can be determined by touching the conjunctiva; while the patient is generally aware of what is going on around, and looks out from under her eyelids occasionally. Spasmodic movements are observed, varying from slight twitchings in the limbs to powerful general convulsive movements, or almost tetanic spasms, with opisthotonos. Hysterical patients often struggle violently and throw themselves about, while the thumbs are frequently turned in, and the hands clenched. During these movements, which may last only a few moments, or for an indefinite time with or without intermissions, there is no lividity of the face or other sign of interference with respiration. Breathing is noisy and irregular, while gurgling and spluttering sounds are frequently produced in the throat and mouth. The pupils are not dilated; in many cases slight internal strabismus is observed, and the eyes are turned up from time to time, or the eyelids are kept forcibly closed. The pulse is normal. There is no biting of the tongue, and rarely any foaming at the mouth. The paroxysm generally terminates

with crying, laughing, sighing, or yawning, and is followed by a feeling of exhaustion, but not usually by coma, though in rare instances the patient falls into a kind of prolonged trance. Frequently abundant eructations of gas take place; and there is often a copious discharge of pale watery urine. Rarely an attack is followed by a state of hysterical mania, in which the patient is not responsible for her actions.

**The hysterical state.**—The chief deviations from the normal state which so-called hysterical patients present may be considered as they affect the *mental*, *sensory*, and *motor* functions respectively.

*a. Mental.* There is defect of will and of mental power in hysteria; while the emotional functions are not under proper control, being very readily excited, and tending to lead to exaggerated actions. Some patients say they cannot perform various acts, such as standing, walking, or speaking, which they do perfectly well when they forget themselves. Frequently the spirits fluctuate very rapidly and without cause, from morbid cheerfulness to despondency, and the hysterical patient sobs, sighs, cries, or laughs without adequate reason. Ideation and thought may be over-active in some respects, but the general intellectual vigour is much impaired. Many hysterical patients talk a great deal of nonsense. They have an exaggerated feeling of self-importance; seek attention from others; and are as a rule never so pleased as when they become objects of attraction or sympathy, or are creating a sensation—which accounts for “fasting-girls,” trances, some cases of supposed somnambulism, and allied conditions. Many are very restless, irritable, and impatient. Others, however, seem simply indifferent to all around, and remain melancholy, silent, motionless, and apathetic for long periods together, caring nothing about dress or anything else. In some cases a form of mania sets in. Hysterical patients are strongly disposed to take to drinking to excess, especially in secret.

*b. Sensory.* Commonly a condition of general exaggerated sensibility, hyperæsthesia, or nervousness exists, both as regards cutaneous sensation and the special senses, an unusually slight stimulus being recognized or producing an undue effect. Further, there is often a condition of dysæsthesia, or of painful sensation from slight irritation. This is evidenced chiefly by cutaneous tenderness in certain parts, sometimes intense, especially in the left side; along some portion or the whole of the spinal column, slight pressure over which will often cause severe pains to shoot to distant parts; around the joints; and over the abdomen. The tenderness is greatly diminished by taking off the patient's attention, and it is very superficial, signs of suffering being elicited by a slight touch or a pinch of the skin, but not when steady and firm pressure is made, or when a joint is rudely jogged. Dysæsthesia from slight stimuli may also be evinced in connection with the special senses. Spontaneous pains are commonly complained of in various parts, of a more or less neuralgic character, frequently described as very intense, and being especially seated at the top or back of the head, here often assuming the characters of *clavus hystericus*; in the left side; along the back; over the sacrum or coccyx; and in the joints. Paræsthesiæ, such as formication, numbness, tingling, flashes of light, tinnitus aurium, or a peculiar smell or taste, are also common. A curious sensation often complained of is that named *globus hystericus*, which is a feeling of constriction or of a “ball in the throat,” either fixed there and giving rise to the sensation of choking, the patient making all kinds of ineffectual efforts to get rid of it, or ascending upwards from the epigastrium, or even from below



this region. In exceptional cases hypæsthesia or even complete anæsthesia of the skin and deeper structures, or of the special senses is observed. Anæsthesia is generally limited in extent, and irregular in distribution, but there may be hemi-anæsthesia, or the loss of sensation is confined to the lower limbs, or may be generally distributed. Hysterical hemi-anæsthesia may be complete or incomplete. There is often analgesia, with or without insensibility to heat and cold. Its characteristic features, when the hemi-anæsthesia is complete, have been described by Charcot and others as follows:—There is a distinct line of demarcation separating the anæsthetic from the healthy part, often corresponding closely to the median line; the affected side is comparatively cold and pale; there is a more or less permanent ischæmia, and in intense cases there may be a difficulty in inducing bleeding by pricking the anæsthetic part with a pin; the mucous membranes are involved as well as the skin; the organs of the special senses are affected to some extent on the anæsthetic side, and in connection with vision the phenomenon called *achromatopsia* may be observed in some cases; the viscera do not seem to be implicated, but, on the contrary, ovarian hyperæsthesia is observed on the anæsthetic side. If paresis or contracture supervenes, it shows itself on the affected side. The hemi-anæsthesia is mostly permanent, but presents variations in degree, and in the intensity of its phenomena, some of which are also liable to fluctuate. Patients may be quite unaware of the existence of this symptom. Charcot attaches great importance to ovarian hyperæsthesia or *ovarialgia* in cases of hysteria, to which he attributes the following characters:—It is indicated by pain in the lower part of the abdomen, usually felt on one side, especially the left, but sometimes on both, and occupying the extreme limits of the hypogastric region. It may be extremely acute, the patient not tolerating the slightest touch; but in other cases pressure is necessary to bring it out. The ovary may be felt to be tumefied and enlarged. When the condition is unilateral, it may be accompanied with hemi-anæsthesia, paresis, or contracture on the same side as the ovarialgia; if it is bilateral, these phenomena also become bilateral. Pressure upon the ovary brings out certain sensations which constitute the *aura hysterica*, but firm and systematic compression has frequently a decisive effect upon the hysterical convulsive attack, the intensity of which it can diminish, and even the cessation of which it may sometimes determine, though it has no effect upon the permanent symptoms of hysteria (Charcot). The bladder or rectum may be affected as regards their sensations in cases of hysteria, leading to great accumulation of urine or fæces, of which the patient is not aware.

*c. Motor.* Voluntary movements are generally defective in the hysterical state, and the power of the will over the muscles is weakened; while all kinds of involuntary movements are exaggerated and very readily excited, namely, those due to emotions, ideas, sensations, reflex irritation, and organic causes. The patient starts suddenly from any slight disturbance, rushes about under the influence of some notion or other, and does various other silly acts. Spasmodic movements or fixed rigidity of different muscles are not uncommonly observed, independent of fits of hysteria; while cramps are very common, as well as spasms of internal organs. Occasionally some form of motor paralysis is noticed; generally it follows a hysterical paroxysm, and is limited to one limb, or more often to a part of it, but may be more or less hemiplegic, paraplegic, or even general in its distribution. As a rule sen-

sation is not impaired in the paralyzed part; the paralysis is incomplete; nutrition is not at all impaired, or only slightly after long duration of the paralysis; while electric irritability is usually unaffected, though electric sensibility may be lessened, and now and then both are diminished. Sometimes rigid flexion of one or more joints is observed, difficult to overcome, which is evidently partly due to voluntary opposition on the part of the patient, and when it is overcome the limb rapidly assumes its former position, sometimes flying back with a sudden spring or jerk. Hysterical paralysis is liable to rapid changes, and may cease suddenly. Under chloroform it completely disappears, and power is restored. The important diagnostic marks of *hysterical hemiplegia* are that it is usually incomplete; that the tongue and face are rarely involved, though there may be ptosis; that the manner of walking is different from that characteristic of true hemiplegia, there being merely a dragging of the leg without any swinging movement, while the toes are raised; and that when the patient is made to bend forward, the arm is held back. In *hysterical paraplegia* also the paralysis is rarely complete, and one leg is more affected than the other, generally the left; movement of the limbs can often be readily performed in the recumbent posture, but when an attempt is made to walk, the patient being well-supported on either side, all power and control over the muscles seems to be gone, and she falls if the support is removed, but generally manages to recover herself suddenly when near the ground. The patellar reflex is often exaggerated, and ankle clonus may temporarily be developed. The bladder and rectum are usually unaffected. *Aphonia* is a frequent symptom in hysterical patients, resulting from laryngeal paralysis. Here there is no alteration in the quality of the voice as a rule, but it becomes a mere whisper, and if the patient is asked to make an effort to speak, even the power of whispering may be lost. Cough is attended with the usual sound. This aphonia often comes on and disappears with remarkable suddenness, especially under the influence of a strong emotion. Some hysterical patients refuse even to attempt to speak. A curious enlargement of the abdomen is observed sometimes, constituting the so-called *phantom tumour*. This region presents a symmetrical prominence in front, often of large size, with a constriction below the margin of the thorax and above the pubes. The enlargement is quite smooth and uniform; soft; very mobile as a whole from side to side; somewhat resonant but variable on percussion; and not painful. Vaginal examination gives negative results; and under chloroform the prominence immediately subsides, returning again as the patient regains consciousness.

Most hysterical patients are out of health, many of them being weak and anæmic. It is a remarkable fact, however, that even when they eat but a very small amount, nutrition often does not seem to fail. Among the numerous symptoms complained of or noticed in different cases may be mentioned:—*a. Digestive disturbances*, especially flatulence; borborygmi; copious eructations; cardialgia; depraved appetite; fullness after food; obstinate constipation; intestinal colic or gastralgia. *b. Circulatory disorders*, many of them due to vaso-motor disturbance, such as palpitation; tendency to syncope; epigastric pulsation; throbbing of vessels; coldness of the extremities; sudden flushing and heat of the face. *c. Respiratory symptoms*, for example, a sense of oppression across the chest; fits of hurried and laboured breathing, sometimes assuming a very serious aspect; spasmodic, irritable, dry cough, of long

duration, and having a peculiar squeaking, barking, or howling quality; hiccup; and spitting of blood. *d. Menstrual disorders.* *e. Disorders of micturition.* There may be great irritability of the bladder, with frequent micturition, or in other cases dysuria is present, the urine being retained. Oliguria or even total suppression of urine may be a transient phenomenon in hysterical cases; and Charcot believes that hysterical ischuria may occur as a permanent symptom, in connection with which repeated vomitings take place, the ejected matters occasionally, it is said, presenting the appearance and exhaling the odour of urine, and yielding on chemical analysis a certain quantity of urea. This condition is not accompanied with any of the signs of uræmia. Many authorities doubt the reality of its occurrence, and in most cases it is unquestionably merely a pretended symptom.

The exact grouping of the phenomena above described is extremely variable in different cases, and also in the same case from time to time. The hysterical state may be permanent; or it only breaks out at intervals, with greater or less intensity. It is in connection with hysteria that the peculiar phenomena supposed to arise from applying different metals to the surface of the body have been noticed.

**Hystero-epilepsy.**—A few observations may be made here with reference to the condition which has been termed *hystero-epilepsy* or *epileptiform hysteria*. In this condition paroxysms occur, characterized by great intensity of the convulsive phenomena, combined with certain more or less marked features which recall the phenomena of epilepsy. The cases thus denominated present various characters. 1. In one group, which is the most frequent, the hysterical seizures and epileptic fits remain distinct—*hystero-epilepsy with distinct crises*, and as subdivisions of this group Charcot gives the following:—*a.* Epilepsy is the primary disease, upon which hysteria becomes grafted, most frequently at the period of puberty. *b.* Epilepsy is superadded to hysteria. This variety is much rarer. *c.* Combinations of a secondary order—(i.) Convulsive hysteria coexists along with *petit mal*. (ii.) Convulsive epilepsy is superadded to some of the phenomena of non-convulsive hysteria, such as contracture, anæsthesia, &c. 2. In another group the attacks are of a mixed character—*hystero-epilepsy with combined crises*. This class of cases is thus described by Charcot:—*a.* The mixed attack is from the outset epileptiform hysteria. *b.* The hysterical aura always constitutes a prominent symptom. It occupies the abdomen, being generally of long duration, and does not affect the head from the first, or one of the extremities, as takes place in epilepsy with aura. *c.* In the convulsive attack there is at first an *epileptic phase*—a sudden shriek, extreme pallor, loss of consciousness, a fall, distortion of the features—then tonic rigidity seizes on all the members. This rigidity is rarely followed by the clonic convulsions, brief in duration, limited in oscillation, predominating on one side of the body. The face may become greatly tumefied and violet-coloured. There is foaming at the mouth, and the foam is sometimes bloody. Finally, general relaxation of the muscles may follow, with coma, and stertorous respiration during a less or greater length of time. *d.* To this first phase the *clonic phase* succeeds. Then all is hysteria; great gesticulations, having a purposive character, supervene, and sometimes violent contortions are made, characteristic of the most various passions, such as terror, hatred, &c. At the same time paroxysmal delirium breaks out. *e.* The termination of the attack is marked by sobs, tears, laughter, &c. These



different phases do not always succeed each other in so regular a manner; they get entangled occasionally, and now one, now the other predominates.

As to the *nature* of these hystero-epileptic seizures, some authorities regard them as a mixture or combination of the two complaints—a hybrid composed half of hysteria and half of epilepsy. According to another view, hysteria is the sole and original disease, and the convulsion, epileptic in form, only appears as an accessory element. This is the view which Charcot supports, on the following grounds:—The epileptic type is never represented in the seizure-fits, save in an imperfect manner; there is never any history of *petit mal* or of epileptic vertigo; and even when the attacks are frequently repeated, obnubilation of the intellect and dementia are never the consequences. Again, in rapidly succeeding fits of true epilepsy the temperature rises quickly to a high degree, accompanied with serious symptoms, and often followed by a fatal termination; whereas in hystero-epilepsy the temperature rarely exceeds the normal standard, and the general state of the patient is not of a kind to inspire uneasiness, even if the fits are very numerous, and continue for several days. In this country hystero-epilepsy is certainly very much less severe than in France, and does not present such marked phases.

It will be convenient in this connection also just to allude to certain curious nervous phenomena occasionally observed. 1. **Catalepsy.**—In this condition the will seems to be cut off from certain muscles, and whatever position the affected part is placed in—for instance—a limb, it will remain fixed thus for an indefinite time. Catalepsy may or may not be accompanied with unconsciousness. Sensation is usually much impaired, and may be lost. The cataleptic state is sometimes associated with organic disease of the brain; or with serious organic visceral disease. 2. **Trance.**—Here the individual lies as if dead, being ghastly pale, circulation and respiration having almost ceased. Persons in a trance have even been “laid out” as dead. 3. **Ecstacy.**—The patient pretends to see visions. Often this is combined with ridiculous dancing movements, such as are practised by certain religious communities.

**DIAGNOSIS.**—Attention to the characters described as pertaining to a hysterical paroxysm, and the circumstances under which it arises, will usually enable it to be distinguished from epileptic and all other kinds of fits. In women the hysterical state should always be borne in mind as explaining many of the ailments of which they complain. Among the most important affections which it may simulate are diseases of the brain and spinal cord; disease of the spinal column; peritonitis; abdominal tumours; laryngitis; and diseases of joints. The general signs of hysteria; the absence of pyrexia or of the characteristic symptoms belonging to the several affections; the peculiar superficial nature of any pain or tenderness present; the characters of the different kinds of paralysis, as already described; and the effects of the administration of chloroform, will in most cases enable a satisfactory conclusion to be arrived at.

**TREATMENT.**—1. **Of a hysterical fit.**—But little interference is needed as a rule. An important matter is to get rid of the numerous officious and sympathizing individuals who generally surround the patient. She should be treated firmly but kindly, an endeavour being made to gain her confidence, first ascertaining, if possible, the cause of the fit. Care must be taken to prevent injury, and the clothes should

be loosened about the neck and chest. If anything further is demanded, affusion of cold water over the face; the application of ammonia to the nostrils; or the plan of closing firmly the nostrils and mouth for an instant, so that the patient cannot breathe, may be resorted to. In obstinate cases a moderate faradic shock does no harm. If any medicine is needed, aromatic spirit of ammonia with valerian or assafoetida may be given. Charcot has revived the treatment formerly adopted, of making firm pressure over the ovarian region to check hysterical fits, especially if they are of a severe type, but this method frequently fails in this country.

2. **Of the hysterical state.**—The management of persistent and confirmed hysteria is often very difficult. Mental and moral guidance is most important, and the patient should be taught to look away from herself and her grievances, and to engage in some useful occupation. Any injurious habit must be rectified. Change of scene and associations, especially with travelling, is often very serviceable. Any cause of discomfort at home or elsewhere should be removed, if possible. General treatment, directed to the state of the system and of the blood, is often most beneficial; attention being also paid to diet, and to the state of the digestive organs. On no account should hysterical patients be encouraged to take alcoholic stimulants. Dr. Weir Mitchell has introduced a special method of treatment in certain severe cases of hysteria, which has in this country been specially recommended and practised by Dr. Playfair. It consists in separating the patient entirely from her friends and home; placing her under a competent nurse; enforcing absolute rest of body and mind; giving her a large quantity of food; and employing systematic daily shampooing and faradization of the muscles. This treatment is continued for some weeks.

Various symptoms often call for interference in hysteria. Pains in different parts are best relieved by belladonna or opium plasters or liniments; that about the joints by warm poultices or fomentations sprinkled with laudanum. Hypodermic injection of morphia may be required. For restlessness and sleeplessness bromide of potassium is the best remedy. Paralysis must be treated by electricity; and rigidity counteracted by fixing the limbs in other positions by means of splints or other mechanical apparatus, and by passive movements. If necessary, chloroform may be used; this agent may also be employed to get rid of a "phantom tumour." I have often found hysterical aphonia to be cured by applying a small blister across the larynx, or even a strip of belladonna plaster, these probably acting by exerting a mental influence. In obstinate cases the vocal cords may be galvanized; or the patient may be charged with franklinic electricity, and sparks then taken from over the larynx. It is questionable how far such drugs as assafoetida and valerian are useful in hysteria, when used as a means of cure, except in being very disagreeable; they are valuable, however, as *antispasmodics*.

---

## CHAPTER LXXX.

## HYPOCHONDRIASIS.

**ÆTIOLOGY.**—The affection thus named is in reality merely a mental condition, characterized by inordinate attention on the part of the patient to his own real or supposed bodily ailments and sensations. Adult males of the better class, who have no occupation, are the usual subjects of the complaint, but it is not uncommon to meet with it in lesser degrees among working men in out-patient hospital practice.

**SYMPTOMS.**—As a rule some actual disease sets up the hypochondriacal state originally, especially digestive or biliary disorders; venereal disease; or some acute illness. In other instances the symptoms are from the first quite imaginary. The precise symptoms complained of vary much, and they are liable to change from time to time, or new phenomena are added, for which the hypochondriac is ever on the look-out. These patients generally present a healthy appearance, while they sleep and perform their ordinary functions satisfactorily. In typical cases they go the round of the “doctors” if they can afford it; take any amount of physic, which they want to be always changing, being particularly anxious to try any new remedy that becomes fashionable; are delighted to talk about their ailments, often making use of scientific terms; consult every medical work they can get hold of; like to be examined again and again; and are often themselves much addicted to examining their pulse, tongue, urine, and stools. They are very particular about their food and drink, and often as to their dress and general “get-up.” Hydropathic and similar establishments, as well as districts famed for mineral waters, are favourite places of resort for hypochondriacs. Their moral character, and their feelings towards friends remain unaltered. These cases are always very difficult to improve, being frequently quite incurable. Ultimately they may become wretched misanthropes, and exclude themselves from all society. In the less pronounced cases hypochondriacs merely fix their attention on one or more symptoms, and cannot be persuaded that these are not due to some serious disease. The condition named *agoraphobia* may be regarded as a form of hypochondriasis, in which the patient dreads being out in the streets alone, and cannot go into the midst of any public gathering.

**TREATMENT.**—The main point in treating hypochondriacs is for the practitioner to try to acquire some control over them, and to make them believe in him, by investigating their case properly, and showing that he takes an interest in and thoroughly understands it. They cannot be talked out of their ailments, but kind and judicious reasoning may often do much, and they should be urged to take off their attention from their symptoms, to mingle in society, travel, or otherwise occupy themselves. Attention must be paid to bathing, exercise, and other modes of promoting the general health. The diet and state of the digestive organs should also be regulated. As to medicines, something has generally to be administered to hypochondriacs, and the best plan is just to treat the prominent symptoms, taking care not to give anything that can do harm. It is often very useful to send such subjects to hydropathic institutions or mineral spas, chiefly on account of the change they experience, and the society they meet.



## CHAPTER LXXXI.

## CHOREA—ST. VITUS'S DANCE.

**ÆTIOLOGY AND PATHOLOGY.**—Many views have been advanced as to the nature of the complaint named chorea, but only the most important of these views can be alluded to here.

No morbid appearances have yet been acknowledged as characteristic of chorea. Dr. Dickinson has described the morbid appearances observed in several fatal cases as widely-spread symmetrical hyperæmia and its consequences, affecting the nervous centres, but especially the ganglia at the base of the brain and the spinal cord, particularly the upper part of the latter, and the posterior and lateral portions of its grey matter. The hyperæmia was most marked in the arteries, and its effects become apparent according to the duration of the disease, namely, hæmorrhages, peri-arterial exudations and degenerations, and spots of sclerosis in chronic cases. To this increased vascularity and its consequences Dr. Dickinson would attribute the phenomena of chorea, localizing the disturbance chiefly in the spinal cord. He considers it as mainly produced by causes belonging to two classes, namely, the rheumatic condition; and various kinds of irritation, mental and reflex, in connection with the nervous system. He regards the lesions as points of irritation, calculated to excite nervous functions unduly, and thus to lead to muscular excitement.

A theory which was first started by Kirkes, and which is supported by Hughlings Jackson, Broadbent, and others, attributes certain cases of chorea to minute emboli, which are conveyed from vegetations on the valves of the heart, and become lodged in the small vessels of the convolutions near the corpora striata and optic thalami; or in these centres themselves, and other neighbouring parts of the brain. Broadbent localizes the mischief in the corpora striata; Jackson in the adjoining convolutions. Bastian regards the plugs as being of the nature of thrombi, which are formed of accumulations of white corpuscles. As a consequence impaired nutrition of the affected nerve-centres results, leading to disturbance, but not to complete abolition of their functions. The embolic theory is believed especially to apply to those cases in which chorea accompanies acute rheumatism.

Another view adopted with regard to the pathology of chorea is that it is entirely a *functional* disorder, affecting the motor centres or the spinal cord.

With regard to the *exciting causes* of chorea, cases of this disease come mainly under three categories, according as they are associated with (1) acute rheumatism; (2) some mental shock or emotional disorder, especially a sudden fright; (3) an anæmic condition of the blood and general debility. Each of these demands brief consideration.

The relation of chorea to rheumatism is now generally recognized, but different observers have come to very different conclusions as to the

frequency and importance of this relationship. Thus Dr. Stephen Mackenzie, from an analysis of seventy-two cases, found "that rheumatism had pre-existed in nearly half the cases, and that there were strong grounds for believing that it had been an antecedent in a very much larger proportion of cases." On the other hand, Dr. Sturges concludes that "chorea has nothing to do with rheumatism in three-fourths of the cases;" and "that acute articular rheumatism, although rare in the history of chorea, occurs in such association with it as to justify the assumption of some direct relationship existing in a very small proportion of examples, but that no such connection can be asserted on behalf of rheumatism generally." As this writer states, however, "until we agree upon the particular signs and symptoms which are to be accepted as valid evidence of rheumatism, we have no common factors to deal with, and may expect the remarkable discrepancies in results which actually appear." It is commonly believed that rheumatism in children often occurs without any of the usual prominent symptoms, or that these are very slight. Certainly there may be endocarditis or pericarditis with choreic symptoms and a high temperature, and no joint-symptoms. As already stated, embolism is supposed to be the usual cause of chorea associated with acute rheumatism.

Mental or psychical disturbance is a very common cause of chorea, and fright is of peculiar frequency in this relationship. Dr. Sturges affirms that psychical disturbance far outweighs all other immediate causes of chorea put together; and asserts that two-thirds of the cases analyzed were due to fright, or something allied to fright. This observer thinks that there are many causes of mental distress which are not revealed in the history of the patients. Chorea may, however, be produced by other emotional disorders besides fright; and it may also be mentioned in this connection, that the complaint may arise from imitation, when children associate with others who are suffering from it. This class of causes acts more readily upon those who are weak and anæmic.

In some instances chorea cannot be traced to any definite cause, and must be attributed merely to marked anæmia and general debility. The complaint is referred by some writers to a certain definite change in the blood, and they consider that it is allied to the acute specific diseases.

Amongst individual causes to which chorea has been attributed, are slight local disease or hæmorrhage in connection with the nerve-centres; disease of the cerebral vessels; injury to the head or to some local nerve; reflex irritation from different sources, such as worms or painful dentition; masturbation; menstrual derangements; and pregnancy.

There are some important *predisposing causes* of chorea, namely, the female sex; early age, especially from 5 to 15; the period of sexual development; hereditary tendency to various neuroses; a nervous temperament probably, and Dr. Sturges has shown that whooping-cough has more than double the frequency in choreic children than it has in others; bad living and unfavourable hygienic conditions, with consequent imperfect nutrition; a recent attack of some acute lowering illness; and a damp and cold climate or season. Anomalous choreiform movements may occur at any period of life, in connection with various organic cerebral diseases. Certain movements observed in children, and also in adults, are merely the result of a bad habit, such as frequent closure of the eye-lids, or twitching of the mouth.

**SYMPTOMS.**—Chorea is characterized by peculiar persistent involuntary movements of various muscles, partaking of the character of clonic

spasms; with loss of control over voluntary actions, the influence of the will over the muscles being diminished, while co-ordinating power is impaired. The complaint generally runs a definite course, though of variable duration, the symptoms setting in gradually; reaching their height in about two or three weeks, at which they remain for a variable time; and then subsiding. Sometimes, however, chorea remains as a chronic condition. The first signs which attract notice are that the patient seems restless and fidgety, cannot keep quiet, jerks one of the limbs about occasionally, halts or drags one of the legs in walking, makes grimaces, performs various acts awkwardly, or drops and breaks things. Dr. Sturges gives the following as the order in which the several muscular groups yield to chorea:—The hands most, the right hand, the left arm, the face, the left hand, the arms, the right arm, the legs, the left leg, the right leg. He concludes that “the muscles chiefly affected by chorea are the same which are devoted to the higher intellectual uses, and such as children have but imperfectly acquired the uses of;” and “hence the parts selected by chorea are not those which depend upon a common motor centre, but those which habitually combine in purposeful and emotional movements.”

The phenomena of chorea, when the disease is established, are very characteristic. The term “insanity of the muscles” has been appropriately applied to the absurd, disorderly, involuntary movements which are observed. As a rule they are moderate in intensity, and not painful; they exhibit great variety in combination, being not mere jerks of the muscles, but more like restless movements indicating complex co-ordinations, and often conveying an idea of purpose or design. The head is moved about in various directions; the face exhibits all sorts of ridiculous smiles, frowns, and grimaces; the tongue is often thrust out and coiled and then withdrawn again, or pushed into the cheek, or drawn into the throat, as if an attempt were being made to swallow it. The shoulders are jerked up, and the arms thrown about, while various fidgety movements are carried on with the hands and fingers. The legs are frequently unaffected, being in all cases much less disturbed than the arms. Respiratory movements are infrequent, jerky, and irregular, the natural relations of the abdominal and thoracic movements being perverted during breathing; sometimes there is a dry nervous cough or grunting sound. It does not often happen that the muscles of the trunk seem to be much affected, but choreic patients are usually unable to sit or lie quietly for any length of time. The muscles of the larynx are rarely implicated, those of the pharynx never. Very commonly these involuntary movements commence and are more marked on one side than the other; or they may be entirely unilateral—*hemichorea*, or even confined to one limb. They are much intensified by attention being directed to them, as well as under the influence of emotion. A strong effort of the will or a deep inspiration may temporarily control them, but they become worse afterwards. During sleep they cease, but may be excited under the influence of dreams.

The want of control over the voluntary movements is seen in every act which the patient performs, such as walking, holding out the hand, putting anything to the mouth, eating or drinking, smiling, attempting to take hold of or to carry any object, which is generally allowed to fall or is thrown down. Articulation is commonly indistinct and jerky. Micturition may be difficult, on account of the jerking of certain muscles. The sphincters are never affected. The muscles are in a state



of decided weakness, amounting to slight paresis. A sense of fatigue and nervous exhaustion is usually experienced; while aching in the limbs, headache, and pains in the back are often complained of. The expression seems to point to some degree of mental defect, but this is mainly due to the movements of the muscles of the face, though in many cases, especially if the disease is of long duration, the intellectual faculties become somewhat obscured.

The *general* health is almost always below par, anæmia being often a prominent feature in cases of chorea. Temperature is normal unless the disease is associated with some pyrexial condition. The digestive organs are out of order in many cases. The urine is usually concentrated at first; contains excess of urea; and frequently deposits urates abundantly, as well as oxalates and phosphates sometimes.

The state of the *heart* in chorea requires special consideration. In all cases of chorea it is desirable to examine this organ every day, if practicable. There are differences of opinion as to the cardiac phenomena observed in this disease, as well as with regard to their explanation. Dr. Sturges states that the commonest heart symptom is increased frequency. In many cases the cardiac action is easily disturbed, and it may be irregular. With respect to the *physical signs*, in a certain proportion of cases the heart sounds are merely modified, and there is no distinct murmur. The most frequent and important sign, however, is a murmur of some kind, and Dr. Stephen Mackenzie found that this was present in 54.26 per cent. of the cases analyzed by him. The murmur infinitely most common is mitral systolic, but occasionally a double murmur is heard in connection with the mitral orifice, and still more rarely it is simply præ-systolic. Sometimes there is a basic murmur. In markedly anæmic cases an anæmic murmur is heard. With regard to the explanation of the murmurs, and the mitral systolic murmur more especially, in a large proportion of cases they are due to organic mischief from endocarditis, and are persistent. This is generally believed to be of a rheumatic nature; and in fatal cases of chorea, *post-mortem* examination has usually demonstrated the existence of definite morbid conditions accounting for the murmur. In some cases, however, mitral systolic murmur is not associated with organic mischief, but is an *inorganic* murmur, and different explanations have been given of the regurgitation under these circumstances. Thus it has been attributed to irregular or spasmodic action of the muscoli papillares; to fatigue-paresis on the part of these muscles, so that the valves do not close properly; or to a weakened condition of the cardiac muscles, so that temporary dilatation occurs, and consequent enlargement of the mitral orifice with regurgitation. It is also supposed that in some instances a murmur may disappear, owing to recent lymph being washed away or absorbed. Dr. Sturges does not attribute the cardiac mischief usually to acute rheumatism, but to the fact that chorea affects the heart along with other muscles which are influenced by emotion. Dr. Dickinson has advanced the opinion that endocarditis may be the consequence of chorea, being brought about by the irregular action of the heart.

Cases of chorea are occasionally met with in which the symptoms present an extremely acute and aggravated character, the spasmodic movements being excessively violent and constant, and extending throughout the body. The patient is unable to swallow or to perform any voluntary act, and becomes greatly distressed and exhausted, sleep being rendered impossible. Death ensues if the movements do not

abate, often preceded by adynamic symptoms, delirium, or coma, but the intellect may be clear almost to the last. Two such fatal cases, occurring in girls about the period of puberty, have come under my notice, and at this period these violent attacks are usually noticed; similar attacks have been observed in connection with parturition.

DIAGNOSIS.—The symptoms of well-marked chorea are so characteristic, that it is scarcely possible to make a mistake in diagnosis, and therefore no special remarks need be made on this subject.

PROGNOSIS.—Chorea almost always terminates in recovery, except when it assumes the severe form alluded to above. No definite opinion as to duration should be given. The circumstances favourable to a speedy recovery are that the disease is due to some condition which is amenable to treatment; that this is commenced at an early period; and that the patient can be placed under proper sanitary conditions. The danger of the development of some cardiac complication should always be borne in mind, and the establishment of permanent heart mischief. Chorea greatly increases the danger from acute rheumatism.

TREATMENT.—It is difficult to estimate the value of remedies in the treatment of chorea, as the complaint usually tends towards spontaneous cure. The indications which should be primarily attended to are:—1. To get rid of any obvious cause of reflex disturbance. 2. To regulate carefully the diet and the state of the digestive organs, especially maintaining a free action of the bowels. 3. To improve the general health and quality of the blood, by nutritious food; proper hygienic conditions; change of air; cold or tepid bathing, or the douche, especially applied over the back, with friction afterwards; and the administration of some preparation of iron, particularly if the patient is anæmic. Many cases do remarkably well under the use of ferruginous preparations, especially the sesquioxide, tincture of sesquichloride, ammonio-citrate, or carbonate. A great many supposed *specifics* have been introduced for the cure of chorea, the chief of these including salts of zinc; liquor arsenicalis; tincture of belladonna; conium juice; hydrate of chloral; tincture of cannabis indica; hypophosphites; Calabar bean in the form of powder, extract, or tincture; a combination of morphia with strychnia; and chloroform by inhalation twice or thrice daily. From personal experience I do not think that any one of these remedies is applicable for all cases, but one or other of them may be found of service in different instances. The application of ice to the spine; the passage of a slight constant galvanic current along this region; and subcutaneous injection of curare, are among other special modes of treatment which have been advocated. The movements may often be diminished by proper discipline, and are greatly improved by gymnastic exercises. When the disease comes under treatment in its very early stage, some practitioners believe that they can check its course by exciting a free action of the skin by means of warm or hot-air baths, followed by saline medicines, or by small doses of tartar emetic. Others employ *emetics* at the outset. Should sleep be much disturbed, some *narcotic* must be given. If the movements are very severe, it will be well to let the patient sleep on an air-bed or water-bed. Chorea complicating acute rheumatism usually needs no special treatment. Those dangerous cases in which the movements are extremely violent are but little amenable to any treatment. Inhalation of chloroform; subcutaneous injection of morphia, or, perhaps, of curare; and supporting the patient, enemata being employed if necessary, seem to me the most

reliable measures to be adopted in such cases. Drs. Goodhart and Phillips have treated successfully some cases of acute chorea by massage and free administration of nourishment.

---

## CHAPTER LXXXII.

### TETANUS—LOCK-JAW.

**ÆTIOLOGY AND PATHOLOGY.**—Though almost always of *traumatic* origin, and therefore occurring in surgical practice, a brief description of tetanus is needed in this work, as it occasionally comes under the notice of the physician as an *idiopathic* affection, resulting from a person being exposed to cold or wet, sleeping on damp ground, or becoming chilled when perspiring; or attacking infants soon after birth. It is probably a functional disorder of the spinal cord, depending upon peripheral nerve-irritation, which leads to reflex disturbance. The condition of the cord has been generally regarded as one of undue excitation, but Dr. Ringer and Dr. Murrell have advanced the view that it is in a depressed state. Certain morbid appearances have been described in the spinal cord, but they cannot in the present state of knowledge be said to be at all characteristic of tetanus.

**SYMPTOMS.**—The peculiar features of tetanus are persistent tonic spasm or rigidity of the muscles; with extremely painful paroxysms of cramp occurring at intervals. The patient first complains generally of pain and stiffness behind the neck, which increases until the muscles of this part become fixed, the head being drawn back. Then trismus or lock-jaw sets in, and swallowing becomes difficult. Next the rigidity extends to the muscles of the trunk; and finally all the voluntary muscles may become involved, except those of the hands, eye-balls, and tongue. They feel hard, tense, knotted, and rigid. The body is generally curved backwards—*opisthotonos*; but may be rigidly stretched out—*orthotonos*; bent forwards—*emprosthotonos*; or laterally—*pleurosthotonos*. A very painful feeling of constriction is experienced in the epigastrium, shooting towards the back. Soon paroxysms of painful spasms commence, at first slight and occurring at long intervals, but becoming rapidly more frequent, intense, and prolonged, so that they are excited by any slight disturbance, such as a touch or a noise, or even come on spontaneously, at last being almost constant. During these fits the patient experiences great distress and suffering; the muscles stand out and become extremely hard, and the back is often so curved that only the head and heels touch the bed; the countenance presents the “*risus sardonius*,” and has a peculiar aged expression, combined with that of intense anguish. Breathing is arrested, owing to the fixation of the respiratory muscles, this causing an extreme feeling of oppression and impending suffocation, but the act of respiration is comparatively free in the intervals. The voice is weak. During the attacks there is much heat and sweating; and the pulse is very frequent and small. Soon it becomes impossible for the patient to swallow anything, though often feeling very hungry and thirsty; while the mouth is clogged with viscid mucus. Sleep is entirely prevented. There are



no head-symptoms, and the intellect remains undisturbed. The pupils are dilated. Cutaneous sensation is not affected, but there is increased reflex excitability. The power over the sphincters is retained; constipation is generally present, and micturition is often difficult. Death is a very frequent termination, either from sudden or gradual apnoea; from asthenia, in consequence of exhaustion and want of support; or from both causes combined. The temperature in many cases rises to a very high degree before death, and continues to ascend after death. Recovery occasionally takes place in cases of tetanus, but convalescence is very slow. Temporary remissions not uncommonly occur, which are apt to mislead as to the ultimate issue. Tetanus now and then assumes a somewhat chronic course. It is usually less acute in its progress when idiopathic than when traumatic.

DIAGNOSIS.—Strychnine-poisoning is the chief condition with which tetanus is likely to be confounded; for their distinctions reference must be made to toxicological works. The complaint might possibly be mistaken for hydrophobia; for acute spinal meningitis; or for certain cases of hysteria.

PROGNOSIS is extremely grave in tetanus, as may be gathered from what has been previously stated, but the disease is not necessarily fatal.

TREATMENT.—The only measures which seem to me of any service in idiopathic tetanus are to use warm, vapour, or hot-air baths freely: to administer subcutaneous injections, either of morphia, curare, or nicotine; to relieve the spasms by inhalation of chloroform; and to support the patient by means of liquid nourishment and stimulants, administered in the form of enemata when they cannot be swallowed. All sources of disturbance must be removed, and the patient kept perfectly quiet. The application of ice to the spine has been recommended, but in one case which came under my observation no good effects whatever resulted from its employment.

---

## CHAPTER LXXXIII.

### TETANY.

THE complaint named *tetany* is a form of neurosis, attended with idiopathic muscular spasms, which has of late years come into prominence, and which therefore calls for brief notice.

ÆTIOLOGY AND PATHOLOGY.—The causation of tetany is very uncertain, and it cannot be traced to any definite lesions in any part of the nervous system. Females suffer more than males; and the complaint is most common from 15 to 30 years of age, but may occur in older persons, and is not unfrequent in children or young infants. It seems to be much more prevalent in France than in England. The neurotic temperament predisposes to tetany; as well as constitutional disturbance or general weakness from various causes, such as bad feeding, dentition, acute diseases, chronic diarrhoea, the establishment of menstruation, pregnancy, or excessive lactation. The chief *exciting causes* of tetany are said to be

emotional disturbance; exposure to cold and damp; and diarrhœa. Dr. Abercrombie has shown that the affection may be associated with rickets. It may also be produced by involuntary imitation, having in this way spread extensively in a girls' school. Nothing definite is known as to the pathology of tetany, but it is regarded as a *functional* disorder of the central nervous system, especially the spinal cord, attended with increased irritability.

**SYMPTOMS.**—Tetany is characterized by tonic spasmodic contractions of certain muscles; of a painful character; varying much in extent, but usually commencing in, and being often limited to, the hands and forearms, though in other cases extending to the lower extremities, and to other parts of the body; usually bilateral; differing also much in duration and intensity, being often intermittent; and unattended with loss of consciousness.

The symptoms usually begin with abnormal sensations, such as numbness and tingling, or actual pain, in the fingers or in the hands and forearms. Very speedily the spasms develop, commencing with contraction of one or more fingers, chiefly induced by attempts to use them. They extend upwards, and the following appearances are thus produced. The fingers are usually drawn together in the form of a cone, but the ring and middle fingers are sometimes separated; they are slightly flexed at the metacarpo-phalangeal joints, but otherwise extended. The thumbs are strongly adducted, or bent into the palm, their terminal joints being extended; rarely the fingers are flexed over them. The wrists are usually somewhat flexed, and the hands tilted towards the ulnar side. In some cases the forearms are semiflexed, the upper arms adducted, and the hands crossed upon the abdomen. In the lower extremities the symptoms commence with tingling and numbness in the toes, which then become strongly flexed towards the sole, and drawn together, the great toes being generally drawn under them, occasionally extended. The dorsum of the foot is arched, and the heel drawn up; while the legs and thighs are extended. The spasms may extend in severe cases to the muscles of the back of the neck, those of the chest and abdomen, the facial muscles, those of mastication and articulation, the diaphragm, and the larynx. Hence the jaws may be firmly clenched, speech much embarrassed, and respiration seriously interfered with.

The extent of the spasms varies considerably in different cases, being in some instances quite localized, in others widely distributed. The lower extremities are often affected after the upper, or all the limbs are attacked in succession or simultaneously; and the spasms may subside in the limbs as they invade the trunk. The affected muscles feel rigid, resist passive extension, and when the extension is discontinued they resume their contracted state. They sometimes present fibrillar tremblings. An important fact in diagnosis is that the spasms continue during sleep. Trousseau affirmed that they relax under chloroform, but Dr. Abercrombie did not find it so in his cases. Trousseau observed that the spasms could be excited by compressing the affected parts, either in the direction of their principal nerve-trunks, or over their blood-vessels, so as to impede the venous or arterial circulation. The application of cold frequently arrests them temporarily. Dr. Abercrombie noticed a peculiar "facial irritability in children affected with tetany." If the finger be drawn across the facial nerve, the orbicularis palpebrarum of the same side contracts, and in some cases also the

levator anguli oris and alæ nasi. This phenomenon is often more marked on one side than the other.

The spasms of tetany occur in paroxysms, usually intermittent, not uncommonly, however, and especially in children, only presenting remissions. The attacks last from a few minutes to an hour or two or longer, but seldom over twelve hours; and they come on at intervals, ranging from an hour or two to some days or weeks. The entire duration of the complaint may be only a few days, but more commonly it lasts several weeks or months, owing to the occurrence of relapses.

The spasms are painful in themselves, and the pain is aggravated by any attempt to overcome them by extension. Severe pain may also be felt along the nerve-trunks, and there may be some diminution of sensibility in the affected parts. Towards the termination of an attack formication and other abnormal sensations are often experienced. Erb states that there is increase of electric excitability in the peripheral nerves to both the constant and induced currents, but not in the facial nerves. Edema, redness, and pain are not uncommonly observed in children on the backs of the hands. Rheumatic inflammation of joints is said to occur occasionally. Dr. Abercrombie states that laryngismus is frequently associated with tetany in children. There is never any loss of consciousness. When the spasmodic attacks are very severe, slight pyrexia is observed, with a rapid pulse and furred tongue.

Tetany is often a very slight and temporary disorder, and even in prolonged cases recovery usually takes place, the paroxysms becoming by degrees less frequent and severe. A fatal result has occurred in exceptional instances, either from asphyxia, from prolonged duration of the complaint, or from implication of the medulla oblongata.

TREATMENT.—The chief indications in the treatment of tetany seem to be to get rid of any obvious exciting cause; to improve the general health; and to attend to any special constitutional disorder, such as rickets. Regular and sound sleep is of much importance; and the nervous system needs due attention. *Tonics* are often of much service. The chief special drugs which have been recommended are bromides, chloral, opium, valerian, musk, and conium. Electricity is not of much service, but the constant current answers best. Baths have not proved useful.

---

## CHAPTER LXXXIV.

### ALCOHOLISM.

ÆTIOLOGY.—The injurious effects produced upon the system by the abuse of alcohol are but too well known. They are the result of its direct irritant action; of its influence on the vaso-motor nerves; of the circulation of its own poisonous elements, or of those derived from its decomposition, through the various organs and tissues; and of its interference with tissue-metamorphosis, oxygenation, and nutrition. The exact effects will depend on the nature, quantity, and strength of the stimulant indulged in. Spirits do by far the greatest harm, especially when taken in frequent drams, strong, and on an empty stomach. Al-



coholism is most frequent in males ; and in those who from their occupation are exposed to intemperance, such as draymen, potmen, or cabmen, or whose calling is a lonely or a sedentary one. It is also predisposed to by various conditions which depress the nervous energy, such as working or sleeping in a hot or vitiated atmosphere ; excessive mental work ; anxiety or worry ; or excessive venery. Persons who suffer severe pain, and hysterical individuals are very likely subjects to drink to excess. In not a few instances there seems to be a hereditary tendency to alcoholism, or to some neurosis, such as epilepsy or mania.

**SYMPTOMS.**—Cases of alcoholism may be included under the following groups :—1. *Acute alcoholic poisoning*, the symptoms being those of narcotic poisoning. 2. *Delirium tremens*. 3. *Chronic alcoholism*. 4. *Acute mania*, in which the patient is extremely violent and dangerous, and has a fixed delusion. 5. *Acute melancholia*, with suicidal tendency. 6. *Oinomania*, where there is a constant craving for drink, which breaks out at intervals into an uncontrollable propensity, the moral sense being entirely deadened, so that the subjects of this condition will do anything in order to obtain drink. Only *delirium tremens* and *chronic alcoholism* can be specially considered here.

**Delirium tremens.**—This condition may come on under the following circumstances :—1. From mere excessive drinking in a temperate person. 2. An individual who is accustomed to drink freely gets very drunk. 3. A habitual tippler, who without being actually drunk is always more or less fuddled from saturation with alcohol, experiences some slight disturbance, especially of a traumatic kind ; or delirium tremens may arise in such persons even without any apparent cause. 4. From deprivation of proper food, with moderate indulgence in stimulants. 5. In consequence of suddenly cutting off the supply of stimulants from an individual who has been accustomed to drink freely, especially if old or debilitated. 6. As the result of inhalation of fumes from a distillery, it is said (?). Most cases of delirium tremens follow abuse of spirits.

Generally delirium tremens is preceded by *premonitory* indications, especially disturbed sleep or absolute insomnia ; general discomfort and feebleness ; agitation and tremulousness ; mental confusion and inability to fix the attention ; timidity and lowness of spirits. The alimentary canal is commonly disordered, as evidenced by anorexia, foul tongue and breath, unpleasant taste, and constipation with unhealthy stools.

The actual symptoms of delirium tremens are usually very characteristic. The patient is either quite sleepless, or only obtains short uneasy dozes. The mind is in a state of general confusion, restlessness, and excitement, and though it may be possible to attract the attention of the patient for a moment, and to obtain a sensible answer to a question, he speedily wanders off and talks ramblingly and incoherently, there being a kind of busy delirium. A variety of mental delusions, illusions, and hallucinations usually exist, the patient fancying he sees or hears all sorts of objects and sounds, often of a hideous character ; or distorting in his imagination what he does see and hear into strange and horrible forms and noises. These delusions are generally transient and changeable, but occasionally the patient fixes upon one, and reasons about it. Further, the mental condition is one of combined irritability, marked cowardice, sense of dread, and suspicion. The patient has an anxious, wandering expression, and looks upon everybody around

with terror and distrust, imagining that they are trying to poison or otherwise injure him; or fears lest he may do an injury to himself, and has a great sense of alarm as to what is going to happen. These feelings may culminate in fits of violent mania, attended with extreme muscular effort and a wild expression, the patient trying to injure those around, to jump out of the window, or to do various other acts with the view of escaping from some imaginary enemy. There is usually no complaint with reference to the head. Creeping sensations over the skin and other paræsthesiæ are common, and the patient often fancies he sees or feels horrible insects crawling over him. The prominent symptoms connected with the muscular system are restlessness, carphology, and general tremors, the latter being especially observed in the hands and tongue. After fits of violence the patient is much exhausted and prostrated. The pupils are generally dilated and sluggish.

The important *extrinsic* symptoms in delirium tremens are profuse perspiration, usually with little or no fever, the sweat having often a very disagreeable smell, the skin feeling moist and clammy, especially that of the palms, or being even drenched; a weak, large and soft, or small and frequent pulse, the sphygmographic tracing often exhibiting marked dirotism; and disorder of the alimentary canal, as indicated by foulness of the mouth and tongue, which are covered with sticky mucus, peculiarly unpleasant breath, complete loss of appetite, much thirst, nausea but rarely vomiting, and constipation with offensive stools. The urine is sometimes much diminished in quantity, and is deficient in phosphates and urea, but often deposits urates on standing. In exceptional cases of delirium tremens there is severe pyrexia, the temperature rapidly rising to 105°, or even to 108° or 109°.

Recovery is in many cases of delirium tremens preceded by restoration of sleep, but this by no means necessarily leads to a favourable termination. In fatal cases typhoid symptoms frequently set in, with a dry brown tongue, sordes on the teeth, and low nervous phenomena, such as muttering delirium, epileptiform convulsions, and coma. Pneumonia or some other inflammatory complication may also arise. Sometimes death results from sudden collapse.

**Chronic alcoholism.**—Various grades of this condition are to be constantly seen, especially in hospital practice. A very able description of the phenomena observed has been given by Anstie. The ordinary signs may be summed up as follows:—1. *Nervous phenomena.* These include muscular restlessness and fidgetiness, culminating in tremors, beginning in the limbs, at first slight and controlled by an effort of the will, but afterwards becoming more marked and constant, being worse in the mornings, and then diminished by food and drink; insomnia, or very disturbed and unrefreshing sleep with horrible dreams; diffused dull pain or heaviness in the head, and sudden attacks of vertigo; disorders of the special senses, as evidenced by photopsia or muscæ volitantes, and noises in the ears; mental disturbance, indicated in the early period by mental disquietude, uncertainty of purpose and inability to fix the attention upon anything, indecision of character, a vague sense of dread, or fits of violent temper; later on by impairment of the mental faculties, in some cases very marked, the patient having horrible visions or delusions as to people plotting his ruin, and exhibiting great cowardice with loss of moral power, and a particular tendency to tell falsehoods about drink; impairment of muscular co-ordination,



which explains the sensation sometimes experienced by the patient, as if he were going to fall down a precipice when walking on firm ground. 2. *General appearance.* The signs coming under this head are more or less obesity or emaciation, the former being chiefly observed in beer-drinkers, the latter, which may be extreme, in spirit-drinkers; flabbiness or a bloated aspect of the features, with red and watery eyes, yellowness of the conjunctiva from fat or jaundice, and often redness of the face with enlarged vessels, especially about the nose and cheeks, or acne. 3. *Disorder of the alimentary canal,* indicated by total anorexia or disgust for food, especially in the mornings, the patient often making this an excuse for taking stimulants in order to "keep up" the system; thick dirty furring of the tongue as a rule, but not always; dryness and cracking of the lips; catarrh of the pharynx; peculiar and disgusting foulness of the breath; severe morning nausea or actual sickness; irregularity of the bowels, with foetid stools; and occasionally serious hæmorrhage from the stomach or bowels. 4. Symptoms due to *organic visceral changes* and to *degenerations.* These have been pointed out in previous chapters, and, as already stated, there is much difference of opinion as to the influence of alcohol in their production. There can be no doubt as to the direct effect of strong spirits upon the mucous membrane of the alimentary canal, and especially upon that of the stomach, these agents inducing congestion, chronic inflammation, fibroid changes, and glandular degeneration; or that alcoholic abuse tends to lead to fibroid and fatty degeneration with atrophy of various organs and tissues, including the nerve-centres.

In very advanced cases of chronic alcoholism still more grave nervous symptoms are met with, such as absolute dementia; marked sensory paralysis in different parts; extreme muscular trembling, simulating paralysis agitans; general muscular weakness; paralysis or ataxia; epileptiform attacks; or finally coma. Such phenomena are necessarily attended with serious organic changes in the nervous system.

DIAGNOSIS.—*Delirium tremens* has to be mainly distinguished from acute mania or meningitis. The history of the patient, and the circumstances under which the affection occurs; the characters of the nervous and extrinsic symptoms; and the absence of any fixed delusion, generally leave no doubt as to the nature of the case. Sometimes acute alcoholism closely simulates low fevers. *Chronic alcoholism* should always be suspected if any of the symptoms mentioned are complained of, not forgetting digestive disorders, but especially should there be morning sickness, insomnia, fidgetiness or tremors, mental restlessness, or disturbance of the special senses. Close inquiry is often needed in order to elicit a history of intemperance in these cases, many patients trying to conceal their evil habits in every possible way. The odour of the breath is frequently very characteristic. Anstie enumerates the following nervous diseases as being particularly liable to be simulated by chronic alcoholism:—commencing general paralysis of the insane; paralysis agitans; lead-poisoning; locomotor ataxy; softening of the brain or cord; epilepsy; senile dementia; hysteria; and the nervous malaise associated with some forms of dyspepsia.

PROGNOSIS.—*Delirium tremens* usually terminates favourably. The chief unfavourable circumstances are:—A history of chronic indulgence in excess of alcohol, so that the system is more or less saturated; the patient being advanced in years, enfeebled in constitution, or suffering from organic visceral disease, especially disease of the kidneys; a history



of previous attacks, particularly if they have been numerous; difficulty in getting nourishment into the system, either from the patient refusing food, or from assimilation being impaired; inability to procure sleep before the patient is much exhausted; an unfavourable condition of the pulse, as evidenced by the sphygmograph; the occurrence of typhoid or low nervous symptoms; and the development of inflammatory complications, especially pneumonia. In the early period *chronic alcoholism* can always be cured if patients will keep away from drink, but it is often a very difficult matter to get them to do this. When serious nervous symptoms have become developed, there is but little hope of improvement.

**TREATMENT.—Delirium tremens.**—1. In treating delirium tremens, the first object aimed at should be to *withdraw* or to *reduce the quantity* of all forms of alcoholic stimulant, so far as this is practicable, but especially of spirits and wine. In a large proportion of cases it has been found that no harm whatever results from cutting off stimulants completely, especially in young patients and in first attacks; in others they must be moderated as much as possible, being chiefly needed if the patient is an habitual drunkard, old, or feeble, or if there are signs of adynamia. It is well to keep to malt liquors, if it can be managed, but brandy may be required. At the same time it is highly important to introduce as much nourishment as possible into the system. Strong beef-tea, beef-juice, hot soups, milk, eggs beaten up, and other forms of nutritious food which are readily assimilated, must be given at frequent intervals, by night as well as by day. If the patient refuses food, white of egg mixed with iced water is useful, and nutrient enemata must be regularly employed. In the treatment of strong patients, especially if they are young, and a large quantity of spirits has been taken, a brisk *watery purgative* is decidedly beneficial at the outset, but this is not advisable in all cases.

2. The next indication is to endeavour to *procure sleep* before the patient is exhausted. For this purpose certain drugs are most useful when employed in moderate doses, especially opium or morphia, the latter being best introduced by hypodermic injection (gr.  $\frac{1}{8}$  to  $\frac{1}{2}$ ); hydrate of chloral (gr. xx every hour or two); bromide of potassium (gr. xx every two hours); and extract or tincture of cannabis indica. As a general rule I quite agree with Anstie and others in opposing the notion that "patients in delirium tremens require to be narcotised into a state of repose, but I have met with cases in which the only chance of recovery seemed to be in procuring sleep at any risk, and where the administration of considerable doses of morphia, combined with abundant nourishment, proved, I believe, the means of saving life. Other remedies employed in the treatment of delirium tremens are tincture of digitalis in large doses (3ij to 3i every four hours), originally introduced by Mr. Jones, of Jersey; capsicum in the form of powder or tincture in full doses; tartar emetic in sthenic cases attended with wild delirium; and chloroform, either by inhalation or internally. Chloroform inhalation carefully employed may be decidedly serviceable sometimes.

3. *Symptoms* often require attention in acute alcoholism, especially vomiting. Should there be adynamic signs, *stimulants* must be given, such as ammonia, ether, musk, or camphor, along with brandy. High fever may demand antipyretic measures. *Complications* may also call for interference, particularly pneumonia, which always needs a supporting treatment in these cases. A patient suffering from delirium tremens

should be placed in a comfortable and well-ventilated room; kept perfectly quiet and apart from friends, only one or two trained attendants being permitted to be present, according as the patient is peaceable or violent; treated kindly, but with firmness; and constantly watched, lest he should injure himself. Mechanical restraint, such as that by means of the strait-waistcoat, is but rarely admissible, though it is needed now and then in cases of extreme violence.

**Chronic alcoholism.**—In treating chronic alcoholism, there should in most cases be no hesitation in forbidding stimulants entirely, but especially spirits or wine. It is often, however, difficult to persuade patients to carry out this advice. A glass of good bitter ale or stout along with food may be useful in some instances, and Anstie recommended a glass of stout at night, in order to procure sleep. It is most important to induce the patient to take nourishment, and as there is generally a great distaste for food, small quantities of milk, concentrated beef-tea, soups, or meat-juices should be given at frequent intervals. It is wonderful, however, how soon the appetite returns in many of these cases when the intemperate habits are relinquished. If there is much sickness, an effervescent mixture may be given, or soda-water with milk. I have found a mixture containing bicarbonate of soda or nitro-muriatic acid with infusion of gentian and hydrocyanic acid (℥ iij-iv), very serviceable in many cases. Anstie recommended one or two grains of quinine twice or thrice daily. Marcet found oxide of zinc useful, beginning with gr. ij twice daily, and gradually increasing the dose. Others have much faith in tincture of capsicum. If there is much restlessness and sleeplessness, a full dose of bromide of potassium at night will generally procure sleep, or this drug may be given more frequently if necessary. Some practitioners prefer subcutaneous injection of morphia; hydrate of chloral; extract of *cannabis indica*; or a full dose of sulphuric ether. Baths are often serviceable in chronic alcoholism; and rest from occupation, with change of air, aids recovery materially. The bowels should be kept well-opened.

In advanced cases the treatment must be varied according to the prominent symptoms present. Anstie found the long-continued use of good doses of cod-liver oil most beneficial, with hypophosphite of soda or lime if there is commencing paralysis of sensation; bromide of potassium should there be epileptiform convulsions; and very minute doses of strychnine when marked muscular tremor is observed.

---

## CHAPTER LXXXV.

### ON CERTAIN FORMS OF METALLIC POISONING.

#### I. LEAD-POISONING—SATURNISM.

**ÆTIOLOGY AND PATHOLOGY.**—The introduction of lead into the system is most important in connection with certain occupations in which this metal is used, saturnism being especially common among painters, plumbers, and workers in white-lead. Sometimes it acts as a poison through being taken in water kept in leaden cisterns, in cider, in adulterated articles, or medicinally; it may be inhaled from fresh paint;



or now and then it gains access into the body in curious ways, as from using adulterated snuff, rubbing the ointment into the skin, or using a hair-wash containing lead. Occasionally, although there is clear evidence of the presence of this metal in the system, very minute investigation has to be made before the channel of its introduction can be discovered. As a rule lead is either swallowed or inhaled, and often enters the system by both the alimentary canal and the lungs.

With regard to the pathological effects of the metal upon the different structures, and the explanation of the phenomena which it produces, there is much uncertainty. It becomes deposited in almost every tissue, being said to be most abundant in the following structures, in the order given:—bones, kidneys, liver, brain and spinal cord, and muscles. The chief morbid changes which have been described in cases of chronic lead-poisoning are contraction and apparent hypertrophy of the muscular coat of the large intestine, and atrophy of the intestinal mucous membrane; atrophy and degeneration of paralyzed muscles, with increase of the connective tissue, and sometimes of fat; changes in the spinal cord, although in some cases careful investigation has failed to discover any such changes; atrophy of nerve-elements, with increase of connective tissue in the abdominal ganglia of the sympathetic; and degeneration of nerves supplying paralyzed muscles. By different writers the phenomena of lead-poisoning have been attributed mainly to the effects of the metal upon muscular tissue directly; upon the nerve-centres, which then influence the blood-vessels, and lead to their contraction; or upon the nerves themselves. Lead-paralysis has even been referred to arterial contraction; and its special form, in which the extensors of the forearm are alone involved, has been attributed by Hitzig to a peculiar disposition of the veins, but on insufficient grounds.

**SYMPTOMS.**—Certain objective appearances are usually very obvious in connection with chronic saturnism, namely, the so-called blue line on the gums at their junction with the teeth; a dirty brown or black incrustation of the latter, if they are not cleaned, with rapid tendency to decay; more or less wasting, with a dry harsh skin, anæmia, and a peculiar sallow, earthy, pale or yellowish tint of countenance, with yellowness of the conjunctivæ—*saturnine cachexia*. The blue line is not always present even when there are other marked signs of lead-poisoning; while, on the other hand, it may be very distinct, and yet the health is apparently unimpaired. Much depends on the habits of the individual, with regard to cleaning the teeth, for the discoloration seems to be due to the action of sulphuretted hydrogen upon lead in the tissues of the gums, the gas originating in the decomposition of food and tartar upon and between the teeth. The breath is generally offensive; the tongue is furred; and a sweetish astringent taste is frequently experienced. In some cases the pulse is very infrequent and slow. The other prominent clinical phenomena which may be associated with lead-poisoning may be summed up thus:—1. *Lead-colic*, having the characters of more or less severe intestinal colic, accompanied usually with a retracted abdomen; absolute constipation; nausea and sometimes vomiting; eructations; and in some cases hiccup. 2. *Disorders of sensation*, such as hyperæsthesia or hypæsthesia of different parts, numbness, formication, neuralgic pains, aching in the limbs and joints, and headache. 3. *Ammaurosis*, either single or double, usually associated with other grave nervous symptoms, and accompanied with changes visible with the ophthalmoscope. 4. *Mental disorder* in bad cases, such as delirium, mania, or



melancholia. 5. *Motor disturbance*, in the way of tremors, epileptiform convulsions, or local paralysis. The most common and important variety of paralysis is that of the extensors of the forearm, giving rise to *wrist-drop*, the patient being unable to extend the wrist-joint. Careful observation has shown that the supinators usually escape, and are able to produce their normal movements. The muscles of the hands not uncommonly suffer; and the upper limbs may in time become more or less affected throughout. In some instances the paralysis involves the lower limbs, and may even extend to those of the trunk, preferably the extensor muscles, so that the patient assumes a stooping and tottering gait. Occasionally voice is lost. As a rule both forearms are implicated, but not equally. In a case recorded by Dr. Buzzard, the paralysis appeared to be confined to the right hand and left foot, but careful investigation showed that all the limbs were affected, but to different degrees. The muscles are generally considerably wasted, giving rise to a marked depression on the back of the forearm, and those of the hands may also be much atrophied, so as to make these parts assume the "crow-foot" shape. Sometimes the hands are strongly closed, as if the flexor muscles were rigid. Oval or elongated swellings are also sometimes found in the tendons behind the wrist. Occasionally Dr. Buzzard has noticed an almost unnatural roundness of limb, but with a pulpy feeling of the flesh, which this observer attributes to a large overgrowth of adipose and connective tissue, masking the muscular atrophy. With regard to the electrical condition of the affected muscles, their excitability to faradism becomes much diminished or lost; in cases of lead-palsy of not long standing, exaggerated reaction to galvanism is not at all unfrequent (Buzzard). Ultimately the muscles also cease to react to galvanism. The predisposing influence of lead in the system with reference to gout has been alluded to in connection with that disease, and in this relation lead may be the original cause of chronic renal disease. Abortion is said to occur frequently in women who work at white-lead works.

TREATMENT.—Preventive measures are most important in the case of those working with lead. They should be very particular as to cleanliness, especially in washing their hands and cleaning their nails before eating, and in cleansing their lips and teeth. Every precaution should be taken against inhaling particles of lead. I believe a good deal of the metal is often introduced during meals, and the practice of taking a small quantity of dilute sulphuric acid in water at these times may be useful, as this would form an insoluble compound with any lead entering the stomach. The bowels must always be kept well-opened, especially by sulphate of magnesia. If there is any lead in the system, iodide of potassium may be given from time to time. Lead-colic must be treated in the same way as other forms of intestinal colic. The great remedy for getting the metal out of the system, which is the main object to be aimed at in all cases, is iodide of potassium, a soluble iodide of lead being formed, which passes away in the urine and other excretions. This drug must be given for a long time; and may often be efficiently combined with sulphate of magnesia. Sulphur-baths are also said to be useful. Paralysis, neuralgic pains, and other nervous symptoms must be treated according to the principles previously laid down. Galvanism to the muscles and musculo-spiral nerve is frequently of great service in the treatment of lead-paralysis.

## II. MERCURIAL POISONING.

Individuals who work with mercury are liable to peculiar tremors from the inhalation of this metal, and these have also occasionally followed its medicinal employment. There are the usual signs of mercurialization in connection with the mouth and general system. The tremors almost always begin in the upper limbs, being accompanied with numbness or formication, and pains in the joints, but may afterwards extend to the legs, trunk, face, tongue, and respiratory muscles; in short, to all the muscles except those of the eyeballs. At first the movements are but slight, but afterwards they increase so as to become spasmodic or convulsive, voluntary acts being performed in a violently jerking or spasmodic manner. They are greatly increased by any mental excitement. Finally more or less trembling becomes constant, and the patient is rendered quite helpless as regards voluntary movements, speech and breathing being also gravely affected. In most cases the tremors subside if the patient is supported in a sitting or recumbent posture, and they also cease during sleep. Stimulants diminish them temporarily, but they are worse afterwards. The tremulous muscles are decidedly weak. In very advanced cases serious nervous symptoms arise, such as sleeplessness, delirium, coma, or epileptiform convulsions.

TREATMENT.—As soon as any of the symptoms above described appear, the patient should immediately give up his occupation for a time. For the elimination of mercury from the system, the chief remedies are warm, vapour, or sulphur baths; sulphur or iodide of potassium internally; and *purgatives*. Medicinal *diaphoretics* and *diuretics* may also be employed. For the nervous symptoms, quinine, iron, opium, nitrate of silver, and galvanism are recommended.

---

CHAPTER LXXXVI.

## DIPHThERITIC PARALYSIS.

ÆTIOLOGY AND PATHOLOGY.—Diphtheritic paralysis is the most pronounced type of a class of cases, in which paralysis follows an attack of certain fevers and other acute diseases. Thus it has been noticed after typhoid and relapsing fevers, scarlet fever, small-pox, pneumonia, cholera, dysentery, and rheumatic fever. As regards diphtheria, the symptoms generally supervene during the period of convalescence, or when the patient seems to have quite recovered, the interval varying from a few days to some weeks. Dr. Abercrombie found that in sixteen cases the paralysis appeared in from two to five weeks from the commencement of the diphtheria. Occasionally, however, it sets in during the acute stage of the disease, even at a very early period. Another important fact is that the paralytic phenomena may follow the mildest attack, so mild, indeed, that the diphtheria has not been diagnosed, there having been apparently only a slight sore-throat. It has been stated, however, that they are more frequent and marked after severe attacks of the disease; but Dr. Abercrombie concludes that the tendency

to the nervous affection is at least as severe after a mild attack as after a severe one.

With regard to the *pathology* of diphtheritic paralysis, this is very uncertain. By some it has been regarded as merely the consequence of anæmia and debility, produced by diphtheria and other affections, and of a functional nature. Dr. William Squire is of opinion that this and allied forms of paralysis may be produced in three ways:—1. By the general effect of waste matters in the blood, where fever has been suddenly high, and the means of elimination insufficient. 2. By the effects of local inflammation on the innervation of the part, with or without change in the nutrition of the nerve-tissues. 3. By nutrition changes in the nerve-centres, as a consequence of altered blood-supply during or after the illness.

The results of *post-mortem* examination in fatal cases of diphtheritic paralysis are not conclusive. The nerves have been found diseased, disintegration of grey and white nerve structure, and of the nerve-axis having been described by Sir W. Gull, Charcot, and Dèjerine; the last observer has examined the anterior and posterior roots of the spinal nerves, and found the latter invariably healthy. Some have supposed that diphtheritic paralysis may originate in a local change in the nerves, which are either functionally disordered from over-stimulation, or become structurally diseased, and that the nerve-centres become secondarily affected, either by direct extension of morbid changes along the nerves, or by consecutive functional derangement. Dr. Abercrombie has examined the spinal cord and medulla oblongata in fatal cases of diphtheria. There were no lesions discoverable with the naked eye. He found a swollen condition of the large motor cells in the grey matter of the anterior cornua of the cord. Their margins were very ill-defined, and the processes had in most instances entirely disappeared. The contents had a granular aspect; and the nuclei had disappeared, or where still visible were highly granular. These changes only occur in very limited areas, and are not constant in any one region of the cord, but are most common in the upper and middle dorsal regions. Where one cell of a group is affected all the cells of that group show some change. In some places the cells appeared shrunken rather than swollen. The examination of the medulla oblongata in one case only revealed that some of the outermost cells of the vagus nucleus were rounded and more or less completely deprived of their processes. Dèjerine has described similar changes, but more advanced; and they have also been found by Dr. Percy Kidd. Whether these appearances support the view which is maintained that diphtheritic paralysis depends on primary changes in the nerve-centres, or whether they are of a secondary nature, must be determined by future observations.

**SYMPTOMS.**—Diphtheritic paralysis varies considerably in different cases as regards its extent, severity, and duration. In some instances the phenomena are localized, especially in connection with the pharynx and palate, and the latter may be alone implicated; the voice then becomes altered, and swallowing is performed with difficulty, which condition may be merely transitory, or may last for a considerable time. This limited and temporary paralysis is especially observed when the condition occurs during the acute stage of diphtheria. The more severe form of diphtheritic paralysis is characterized by being more or less progressive, attacking different parts in succession, so that ulti-



mately the whole body may become implicated. It is very slow and insidious in its origin; and starts usually in the throat and palate. The voice consequently becomes snuffling, nasal, or inarticulate; while deglutition is difficult, fluids being apt to pass into the posterior nares, and fluids or solids giving rise to a choking sensation, with violent irregular action of the muscles, and a choking spasmodic cough, which may be the first symptom noticed. At the same time the mucous membrane covering this part is more or less deficient in sensibility, the soft palate being often quite anæsthetic. Dr. Abercrombie noticed that usually these symptoms were followed by weakness in the legs, back, and arms in succession. Occasionally this order is reversed, the legs being first involved; and in exceptional cases the arms are affected at the outset. The implication of the limbs is indicated at first by tingling and numbness in the toes and fingers, with impairment of touch, which phenomena spread upwards, power at the same time becoming diminished, so that at last the patient has no control over the voluntary movements, and cannot stand or move. After a time the muscles waste and become flabby. The tongue, lips, and cheeks are often affected, but Dr. Abercrombie only noticed slight and transitory facial paralysis in three cases. The cheeks may become flabby and lose their expression; the lips flaccid, allowing the saliva to escape; and the eyelids may droop. The speech in diphtheritic paralysis is variously modified, according to the implication of the different parts concerned in the act. It is frequently thick, nasal or guttural, stammering, or slow; or the voice may be more or less weakened until it becomes a mere whisper. Vision is often affected, and may become suddenly impaired. Amblyopia is most common, but there may be presbyopia, myopia, or diplopia. The pupils are always dilated and sluggish, and may be unequal. Strabismus is not uncommon, which Dr. Abercrombie found was always convergent. There are no ophthalmoscopic changes. Other special senses are sometimes affected. The head sometimes rolls from side to side, owing to paralysis of its supporting muscles. The bladder may be involved, giving rise to retention and dribbling of urine; or there is marked constipation, owing to the abdominal muscles and intestines being affected. Involuntary passage of urine or fæces only occurs in the last stages. In some cases urgent danger arises from the respiratory muscles being attacked, so that breathing cannot be carried on properly, and pulmonary congestion is very liable to arise. Serious symptoms may also supervene in consequence of implication of the heart, its beats becoming very infrequent, being sometimes reduced to 16 per minute, as well as slow and weak, and finally the organ may entirely cease to act. Sudden death from syncope may occur when the heart is affected. Dr. Abercrombie noticed irregularity of the cardiac action as an early symptom, which subsided often after the patient had been confined to bed for a few days.

Abnormal sensations are often complained of in various parts, as well as hyperæsthesia and tenderness. Local or general anæsthesia is frequently present in diphtheritic paralysis. The tendon reflexes may be lost.

PROGNOSIS.—The duration of the nervous symptoms associated with diphtheria varies much. Usually the termination is favourable in adults, provided the respiratory muscles and heart do not become involved, but in children it is frequently fatal. Even very grave cases may ultimately recover.

TREATMENT.—In the treatment of diphtheritic paralysis one of the essential points is to promote the general health by good nutritious food; by healthy hygienic surroundings, with plenty of fresh air; and by *tonics*. Ferruginous preparations, quinine, and strychnine are the most serviceable medicinal agents, and these may be required for a long time. Friction with stimulating liniments, shampooing, or the application of small blisters, often prove useful in connection with paralyzed parts. The employment of galvanism is of the greatest service in treating the paralyzed muscles, but this agent must be used very carefully and judiciously. In protracted cases change to the sea-side, seawater baths, and hydropathy may prove beneficial. Should the respiratory muscles become involved, large mustard poultices may be applied over the chest. For cardiac paralysis Duchenne has recommended faradisation of the præcordial region.

---

#### CHAPTER LXXXVII.

#### SUN-STROKE—INSOLATION—COUP DE SOLEIL.

ÆTIOLOGY.—Long-continued exposure to the direct and powerful heat of the sun often gives rise to grave nervous symptoms. These are chiefly met with in soldiers, and of course cases of sun-stroke are by far most frequent in tropical climates, but several have occurred in this country during late years. There are certain powerful *predisposing* causes, namely, wearing heavy or tight clothing and accoutrements; physical fatigue and exhaustion; the state of system induced by overcrowding and bad ventilation; and deficiency of drinking water. Most authorities are of opinion that a moist atmosphere is worse than a dry one. The immediate cause of sun-stroke is believed to be interference with evaporation and radiation from the skin, so that the blood gets overheated, and thus exerts an injurious and depressing effect upon the nerve-centres.

ANATOMICAL CHARACTERS.—The only *post-mortem* appearances which have been observed in cases of sun-stroke are fluidity of the blood; some congestion of the brain generally; and extreme pulmonary congestion, with distension of the right heart.

SYMPTOMS.—Generally there are *premonitory* symptoms of sun-stroke, namely, great heat and dryness of the skin, with a subjective feeling of burning or stinging, the temperature being often hyperpyrexial; marked debility and sense of exhaustion; thirst and nausea; vertigo, but not often headache; conjunctival redness; frequent desire to micturate; and sometimes delirium or delusions. Dr. Muirhead describes three varieties of the actual attack, named respectively *cardiac*; *cerebro-spinal*; and *mixed*. In the *cardiac* variety there is sudden syncope, often terminating in speedy death. The *cerebro-spinal* form is characterized by coma; hurried, laboured, noisy, or stertorous breathing; contracted and immovable pupils; reddened conjunctivæ; convulsions in many cases; tumultuous action of the heart, with a very rapid, and

in a short time a feeble, compressible, and irregular pulse. The temperature may reach  $112^{\circ}$  or more, and may continue to rise after death in fatal cases. Should recovery take place, sequelæ are liable to remain behind, such as constant headache, mental disturbance, choreiform movements, or a tendency to epileptiform attacks.

TREATMENT.—Attention should be at once paid to any premonitory symptoms of sun-stroke. As a rule the great remedy is the assiduous use of the cold douche over the head, neck, and chest, many repetitions of which may be required, but care is necessary in its employment. It helps to lower the temperature, and to restore the breathing. The wet sheet, with constant fanning; enemata of iced water; and application of ice to the shaven head and spine are also recommended. Subcutaneous injection of quinine has been found useful in some cases. The patient should drink iced water freely, if he is conscious. If coma persists, a blister may be applied to the nape of the neck or to the shaven scalp. The bowels should be freely opened by enemata. The patient must be properly supported by nutriment and stimulants; and medicinal stimulants may be useful, especially in syncopal cases. Inhalation of chloroform is recommended for the relief of severe convulsions.

---

## CHAPTER LXXXVIII.

### ACUTE CEREBRAL INFLAMMATIONS.

#### I. SIMPLE OR PRIMARY MENINGITIS.

ÆTIOLOGY.—The *exciting causes* to which simple meningitis has been ascribed are:—1. *Direct injury* to the membranes, especially from fracture of the skull. 2. *Disease of the cranial bones*, particularly of the temporal in connection with ear-affections. 3. Prolonged direct *exposure to the sun*. 4. *Excessive mental labour*. 5. *Erysipelas* of the head and face. 6. *Local irritation* from adventitious growths, &c. 7. *Exposure to cold and wet*. 8. Certain *acute exanthemata* in rare instances. 9. Sudden *disappearance of chronic cutaneous eruptions* (?). Inflammation of the cerebral membranes is also a part of cerebro-spinal fever; and may result from extension upwards of spinal meningitis. The disease is most frequently met with in male adults. A hot climate and season; undue mental work, especially if combined with loss of sleep; a weak and exhausted condition of the system, from previous illness or any other cause; intemperate habits; and the presence of Bright's disease, are regarded as *predisposing causes*.

ANATOMICAL CHARACTERS.—As a rule acute simple meningitis involves the membranes extensively, but it is most marked over the convexity of the cerebral hemispheres. It may, however, be localized, or be evident chiefly or solely about the base. When the dura mater is affected, which usually results from injury or bone-disease, the inflammation is localized, and the membrane may be softened and thickened, reddened, black and sloughy, or unusually adherent to the surface of the brain; occasionally exudation or pus collects between it and the



bone, and if pus forms, it is apt to perforate the dura mater, and to escape into the arachnoid cavity. Sometimes also inflammation is set up in the venous sinuses, leading to the formation of a thrombus, which may soften into a purulent-looking fluid, and give rise to embolism and blood-poisoning. Generally in cases of meningitis the cerebral arachnoid appears dry and parchment-like, and more or less opalescent or opaque; sometimes it presents over its surface a thin layer of exudation or pus. The pia mater is extremely red and vascular, more so in some parts than others, while frequently small extravasations are observed, with patches of opacity around. In the early period a small quantity of serum, clear or more generally turbid and flocculent, and sometimes blood-stained, is seen in the arachnoid sac, and in the meshes of the pia mater. More commonly there is little or no fluid, but a soft yellowish opaque exudation, often more or less purulent-looking, covers the surface, being particularly abundant in the sulci between the convolutions, and along the course of the larger vessels. When the inflammation affects the base, the exudation involves some of the cranial nerves. The brain frequently presents an inflammatory condition of the superficial layer of its grey matter, especially in prolonged cases, indicated by redness, softening, and adhesion to the pia mater. The ventricles are normal in many cases, but may contain excess of serum or pus, or their walls may be covered with exudation.

**SYMPTOMS.**—Acute meningitis is usually preceded by *premonitory* symptoms, such as increasing headache or a sense of heaviness; vertigo; disturbances of general sensation or of the special senses; irritability, with a feeling of depression and restlessness; or sickness. The immediate attack is in most cases ushered in by a marked rigor or feeling of chilliness, speedily followed by pyrexia, and severe headache, with cerebral vomiting. In exceptional instances the first symptoms are epileptiform convulsions, hemiplegia, aphasia, or stupor ending in coma. The clinical history of the established disease in typical cases is divided into certain stages, as follows:—

**I. Stage of excitement.**—At this time the symptoms may be arranged thus:—*a. Local.* Intense and constant headache, in most cases chiefly frontal, of a tight or binding character, with sudden darting or plunging exacerbations, which may be so violent as to elicit sharp cries or shrieks, the pain being increased by any slight disturbance, such as movement, noise, or light; marked vertigo; great heat of head, with flushing or alternate flushing and pallor of the face; and conjunctival injection. *b. Mental.* Great irritability and unwillingness to be disturbed, with sleeplessness, culminating speedily in delirium, almost always of an active character, and not uncommonly almost maniacal, the expression being wild, staring, and savage, or sometimes indicating great terror, and the patient shrieking and gesticulating, or being very violent. Occasionally the delirium is more of a muttering kind. *c. Sensorial.* General hyperæsthesia; tingling or formication in various parts; diplopia or dim vision, marked photophobia, photopsia, or muscæ volitantes; tinnitus aurium, and undue sensibility to sound. *d. Motor.* General restlessness and jactitation; twitchings or spasmodic movements in various muscles, especially those of the face and limbs, either unilateral or bilateral; or sometimes general convulsive movements, rigidity, tetanic spasms, local or unilateral spasms. Slight strabismus is usually observed, and it may be very

distinct, while the eyeballs move about convulsively or stare fixedly. The pupils are very variable, but most frequently contracted or oscillating. *e. Extrinsic.* There is marked pyrexia, without prostration, the skin being very hot and dry; the temperature considerably raised; the pulse remarkably frequent, hard, and sharp; the tongue white, and the mouth clammy; with great thirst and loss of appetite. Cerebral vomiting is a prominent symptom; and also constipation as a rule, the stools being offensive and dark. Breathing is generally irregular and moaning. The duration of this stage may vary from one to fourteen days or more.

**II. Stage of transition.**—This stage is characterized by the cessation of the symptoms of excitement just described, with the development of those indicating failure of the cerebral functions, and there may be apparently a remarkable improvement at its commencement. Generally the change is more or less gradual, but may be very rapid, a sudden fit of convulsions occasionally ushering in the final stage. As a rule the headache, delirium, exalted sensations, and fever subside; while a tendency is observed towards heaviness, somnolence, or muttering stupor ending in coma, with cutaneous hypæsthesia or anæsthesia, and impairment of sight and hearing. Motor disturbances become more prominent and general, in the way of carphology, subsultus tendinum, twitchings or tremors, spasmodic movements or convulsions, or paralysis. The pupils become dilated and motionless. The body and limbs cool down considerably, though the head may still remain hot; the pulse is less frequent, but very variable, and sometimes intermittent; the tongue tends to become dry and brown. Respiration is irregular and sighing. Urine is retained and may overflow. These symptoms culminate in:—

**III. Stage of depression,** in which there is complete abolition of all the cerebral functions, as shown by absolute coma with stertorous breathing; general anæsthesia, with muscular paralysis and relaxation; great dilatation and immobility of the pupils; and involuntary escape of fæces and urine. The patient presents an aspect of extreme prostration and adynamia, the features being shrunk and ghastly; the surface bedewed with cold clammy sweats; the tongue dry and brown; the teeth and gums covered with sordes; while the pulse is excessively rapid, thready, and fluttering. In this condition the patient sinks more or less rapidly.

**VARIETIES.**—Differences are observed in the symptoms of meningitis according to the seat and extent of the inflammation. If it affects only one hemisphere, there may be hemiplegia. If it is localized, the symptoms are correspondingly limited. When the base of the brain is most affected, it is said that the pain is more sub-orbital and sub-occipital; the mental and sensorial excitement is less marked, delirium being comparatively slight and transient: special paralysis of some of the cranial nerves is observed; while coma sets in early, and speedily becomes profound. Ophthalmoscopic signs are evident when the inflammation is conveniently situated, namely, those indicative of neuritis or ischæmia, and hyperæmia is commonly present.

Local inflammation of the *dura mater*, due to injury or bone disease, is generally very obscure in its clinical history. The symptoms which may indicate this condition are pain, at first localized, being often seated behind the ear, but gradually extending over the head; local tenderness over this part in some cases, or painful œdema; little or no cerebral disturbance at first, but in course of time gradual somnolence ending in

coma, or sometimes delirium and convulsions; rigors, which may be periodically repeated, with pyrexia; diminished fulness of the jugular vein on the affected side, if a thrombus forms; and signs of pyæmia, or of embolic deposits in other parts.

## II. TUBERCULAR MENINGITIS—ACUTE HYDROCEPHALUS.

**ÆTIOLOGY.**—This variety of meningitis is immediately due to local irritation set up by tubercles in connection with the membranes of the brain. Therefore all causes predisposing to tuberculosis may be considered as predisposing to tubercular meningitis; and where a hereditary tendency exists, whatever leads to local excitement in connection with the brain, such as unduly forcing the mental faculties in young children, tends to give rise to the formation of tubercle in this part. Children are by far the most frequent subjects of tubercular meningitis, especially those from two to ten years of age, but the disease may be met with from earliest infancy to old age, being not uncommon up to the time of puberty and in young adults. Hereditary predisposition can be traced in a large majority of cases. This complaint not unfrequently follows one of the exanthemata.

**ANATOMICAL CHARACTERS.**—Miliary tubercles are found in greater or less abundance in the meshes of the pia mater, often adhering to the under surface of the arachnoid. They are frequently whitish and opaque, or may be softened and yellowish in the centre. They may appear scattered all over the surface, but are principally seen about the base of the cerebrum; in the fissures, especially the fissure of Sylvius; and along the chief branches of the vessels. The membranes are injected, particularly the pia mater. The surface of the arachnoid feels sticky, and a thin layer of soft lymph or puriform matter can often be scraped off; this substance usually collects in abundance between the arachnoid and pia mater, especially about the base and in the fissures. The pia mater is thickened, and its meshes are infiltrated with the same material or with serum. As a rule there is little or no fluid in the arachnoid sac. Occasionally the signs of inflammation are chiefly observed over the convexity. The ventricles of the brain generally contain a considerable quantity of colourless, usually somewhat turbid and flocculent serum, often amounting to some ounces in each lateral ventricle, and this leads to œdema, maceration, and softening of the surrounding brain structure; to dilatation of the spaces and their communicating channels; as well as frequently to compression of the convolutions of the cerebrum against the skull, so that they appear flattened and pale. Sometimes when the dura mater is opened the brain gives way, and the serum escapes. The walls of the ventricles are generally covered more or less with fine granulations. The exact appearances vary considerably in different cases; in some the signs of meningeal inflammation are most prominent; in others the quantity of fluid in the ventricles is most striking. There is no necessary proportion between the amount of tubercle and of the products of inflammation. Tubercle is generally present in other structures, and sometimes in the brain itself.

**SYMPTOMS.**—Tubercular meningitis in children is generally preceded for a variable period by *premonitory* symptoms indicative of tuberculosis; while nervous symptoms are often prominent, such as fretfulness, drow-



ness, sudden starting, screaming, grinding of the teeth during sleep, headache, vertigo, or a staggering gait. These may, however, be entirely absent; while in some cases the disease supervenes on long-standing tubercular mischief in other parts. The symptoms of the established disease are described as following certain stages, and these have been differently classified by different writers. Certainly they are often very indistinctly marked in practice, and cases present considerable variety in their clinical history. Tubercular meningitis may be very obscurely indicated, being but a part of general acute tuberculosis; or its own special symptoms may be those which chiefly attract attention. The characteristic phenomena are usually those of basic meningitis, with general cerebral excitement; followed by total abolition of the cerebral faculties, owing to the pressure of the fluid on the brain-substance.

The *invasion* is in most cases more or less gradual, and not infrequently very insidious. Sometimes the disease sets in very rapidly or even suddenly. The chief invasion-symptoms which may be met with are severe vomiting; intense headache; rigors, followed by pyrexia; marked irritability, nervousness, and obstinacy or unreasonableness in behaviour; or drowsiness. Occasionally tubercular meningitis is revealed by sudden convulsions, delirium, coma, or paralysis.

The ordinary clinical history of the developed disease is more or less as follows:—The early symptoms are severe constant headache, generally frontal, increased by movement, light, or noise, with intense darting paroxysms, causing the child to scream or cry out—the *hydrocephalic cry*, and to hold the head; vertigo, giving rise to staggering and a tendency to cling to surrounding objects; alternate flushing and pallor of the face, the expression being often frowning or sad, or sometimes vacant and stupid; heat of head; marked intolerance of light and sound; general hyperæsthesia or dysæsthesia; great irritability and peevishness, with unwillingness to be disturbed, to answer questions, or to take food; insomnia or very disturbed sleep; sometimes slight wandering at night, but no marked delirium; unsteady gait, with dragging of the limbs; constant restlessness; grinding of the teeth; alternate contraction and dilatation of the pupils; severe vomiting; usually obstinate constipation and retraction of the abdomen; complete anorexia, without any particular thirst, the tongue being furred, and the breath offensive; moderate but irregular pyrexia, the temperature not often rising above  $101^{\circ}$  or  $102^{\circ}$  in the evenings, the skin being usually harsh and dry, the pulse rather frequent, but easily hurried to 120 or more, and the urine concentrated, but very deficient in chlorides, phosphates, and urea. *Tâches cérébrales* are often easily produced. Subsequently the mental faculties become more disturbed, as evidenced by delirium in some cases, either wild and restless, or muttering; and increasing drowsiness, with tendency to stupor. General sensibility becomes impaired, and the hyperæsthesia of the special senses ceases, while signs are developed pointing to implication of the cranial nerves at the base of the brain, such as dim or double vision or hemiopia; tinnitus aurium and partial deafness; twitchings about the face; strabismus; oscillation of one or both eyeballs; dilatation, inequality, or marked oscillation of the pupils, these being not very sensitive to light. The face assumes a worn, aged, distressed expression, the eyes being half closed. Vomiting ceases, and diarrhoea may set in. Fever diminishes, while cool sweats often break out, and the pulse becomes in many cases remarkably infrequent and slow, but at the same time extremely variable and fluctuating, as well

as often irregular in rhythm and force. Respiration becomes sighing or moaning, and irregular. Still later there is marked general motorial disturbance, as evidenced usually by violent, prolonged, and frequent fits of convulsions; tetanic rigidity, the head being drawn back, boring into the pillow, or rolling from side to side; subsultus tendinum; tremulousness of the limbs; local paralysis or hemiplegia; or occasionally by cataleptic phenomena. The face exhibits grimaces, on account of the muscular twitchings, with partial paralysis; the eyes are half closed, dim, and covered with a film. When not convulsed the child is generally picking at the bed-clothes, or boring the fingers into the ear or nostril. The pupils are dilated and motionless. The final symptoms include gradual anæsthesia of all the senses; deepening coma; general muscular relaxation, with slight twitchings; involuntary passage of urine and fæces; coldness of the extremities, with general cold sweats; and an extremely rapid, feeble, and irregular pulse. Death may take place from gradual coma, or during a fit of convulsions. In some cases the temperature rises considerably before death, or it may sink much below the normal. The ophthalmoscope reveals hyperæmia of the disc, ischæmia, or optic neuritis; but only in very rare instances have tubercles been seen in the choroid. Sometimes the head becomes enlarged, the fontanelles at the same time being very prominent, and presenting pulsation if they are not closed up.

When tubercular meningitis affects only the convexity of the cerebrum, Dr. Gee states that the prominent symptoms are a constant convulsive state, with moderate pyrexia, and a pulse which is rapid and very variable in its frequency.

The ordinary *duration* of cases of tubercular meningitis in children is said to be from 7 to 23 days. Rilliet states that when prodromata are wanting it averages from 20 to 30 days. When the convexity is involved the disease terminates in one or two weeks, or even sooner. In the course of a case a remarkable remission in many of the symptoms is frequently observed, simulating recovery, but some of them still remain, and it is very important not to mistake this improvement for an indication of approaching convalescence.

In the *adult* tubercular meningitis is generally considered as being in most cases secondary to chronic tubercular disease, especially to pulmonary phthisis, the symptoms of which often improve markedly just before those of meningitis are developed. Gee states, however, that *primary* tubercular meningitis is at least as common as *secondary*. The symptoms more or less resemble those observed in the child, the most prominent being severe frontal headache with darting paroxysms; heat of head, with redness of the face or alternate flushing and pallor, and suffused conjunctivæ; often a dull, bewildered, heavy, or stupid expression, with mental confusion; a tendency to somnolence and stupor, alternating with wild delirium; indisposition to speak, or sometimes sudden complete aphasia; photophobia and intolerance of sound; evidences of irritation or paralysis of some of the cranial nerves, such as twitchings or paralysis about the face, ptosis, dilated or unequal pupils, strabismus; convulsive seizures; paralysis of the limbs; and cerebral vomiting. Deep coma follows, with general paralysis, and involuntary passage of fæces and urine, terminating in death.

## III. RHEUMATIC MENINGITIS.

The meningitis which exceptionally complicates acute rheumatism has been distinguished as a special variety, but it only requires to be just mentioned here. Its development is usually accompanied with marked diminution in the joint-symptoms. The symptoms are those of simple meningitis, but it is said that the early stage is less violent, and the progress of the case more rapid. It must be remembered that grave cerebral symptoms may arise in the course of rheumatic fever, which are independent of any meningeal inflammation.

## IV. ACUTE CEREBRITIS OR ENCEPHALITIS—CEREBRAL ABSCESS.

**ÆTIOLOGY.**—Inflammation of the brain-substance may result from :—  
 1. *Injury* caused by fractures, wounds, or mere concussion. 2. *Disease of the bones*, especially in connection with chronic ear-affections, or occasionally with acute disease of the internal ear. 3. *Extension* from meningitis. 4. *Local irritation*, by adventitious morbid products, extravasated blood, or spots of softening. 5. Various *acute and chronic diseases*, especially if attended with suppuration, the inflammation being then probably pyæmic or septicæmic in character. Thus cerebritis has been met with in low fevers, especially typhus; acute pneumonia; chronic pulmonary phthisis; dysentery; and in connection with abscesses in different parts of the body. 6. *Insolation*. 7. *Prolonged mental labour* possibly. Sometimes no cause can be made out.

**ANATOMICAL CHARACTERS.**—Cerebritis is described as *diffuse* or *general*; and *local*. The former does not imply that the whole brain is implicated, which is never the case, but merely that there is extensive inflammation of the superficial grey matter, this condition being only associated with meningitis, and evidenced by redness, softening, and adhesion to the pia mater when this is stripped off. *Local* cerebritis is limited to one or more spots of variable dimensions. Some pathologists regard this morbid change as being the invariable cause of *acute softening* or *ramollissement*, especially of *red softening*. As will be hereafter pointed out, however, it is far more likely that the condition thus described is in the great majority of cases due to other pathological causes. When resulting from inflammation, it is said that the specific gravity of the softened portion is increased. It is supposed that the colour may become yellow or green, from infiltration of the affected tissue with exudation or pus. The most important termination of local cerebritis is the formation of an abscess, which only happens, however, in connection with injury, bone-disease, or pyæmia. Usually the abscess is single, but there may be several collections of pus, especially in pyæmic cases. The white substance in the centre of the hemisphere is its most common seat, but any part of the brain may be involved, and when the abscesses are numerous, they are usually situated chiefly near the surface. Their size varies as a rule from that of a pin's head to that of a nut or egg; but a hemisphere may be occupied by one large abscess, which alters its shape and flattens its convolutions against the skull.



When there are several abscesses they are usually small. Their shape is irregularly round or oval. At first the walls are ragged, softened, or inflamed; but if the disease lasts for some time, a firm fibrous or fibro-cellular capsule is formed, which may attain considerable thickness, becoming lined by a smooth membrane. The pus may be tolerably healthy, yellow or green, or sometimes red from admixture of blood; but in old abscesses it becomes unhealthy, foetid, and alkaline, containing but few pus-cells, with abundance of granular matter. A cerebral abscess may burst in various directions, such as on the surface of the brain; into a ventricle; into the tympanum; or in rare instances externally. In other cases the contents become inspissated, cheesy, or calcified, and a firm capsule forms.

**SYMPTOMS.**—The special characters of the clinical phenomena of cerebritis, as distinguished from those of meningitis, are that they indicate but slight and brief excitement, or none at all; while signs of failure of the cerebral functions speedily set in. The *diffuse* form is always preceded and accompanied by symptoms of meningitis, and in proportion as these are but little marked and of short duration, and to the rapidity with which stupor and coma, sensory anæsthesia, convulsions, and paralysis set in, the more probable is it that the brain itself is involved. Pyrexia also is not so high as in meningitis. *Local* cerebral inflammation is always very obscure at the outset. Frequently there is a severe prolonged rigor at first, which may be repeated on several days with almost regular periodicity. Sometimes, without any particular previous symptoms, the patient is seized with an apoplectic or epileptiform seizure, or gradual coma sets in; occasionally sudden hemiplegia without loss of consciousness has been observed. As a rule, however, there are early symptoms, namely, deep and sometimes fixed headache, often considerable, of a dull character, but not intensified into violent paroxysms; vertigo; heat of head; restlessness and sleeplessness; a heavy expression; mental confusion; irritability; sometimes talkative but not violent delirium; dimness of sight; partial deafness; sensations in various parts of the limbs of tingling, numbness, formication, or deep pain or coldness; general weakness and languor; with tremors, twitchings, rigidity, or paralysis of various muscles. The pupils present all possible variations. There is comparatively little pyrexia. Vomiting is not infrequent. The bowels may be very constipated, and the tongue much furred. Sometimes articulation is impaired, or the patient is disinclined to speak, or complete aphasia may be observed. The subsequent symptoms in fatal cases are stupor, ending in coma; gradual loss of all sensation; convulsions, hemiplegia, or general paralysis, usually with rigidity or tetanic spasms; and involuntary escape of urine and fæces. Some cases do not end fatally, but permanent disorder of the mental, sensory, or motor functions usually remains. In pyæmia it is rarely possible to diagnose cerebral abscess; and in some instances the symptoms closely resemble those of some low fever. Very exceptionally a collection of pus in the brain bursts externally.

## V. GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. **DIAGNOSIS.**—*a.* Acute inflammatory diseases connected with the brain or its membranes have to be distinguished from various *extrinsic* diseases attended with severe nervous symptoms, and this particularly

applies to tubercular meningitis occurring in children. The principal affections of this class with which these diseases are liable to be confounded are the exanthemata, especially typhoid and typhus fever; pneumonia and other acute inflammatory affections; disorder of the alimentary canal in children, particularly if acute and attended with fever and marked cerebral disturbance; anomalous cases of fever with brain-symptoms; great exhaustion of the vital powers, especially as the result of bad feeding, prolonged diarrhoea, or some lowering illness; and various derangements giving rise to reflex convulsions or delirium. The *history* of the case, as revealing some cause of cerebral inflammation, or of one of the other complaints mentioned above; the *age*, *constitutional condition*, and *hereditary tendencies* of the patient; the *mode of onset* of the attack; careful investigation regarding the *symptoms*, particularly as to the degree and character of those referable to the head, and the presence or absence of indications of cerebral lesion in the way of motor or of sensory disorder, the intensity and course of pyrexia, the symptoms connected with the alimentary canal, and those characteristic of the various fevers; *physical examination* of the different organs; and the *course* and *progress* of the case, are the chief data to be relied on in diagnosis. It is frequently impossible in children to determine at first whether they are suffering from tubercular meningitis, or from one of the complaints above enumerated. Under such circumstances the only thing to be done is to watch the case carefully, and observe its progress, at the same time employing some judicious treatment, and the obscurity will in most instances before long be cleared up.

b. Inflammatory diseases have to be separated from *other affections of the nervous system*, which give rise to signs of cerebral excitement. Acute meningitis in an adult may be simulated by active congestion, in which, however, the symptoms are but temporary and usually slight, with little or no fever; by delirium tremens; or by acute mania. In the last two conditions the diagnosis rarely presents much difficulty, if proper attention be paid to the previous history and the symptoms present. In mania the existence of one or more fixed delusions, and absence of fever or of signs of vascular excitement about the head, are important points in diagnosis. In doubtful cases the supervention of spasmodic and paralytic phenomena, or of coma, usually soon reveals the existence of meningitis. Meningeal or cerebral hæmorrhage and cerebral tumour have in rare instances been mistaken for tubercular meningitis. The symptoms of acute inflammatory softening, and of that form due to thrombosis, often closely resemble each other; and occasionally cerebral hæmorrhage simulates cerebritis at first.

c. The diagnosis of *meningitis* from *cerebritis* and its consequences, and of *simple* from *tubercular meningitis*, calls for a few remarks. *Meningitis* and *cerebritis* are frequently more or less combined, but the latter is distinguished mainly by the symptoms of cerebral excitement being much less marked and of short duration, or even absent altogether; while those indicative of failure of the cerebral functions rapidly supervene; there being also much less local vascular excitement and general pyrexia. The chief circumstances by which *tubercular* is distinguished from *simple* meningitis are the age of the patient in many cases; the presence of signs of the tubercular diathesis, or a history of hereditary taint, and the absence of any other obvious cause of meningitis; the more marked and prolonged premonitory symptoms, with insidious mode of onset; the evidences of inflammation affecting

the base of the brain chiefly at first, there being less psychical disturbance, and no wild delirium; the headache being more in severe paroxysms; the minor degree of fever, or of local vascular excitement; the special characters of the pulse; and the less rapid course.

2. *PROGNOSIS*.—Any *acute inflammation* in connection with the brain or its membranes is extremely dangerous, and in the great majority of cases the termination is fatal. It is exceedingly doubtful whether tubercular meningitis is ever recovered from, at all events when the disease is fully developed, although cases of reputed cure have been brought forward. If a case should terminate favourably the cerebral functions are permanently impaired, more or less.

3. *TREATMENT*.—Unfortunately direct treatment is of very little service in any form of acute meningitis or cerebritis, and my own experience of a good number of cases at the Liverpool Northern Hospital is decidedly opposed to the measures usually recommended by some high authorities, namely, bleeding; severe purging; mercurialization; and extensive blistering. The measures which are most likely to be useful in the early stage are to place the patient in a well-ventilated, cool, quiet, and somewhat darkened room, on a comfortable bed, with the head high: to enjoin perfect freedom from every kind of disturbance; to cut the hair very short, or even shave the head, and apply cold assiduously, but cautiously, by means of the ice-bag or irrigation; to open the bowels tolerably freely, a dose of calomel or croton-oil being useful for this purpose, as well as the administration of enemata; to limit the diet to beef-tea and milk; and, if there are signs of vascular excitement, to apply a few leeches over the temples. Convulsions are best treated by bromide of potassium in full doses, especially in cases of tubercular meningitis. Opium must be avoided. In the later stages a blister may be applied to the nape of the neck, or a couple behind the ears, but the advantage of blistering the whole scalp, as has been advocated, seems to me very questionable. Should symptoms of *adynamia* set in *stimulants* are needed, especially brandy, ammonia and ether, with abundant liquid nourishment; if the patient is unconscious, they may be injected between the teeth by means of a syringe, or administered by enemata. Care must be taken throughout to keep the feet warm; to attend to thorough cleanliness; and to see that the bladder is properly emptied. Sinapisms and flying blisters over the limbs are recommended in order to endeavour to rouse the patient in the later stages, but they are of little use. Should meningitis arise in connection with rheumatic fever, application of sinapisms or blisters to the joints might be of service.

---

## CHAPTER LXXXIX.

### DISEASES AFFECTING THE CEREBRAL CIRCULATION.

THERE are four groups of morbid conditions which may be referred to the cerebral circulation, namely:—1. Excess or deficiency of blood in the vessels of the brain—*Cerebral congestion and anæmia*. 2. *Cerebral embolism and thrombosis*. 3. *Cerebral and Meningeal hæmorrhage*. 4. *Diseases of the vessels*.—Only the first three groups need be specially



considered here, for the diseases of the vessels have already been partly discussed, and will be further sufficiently noticed incidentally in dealing with other conditions, with the exception of one particular lesion, namely, aneurism, which it will be more convenient to consider along with tumours of the brain.

Without entering at any length into an account of the peculiarities of the cerebral circulation, it will be expedient to notice here certain points which are of practical importance. There is little or no communication between the arteries of the brain, except at the circle of Willis. Even the finest branches of a particular artery, such as the middle cerebral, do not anastomose, and only communicate through capillary vessels. Moreover, the arterial branches supplying the central ganglia are distinct from those distributed to the surface of the brain; while the arteries of the convolutions give off long and short branches, which are quite separate, the short ones being distributed to the cortical grey matter, the long to the subjacent white substance. Hence, if any artery is blocked up, the blood is entirely cut off from the area which it supplies, and the resulting lesions are correspondingly limited. In the case of the middle cerebral artery, for example, this vessel may be occluded in its main channel, the whole region which it supplies being thus deprived of blood; or only in one of its branches, so that the cortical grey matter and subjacent medulla may be the seat of disease from this cause, while the basal ganglia are quite healthy, and the lesion may be confined to a very limited region. The ophthalmic artery comes off from the internal carotid, and so forms a communication between the anterior and middle cerebral arteries, which come off from the same trunk, and the circulation in the eyeball and other parts to which this artery is distributed. The internal ear is mainly supplied from the basilar artery. With regard to the veins, the ophthalmic vein opens into the cavernous sinus; nearly all the venous blood within the skull is conveyed away by the lateral sinuses, and their continuations the internal jugular veins; and the chief sinuses communicate by branches passing through foramina in the skull with the veins on the outside of the head, and with those of the neck.

## I. CEREBRAL CONGESTION OR HYPERÆMIA.

**ÆTIOLOGY.**—The causes of hyperæmia of the brain are:—1. *General plethora*, especially that associated with excessive eating or drinking, and luxurious habits, with want of exercise. 2. *Increased flow of blood into the brain—active hyperæmia*—from undue cardiac action, whether merely functional, or associated with hypertrophy of the left ventricle; local irritation, especially in connection with inflammatory affections; diminished resisting power of the arteries, particularly that accompanying vaso-motor paralysis, as from excessive mental labour, strong emotion, sunstroke, or the effects of alcohol and other poisons; interference with the general arterial or capillary circulation, in consequence of which an extra amount of blood flows into the main arteries of the neck; or, it is said, atrophy of the brain. 3. *Interference with the escape of blood out of the brain—mechanical hyperæmia*—especially resulting from cardiac and extensive lung affections; violent expiratory efforts with the glottis closed, as in coughing or straining at stool; hanging the head downwards; direct pressure upon the veins returning the blood

from the brain, as by an aneurism or other tumour; or strangulation of the neck.

**ANATOMICAL CHARACTERS.**—The *post-mortem* appearances usually described as indicative of cerebral congestion are overloading of the venous sinuses and of the vessels of the meninges, including the finer branches as well as the larger veins, so that the pia mater appears extremely vascular and opaque; undue redness of the grey matter of the convolutions; and increased number and size of the drops of blood which are visible on making sections of the brain. The convolutions may be compressed, and the ventricles contracted. Niemeyer justly remarked that it is often difficult to decide whether the vessels of the membranes, and still more whether those of the brain-substance, have been congested during life. He considered that the number and size of the drops of blood on section depend chiefly on its fluidity, and that œdema may follow congestion, the brain-substance then becoming unusually pale, and presenting but few and small blood-spots. As a rule the signs of hyperæmia are equally distributed throughout, but sometimes they are more evident in some parts of the brain than others. Long-continued or repeated congestion leads to permanent enlargement and tortuosity of all the vessels; atrophy of the brain, with a moist and slimy condition of its substance; increase in the subarachnoid fluid; and, it is supposed, to hypertrophy of the Pacchionian bodies.

**SYMPTOMS.**—Persistent cerebral congestion is revealed ordinarily by more or less of the following symptoms:—Constant dull headache, not severe, felt all over the head, or chiefly at the vertex or behind; a sense of heaviness, fulness, and oppression in the head; vertigo, which is often a prominent symptom; some degree of mental disturbance, evidenced by dulness of intellect, confusion and slowness of thought, impaired memory, indifference, and indisposition for any effort or occupation; constant drowsiness, though sleep is unrefreshing and often disturbed by disagreeable dreams; photopsia, iridic colours, or specks before the eyes, or sometimes temporary diplopia or dim vision; partial deafness and noises in the head; a feeling of heaviness in the legs, especially after walking, with restlessness and fidgetiness, twitchings, or sudden startings; increased or impaired cutaneous sensibility; pains in the limbs and various paræsthesiæ, these disordered sensations being temporary and variable in their locality. These symptoms are rendered worse by taking a full meal, by mental effort or emotion, by physical exertion, and by the recumbent posture. There are often obvious signs of plethora about the face and head, with throbbing of the carotids.

Occasionally grave symptoms arise from cerebral congestion. The most important are those characteristic of an *apoplectic* attack. Various combinations of symptoms may be met with, but the distinctive features of this *congestive apoplexy* are as follows:—1. Its onset is quite sudden; it reaches its full development at once; and almost always follows some act on the part of the patient which leads to increased congestion in the head, such as hanging down the head, coughing, or straining at stool. 2. The coma is rarely complete, there being usually some indications of sensation, or should there be absolute loss of consciousness, this lasts but a very short time. 3. There is generally partial bilateral motor paralysis; very rarely hemiplegia, or more marked paralysis on one side than the other. 4. Rigidity is never observed, but slight bilateral clonic spasms are not uncommon, or they may be unilateral. 5. Respiration is not stertorous. 6. The pulse is generally slow, infrequent, and full.

7. There are external signs of hyperæmia about the head and face. 8. Urine and fæces are not passed involuntarily. 9. Restoration is rapid and ordinarily complete, no permanent mental defect or paralysis of motion or sensation remaining. Some degree of mental confusion may continue for a time, or there may be general impaired sensation with muscular weakness, but these phenomena soon pass away. The patient may be subject to attacks of a similar kind. Occasionally cerebral congestion gives rise to *epileptiform* seizures; and in some conditions it is said to be attended with delirium and fever.

## II. CEREBRAL ANÆMIA.

**ÆTIOLOGY.**—Cerebral anæmia may be *partial* or *general*. The former may arise from obstruction of some arterial branch, especially as the result of embolism or thrombosis; or in connection with certain intracranial diseases, such as cerebral hæmorrhage or tumour, which either compress neighbouring arteries or capillaries, or give rise to surrounding œdema. Anæmia of the entire brain occurs under the following circumstances:—1. In connection with *general anæmia*, from whatever cause this may arise, the blood being either deficient in quantity, or impoverished in quality and deficient in red corpuscles. 2. From *enfeebled* or *failing cardiac action*, cerebral anæmia being a prominent phenomenon of the syncopal state. 3. As the result of *withdrawal of blood* to other parts of the body, as in the use of Junod's boot. 4. Rarely from *obstruction or compression of the main arteries* supplying the brain. 5. In consequence of *the cranial cavity being encroached upon* by large tumours, hæmorrhages, and other morbid conditions; or by a fractured and depressed skull. 6. It is said, from *vaso-motor disturbance* affecting the arteries which supply the brain, these becoming consequently contracted.

**ANATOMICAL CHARACTERS.**—Cerebral anæmia is indicated by more or less deficiency or want of blood in the vessels supplying the brain; by pallor of its substance, the white matter being very pale and shining; and by the absence or small number and size of the spots of blood usually visible on making sections of the brain-substance.

**SYMPTOMS.**—It is highly probable that the symptoms which occur in connection with several of the morbid conditions which affect the brain are to some extent due to local anæmia of its substance. This partial anæmia is, however, of most importance, and most strikingly evident in association with embolism and thrombosis, and the phenomena observed will be more appropriately described when these pathological conditions are discussed.

In *general* cerebral anæmia the symptoms may be simply those of more or less sudden syncope, namely, loss of consciousness, with pallor, dilated pupils, and other phenomena, in some conditions these being associated with distinct convulsive movements; or they may come on gradually. The phenomena in the latter case are usually those already described under anæmia, such as headache, dizziness, disturbed vision, tinnitus aurium, &c. A condition met with in children as a result of lowering agencies, such as long-continued diarrhœa, which has been termed *hydrocephaloid*, has also been attributed to cerebral anæmia, the symptoms simulating those of acute hydrocephalus. In cases of starvation, as well as in some instances of mere general anæmia, there may be marked mental excitement, restlessness, and delirium, which may end in a violent maniacal condition.



### III. CEREBRAL AND MENINGEAL HÆMORRHAGE—SANGUINEOUS APOPLEXY.

**ÆTIOLOGY AND PATHOLOGY.**—Cerebral hæmorrhage is in the great majority of cases, excluding those of traumatic origin, the result of *structural changes in the minute vessels*, which diminish their resisting power, namely, atheroma or calcification; fatty degeneration; the formation of minute aneurismal dilatations on the small arteries, associated with a fibroid change; or the state of impaired nutrition which is induced by debilitating diseases, such as typhus fever or scurvy. Frequently, in addition to this, the vessels are not properly supported, owing to softening or atrophy of the brain-substance, and hence they are still more liable to rupture. Not uncommonly they give way spontaneously, but this event is far more likely to happen if a state of congestion is brought about in any way, but especially as the result of hypertrophy of the left ventricle, excited cardiac action, or interference with the return of venous blood from the brain. On this account cerebral hæmorrhage is liable to follow sudden effort; straining at stool; a fit of coughing; powerful emotion; hanging the head down; compression of the neck; exposure to the sun; a fit of drunkenness; a warm bath; or general exposure of the surface of the body to cold. Among *predisposing causes* may be mentioned advanced age; hereditary predisposition to early senile changes in the arteries; luxurious habits with want of exercise; and a state of general plethora and want of tone. Should there be signs of marked degeneration in the arteries, particularly if these are combined with left cardiac hypertrophy or dilatation of the right cavities of the heart, and with renal disease, cerebral hæmorrhage is to be feared at any moment. It must be mentioned that *embolism or thrombosis* of a large vessel in the brain leads to capillary extravasation into the surrounding area. In very rare instances hæmorrhage into the brain has resulted from the *rupture of a vascular tumour*.

*Traumatic injury* is the usual cause of meningeal hæmorrhage, but blood may find its way from the brain into or beneath the pia mater, or into the arachnoid cavity. Another important cause of hæmorrhage into the meninges is the *rupture of an aneurism* involving one of the main arteries at the base of the brain, especially the basilar, middle cerebral, or one of the communicating arteries. Meningeal hæmorrhage also occurs in connection with the condition named *pachymeningitis*. Effusion of blood outside the dura mater is always due to injury.

**ANATOMICAL CHARACTERS.**—The situations in which blood may be found extravasated within the cranium are as follows:—1. Into the substance of the brain. 2. Within the ventricles. 3. In connection with the pia mater. 4. Into the arachnoid sac. 5. Between the skull and dura mater.

The pathological anatomy of hæmorrhage into the brain must be considered at some length. *Seat.*—This is by far most frequently the corpus striatum or optic thalamus. Occasionally blood escapes into the pons, cerebellum, convolutions or medullary substance of the cerebrum, crus cerebri, medulla oblongata, corpora quadrigemina, or corpus callosum. Sometimes a part of the brain, as the septum lucidum, is torn through; or the blood makes its way into a ventricle, or out on to the

surface of the brain. *Amount.*—The quantity of blood extravasated varies from a few drops to several ounces, and the effusion may be so large as to alter the shape of a hemisphere, flatten its convolutions, and cause marked anæmia around. *Number of hæmorrhages.*—As a rule there is but one extravasation, but occasionally two or more are observed, though very rarely on opposite sides. Not uncommonly remains of former hæmorrhages are seen. *Recent characters and subsequent changes.*—The blood may accumulate in the form of what is termed a *capillary hæmorrhage*; or as a distinct *clot*. The former presents numerous scattered dark-red points of extravasation in the midst of cerebral substance, which is either normal, or frequently of a yellow or reddish colour, as well as softened, this condition constituting one form of *red softening*. A clot, if small, simply separates the brain fibres, but if it is large, the cerebral tissue becomes broken down and mixed with the blood, while the surrounding portion is torn, at the same time being often softened and discoloured from imbibition. At first the blood may be found to be quite fluid, or partially or completely coagulated into a soft clot. Subsequently it tends to set up inflammation around, and has been known even to give rise to an abscess. In favourable cases, however, the extravasation undergoes changes ending in its absorption. It separates into its fibrinous and serous portions; becomes decolorized by degrees, passing through stages of reddish-brown, brown, yellowish-brown, and yellow; or granular pigment and hæmatoidine crystals form. Proliferation of cellular tissue takes place around, forming a capsule, and the clot may in time be entirely absorbed, an apoplectic cyst remaining, containing fluid, often loculated, and this may also be removed ultimately, nothing being left but a firm, fibrous, pigmented cicatrix. It is even said that this may disappear, a loss of substance, with consequent diminution in the size of the brain, being thus occasioned. The nerve-fibres which lead from the seat of hæmorrhage to the spinal cord frequently undergo degeneration.

When blood collects in a ventricle it is not nearly so readily absorbed, and in many cases becomes organized. In connection with the membranes an extravasation is generally spread out, and forms a soft red coagulum. In its subsequent changes it becomes altered in colour, granular, and pigmented, the brain underneath being somewhat indurated. Finally it forms a depressed pigmented plate, with serum upon its surface.

The heart, vessels, and kidneys will be found diseased in many cases of cerebral hæmorrhage.

*SYMPTOMS.*—In a considerable proportion of cases of cerebral hæmorrhage *premonitory* symptoms have been noticed for a variable period, such as headache or a sense of heaviness in the head; vertigo; mental confusion and impaired memory; irritability of temper; disturbed sleep or drowsiness; disorder of vision or hearing; thickness of speech; slight or temporary limited paralysis about the face or limbs; local twitchings; impaired sensation or paræsthesiæ in various parts. These phenomena may be due to mere vascular disturbance; to the formation of minute thrombi; or to very small extravasations. The frequent occurrence of epistaxis has been considered an important premonitory sign of cerebral apoplexy; and also the discovery by the ophthalmoscope of clots in the retina. There are usually indications of degeneration of the vessels, as well as of cardiac disease and chronic renal mischief in many cases.

The precise clinical phenomena which result from the actual extravasation of blood into the brain differ very materially. This lesion never causes absolutely sudden death, though in rare instances a fatal termination has occurred within a few minutes. In the majority of cases the immediate symptoms which characterize cerebral hæmorrhage may be summed up as those of an *apoplectic seizure with hemiplegia*. The main features of the attack are as follows:—It may follow some evident cause which leads to cerebral congestion, but often comes on spontaneously while the patient is perfectly quiet. The seizure is usually more or less sudden, though not absolutely so, being almost always preceded by some immediately premonitory symptoms, which occasionally last for some time, such as mental confusion, pain in the head, disorder of speech, unilateral numbness, pallor with faintness, or sickness. Sometimes a convulsion ushers in the attack. When fully developed the coma is usually very profound at first, and the deeper it is the more likely is an apoplectic fit to be due to hæmorrhage rather than other cerebral lesions. The accompanying phenomena of the comatose state in a considerable proportion of cases of sanguineous apoplexy are flushing or even some degree of lividity of the face, with a turgid condition and fulness of the veins; slow, laboured, irregular, or stertorous breathing, with puffing out of the cheeks in expiration; and throbbing of the carotids, the radial pulse being infrequent, slow, laboured, full, and soft. Sometimes, however, signs of shock are noticed, the face being pale, and the pulse rapid, small, and feeble. The temperature is frequently lowered. Hemiplegia exists on the side opposite the lesion, having the extent of distribution described in the chapter on paralysis, but in many instances this condition is not easy to make out at first, when the coma is very profound, the whole body being paralyzed for the time. Sometimes tremors or spasmodic movements are observed in the paralyzed limbs. The head and eyes are usually turned to the non-paralyzed side, being drawn by the unantagonised muscles of the sound side, the patient seeming to be looking over the shoulder on that side, and often both upper eyelids fall. The pupils vary much, but they are generally equal and somewhat dilated; sometimes they are unequal, or very large and insensible to light.

As regards the progress of the symptoms, the comatose state may end in death, which rarely happens under some hours, and not usually for two or three days, some cases lingering for four or five days or even longer. Urine and fæces are then passed unconsciously; and secretions accumulate in the air-tubes, attended with loud rhonchal sounds. On the other hand, in a good proportion of cases consciousness is restored more or less speedily, and when this happens the mind is found to be perfectly natural, or there is only slight mental confusion, which soon passes off. Occasionally delirium is observed; or the mental faculties may be permanently enfeebled, the patient sinking in time into a state of more or less dementia. The hemiplegia becomes evident on the return to consciousness, and when the right side is affected aphasia is common, being also occasionally observed in cases of left hemiplegia. Sensation is not nearly so much affected as motion usually, there being merely a certain degree of impaired sensibility, or of numbness and tingling in the extremities, and even these disorders usually disappear before long. Now and then, however, permanent anæsthesia is noticed, either over the whole side or in limited spots, which is an indication of severe lesion. There is usually no complaint of head-symptoms or disorder



of the special senses, or if such exist, they speedily pass away unless the hæmorrhage is extensive. In a few days signs of more or less inflammation from irritation of the clot are generally developed, such as headache, heat of head, restlessness, slight delirium, disturbances of vision, and twitchings or spasmodic movements in the paralyzed parts. These symptoms soon subside ordinarily, but violent inflammation may be set up, ending in extensive softening or abscess, indicated by a relapse into the comatose state, with general paralysis and involuntary evacuations before death, which event may take place from this cause in three weeks or even later. Should a case proceed favourably, the motor paralysis often diminishes markedly in time, the improvement following the usual course, but the restoration is seldom complete, certain muscles generally remaining permanently disabled. In other cases there is little or no improvement, and "late rigidity" may ultimately set in.

The clinical variations due to the *seat* of cerebral hæmorrhage may be gathered from what has been stated in the chapter on LOCALIZATION OF NERVOUS DISEASES, but a few special points may be noticed here, as well as the variations due to the *extent* of the mischief. As regards either *cerebral hemisphere*, the degree and duration of the comatose state depend mainly on the amount of the extravasation. Hence, if this is moderate, there may be only partial loss of consciousness, the patient exhibiting signs of sensation and perception, while the mental faculties are very speedily and completely restored. If the extravasation is very small, there is no impairment of consciousness at all, and the lesion is merely indicated by *sudden hemiplegia*; or this is noticed when the patient attempts to move in the morning, the hæmorrhage having occurred during the night. The degree and persistence of the paralysis depend on the seat of the extravasation, as well as on its extent. Thus a small effusion into either *corpus striatum* or into the *internal capsule* will give rise to hemiplegia, and if it is of some size the paralysis is permanent. It may happen that a clot is so small or so situated that it either gives rise to no symptoms whatever; or only to partial hemiplegia, from which recovery is often rapid and complete. Hæmorrhage into the *cortical substance* is said to be usually attended with convulsions, and subsequently by marked mental disturbance, meningitis being often set up. When a very large clot occupies a hemisphere, so that the opposite one is also interfered with, bilateral paralysis is produced, though it is not equal on the two sides, only some degree of weakness of the limbs being observed on the same side as the lesion. General paralysis may also result from extravasation into both hemispheres, but this is extremely rare. If there is extensive *laceration* of the brain, rigidity and muscular twitchings are prominent phenomena. In some cases no impairment of consciousness is observed at first, or this is only partial and of brief duration, but afterwards, owing to increase of the hæmorrhage, or to the rupture of another vessel, profound coma sets in, ending in death.

*Ventricular hæmorrhage* is attended with deep coma and general paralysis, as well as in some cases with convulsions or marked rigidity and twitchings; or, more commonly, these symptoms follow the signs of hæmorrhage into one hemisphere, which is very significant. Considerable extravasation into the *middle of the pons Varolii* causes profound coma; general paralysis; marked contraction of both pupils; and usually speedy death. The condition greatly resembles that resulting

from opium-poisoning. Hæmorrhage into the *medulla oblongata* generally proves very rapidly fatal. When blood escapes into the *arachnoid sac* or *sub-arachnoid space*, the attack is usually not so sudden, but the symptoms are very variable. At the outset premonitory symptoms are frequently present, such as severe headache, vertigo, partial loss of motion, somnolence, or impairment of intellect. Among the signs which may be regarded as most suggestive of this form of hæmorrhage are gradually-developed coma; hemiplegia without implication of the face, or paralysis beginning on one side and afterwards extending to the opposite one; the occurrence of convulsions, marked spasmodic contractions, or rigidity of the limbs; and the supervention of signs of severe meningitis in a few days. Sensation is rarely affected. When meningeal hæmorrhage is considerable, it may be impossible to distinguish this condition from extravasation into the substance of the brain.

#### IV. CEREBRAL EMBOLISM AND THROMBOSIS—CEREBRAL SOFTENING.

**ÆTIOLOGY AND PATHOLOGY.**—The pathology of *softening* of the brain is much disputed, but I propose to treat of this morbid condition in connection with *embolism* and *thrombosis*, because it seems to me certain that cerebral softening is in the great majority of cases due to vascular obstruction thus induced. A cerebral *embolus* of any size generally has its origin in valvular disease of the heart, but it may come from an aneurism, or from a pulmonary thrombus. Minute emboli may also be detached from old clots, or from the inner surface of diseased vessels. The formation of *thrombi* is almost always associated with degeneration or disease of the cerebral vessels; which, however, may be aided by a feeble state of the circulation, and by certain conditions of the blood.

The main pathological causes to which the different forms of *cerebral softening* have been attributed may be thus summarized:—1. Local inflammation of the brain-substance. 2. Obstruction of arteries or capillaries by emboli; of arteries, veins, or venous sinuses by thrombi. 3. Pressure upon one of the main arteries by a tumour. 4. A diseased condition of the walls of the small arteries and capillaries, narrowing their calibre, and interfering with the nutritive relation between the blood and tissues. Such a condition is important in connection with syphilis. 5. Diminished nutritive activity in the tissue-elements, leading to their degeneration. 6. Effusion of blood into the brain. 7. A peculiar chemico-pathological change in the brain-substance, attended with the liberation of phosphine and one or more of the fatty acids. This form of softening is said to be often observed around adventitious products and old clots, but occasionally is independent of these morbid conditions (Rokitansky). 8. Œdema of portions of the brain. 9. Atrophic softening, due to separation of nerve-fibres from their ganglionic communications. I have already expressed my own view as to embolism and thrombosis being by far the most frequent causes of cerebral softening, the nutrition of the part thus deprived of blood being impaired, sometimes to such a degree as to cause its actual death, œdema and capillary hæmorrhage also in some cases contributing to the process of softening. It must not be forgotten that the brain may become softened as the result of a *post-mortem* change. With regard to the *predisposing causes* of cerebral softening, this lesion is



most common in advanced age, on account of the condition of the vessels, but when due to embolism it may be met with in young adults or even in children. Excessive and long-continued mental strain undoubtedly aids in its production, and it is by no means improbable that this may so disturb the balance of nutrition as of itself to give rise to softening of the brain.

**ANATOMICAL CHARACTERS.**—An embolus may lodge in one of the arteries before reaching the circle of Willis, and then, owing to the collateral circulation being readily set up, no permanent evil consequences ensue. Almost always, however, it passes into a branch beyond this circle, most frequently the *middle cerebral artery*, especially the left. The immediate result is anæmia of the portion of the hemisphere which this artery supplies, and as the result of the anatomical arrangement of the vessels previously described, this is followed by softening, with œdema, while there is a determination of blood into the surrounding capillaries, which often give way, giving rise to capillary hæmorrhage, especially at the circumference of the affected part. The effects may partly disappear in course of time in young persons whose vessels are healthy and distensible, and it is not improbable that the consequences of embolism in the minute vessels are often recovered from. After an embolus has been lodged for some time it may be difficult or even impossible to discover it at the *post-mortem* examination.

In *arterial* thrombosis the vessels will generally be found extensively diseased, many of them being usually blocked up. This condition will also lead to anæmia and softening, but owing to the morbid state of the minute vessels, and to the fact that several of them are obstructed, there is no increased vascularity around the affected part as a rule, this portion of the brain being very pale, while, if a vessel of any size is obstructed, for the same reasons restoration cannot be expected. Thrombosis of the *veins* and *venous sinuses* is generally associated with inflammation, resulting from injury or from bone-disease. It may arise, however, from feebleness of circulation accompanied with blood-changes, the thrombus first forming in the superior longitudinal sinus, and then extending, leading to more or less effusion of serum into the ventricles and sub-arachnoid space, or, rarely, even to hæmorrhage here or into the brain-substance, but especially to scattered patches of red softening with capillary hæmorrhages in the grey matter of the convolutions, or occasionally to more extensive softening.

Three chief forms of *cerebral softening* are described, namely, *red*, *yellow*, and *white*, but the softened part may present numerous grades of these tints, or even other colours, such as brown or greenish. The degree of diminution in consistence varies from what is scarcely appreciable to a condition in which the brain-substance is converted into almost a fluid pulp. This is determined by the aid of the finger; or by allowing a piece cut out of the softened part to stand, and observing how soon the angles round off, or the effects of pouring a stream of water gently upon it. The *seat* and *extent* of softening also vary greatly, and very different statements have been made as to the frequency with which different parts of the brain are involved, but the usual seats seem to be the corpus striatum, optic thalamus, central white matter of the hemisphere, and the convolutions. In *embolic* softening the left hemisphere is most commonly affected. The morbid condition is rarely well-defined, but shades off into the surrounding brain-tissue. A section appears to be swollen, and often rises above the surrounding level. A highly impor-



tant character of all forms of cerebral softening, except, it is said, the inflammatory variety, is that the specific gravity of the softened part is considerably below that of normal brain-substance. Microscopical examination reveals more or less destruction of the nerve-elements, until in extreme cases no trace of them is visible; abundant granular cells, not of inflammatory origin, but chiefly derived from granular degeneration of the cells of the neuroglia or of nerve-cells; granular fatty débris; particles of myeline; blood-cells in some varieties of softening, or their remains in the form of pigment and hæmatoidine crystals. The small vessels often show signs of degeneration, and are in some forms of the disease dilated, or present little aneurismal swellings.

It is necessary to offer a few remarks respecting the three main forms of softening. *Red* softening may be inflammatory in its origin, but in the great majority of cases it depends upon obstruction of large arteries or venous sinuses. The tint at first may vary from pink to deep claret, being most intense in the grey matter. Afterwards it undergoes changes, assuming different hues of yellow, brown, chocolate, buff, and other colours. *Yellow* softening usually results from changes in the red variety, but the peculiar form due to the chemico-pathological change is described as being of this colour. *White* softening is considered by some pathologists as the ultimate condition of the red variety, but undoubtedly it is not uncommonly a primary form, especially in connection with extensive thrombosis and atheroma of the vessels. It is also observed in œdematous and atrophic softening.

Reparative processes may be set up after softening. On the surface of the brain circumscribed yellow patches—*plaques jaunes*—are produced, of tough consistence, implicating the grey matter of the convolutions. In the interior of the organ a cavity forms, bounded and traversed by a white or greyish or sometimes a yellowish vascular connective tissue, which may divide the space into loculi, and these contain a milky fluid, holding in suspension abundant fat-granules and corpora amylacea. This fluid may be absorbed, contraction and ultimate closure of the cavity subsequently taking place.

**SYMPTOMS.**—It will be requisite in treating of the clinical history of cerebral softening to describe it under the two forms of *acute* and *chronic*. The latter is considered in the chapter on Chronic Diseases of the Brain.

**Acute Softening.**—Practically it will only be necessary here to indicate the respective symptoms of *embolism of the middle cerebral artery* and *thrombosis*. *Embolism* may occur at any age, being not uncommon in young persons; some morbid condition likely to originate an embolus can generally be discovered; whilst in most cases no premonitory head-symptoms have been noticed. The usual indications of the lodgment of the embolus in the brain are sudden loss of consciousness and symptoms of shock; with hemiplegia, generally on the *right* side. The case may end fatally, or consciousness may return, but the hemiplegia remains, aphasia being also of very frequent occurrence. The paralysis is rarely recovered from entirely, but power may be partially restored, especially in young persons. In a case mentioned in a former chapter which came under my notice the leg recovered perfectly, but the arm, face, and tongue remained permanently paralyzed, and aphasia was also persistent.

*Arterial thrombosis* is met with either in old persons, or in those who are prematurely aged; and usually, but not always, well-marked signs

of degeneration of the vessels of the limbs are obvious, along with a feeble heart and other evidences of decay. Commonly there have been marked *premonitory* symptoms, due to interference with the cerebral circulation, or to the formation of minute thrombi, similar to those described under CEREBRAL HÆMORRHAGE. The mode of attack varies, but is usually more or less *apoplecticiform*. Should a large artery or several smaller vessels be rapidly obstructed, a sudden apoplectic seizure with hemiplegia occurs, frequently not distinguishable from sanguineous apoplexy. By far more commonly, however, the onset is gradual, there being marked mental disturbance of some kind before the coma supervenes, such as transient excitement, confusion of thought, irritability, or mild delirium, the patient often talking incoherently or acting strangely. There may be complete loss of consciousness for a brief period, but this condition soon passes away, and almost always when the patient is seen the coma is only partial, indications being afforded that sensation and perception are not entirely abolished. In some cases apoplecticiform attacks are repeated, with partial restoration in the intervals, absolute coma being ultimately developed, with general paralysis and involuntary passage of feces and urine, death taking place in a few days. In others the mental faculties become by degrees considerably improved, even up to the previous standard: but as a rule the intellect remains markedly impaired, and this condition tends to become speedily worse and worse. There is frequently permanent aphasia or defect of speech. When sufficiently restored, the patient often gives indications of pain or uneasiness in the head; and of hyperæsthesia, dysæsthesia, or various paræsthesiæ in either or both limbs on one side. Hemiplegia is usually present, and during the apoplectic condition the head and eyes are often turned to the sound side. Sometimes the paralysis is bilateral, or is confined to one limb. Generally it is not complete, being also more marked in the arms than in the legs, and at the extremities of the limbs than in other parts. Early spasmodic contractions in the paralyzed parts are very common, in the way of clonic spasms, jerkings, or rigid flexion of joints, especially of the shoulder, elbow, and knee. The affected limbs are often exceedingly irritable on percussion. The paralysis is not likely to diminish. *Hemiplegia without loss of consciousness* is far more common in thrombosis than in cerebral hæmorrhage, and the paralysis may come on in a *progressive* manner, which is very characteristic, affecting first one limb and then the other, at the same time becoming more marked by degrees. Occasionally the early symptoms assume the characters of irregular epileptiform attacks occurring in rapid succession, attended with mental disturbance, ending in coma and hemiplegia. In other instances delirium is the prominent symptom at first, usually mild, sometimes violent, alternating with and finally ending in coma. The subsequent course of cases of acute cerebral softening varies much; many of them become very chronic, the patients gradually sinking into a state of imbecility, and being permanently bed-ridden and helpless; the opposite side often becoming weak; "late rigidity" setting in in the paralyzed muscles; and nutrition being much impaired.

## V. GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. DIAGNOSIS.—With regard to the *hyperæmic* or *anæmic* disorders of the circulation of the brain, which give rise to the slighter symptoms, nothing need specially be said as to their diagnosis. Let it be borne in mind, however, that persistent symptoms pointing to the head, especially in persons at all advanced in years, should always lead to a careful investigation of the case. It not uncommonly happens that head-symptoms are complained of more or less constantly, such as headache or giddiness, and it becomes a question whether these depend upon some *extrinsic* disturbance, especially connected with the digestive organs, heart, or kidneys; or upon congestion of the brain, or disease of the cerebral vessels. It is always well to give a cautious opinion under these circumstances. The general condition of the patient; the presence or absence of marked symptoms referable to the alimentary canal; the state of the heart, vessels, and kidneys, as determined by physical and other modes of examination; and the precise nature of the symptoms complained of, will usually render the diagnosis evident. Any sensory or motor disorder in the limbs, especially if always noticed on one side or fixed in the same spot, should be looked upon with suspicion. Again, slight nervous phenomena, which are often attributed to mere congestion of the brain, may be due to thrombosis of small vessels or to minute hæmorrhages. Care must also be taken not to mistake between cerebral congestion and anæmia. In further considering the diagnosis of these affections it will be convenient to discuss them under the two main groups, in which they are usually presented in practice.

*a. The apoplectic condition.*—This must be considered, not only with reference to cerebral diseases, but also to other causes by which it may be induced. The comatose state may either be developed more or less suddenly, while the patient is under observation; or he may be found in an unconscious state. The first thing to be determined in a case of insensibility, of which the cause is unknown, is whether the condition is one of *syncope* or *shock*; *asphyxia*; or *coma*. The characteristic features of each of these conditions have been already described, and need not be recapitulated here. The ordinary causes acting directly on the nervous system which are to be borne in mind as likely to account for unconsciousness, the origin of which is not evident, are:—*a.* Injury to the head. *b.* Epileptic or other form of convulsive seizure. *c.* Uræmia. *d.* Poisoning by alcohol or opium. *e.* Sun-stroke. *f.* Certain affections of the brain or its membranes, namely, cerebral congestion; hæmorrhage into the brain or meninges; cerebral embolism or thrombosis; and rapid serous effusion.

In endeavouring to arrive at a diagnosis, the following course of investigation should be pursued:—

(i.) Inquiry must first be made as to the *mode of attack*, and if there is any known *cause* for this. Thus there may be a history of injury, opium-poisoning, or alcoholism. On the other hand, the circumstances under which the seizure occurs may exclude such causes altogether, but it is important to bear in mind that symptoms of opium-poisoning may not appear until some time after the poison has been taken. Not uncommonly, however, the patient is found in a state of insensibility, and no



history can be obtained. Should this happen in a house, it is requisite in any suspicious case to look for bottles which might have contained poison. If the attack has come on under observation, it is very important to ascertain whether it arose spontaneously, or followed some obvious cause, such as sudden effort or a fit of anger; whether it was sudden in its onset, or more or less gradual; if preceded or not by mental disturbance, local sensory or motor disorder, or other symptoms; and if any convulsive movements were observed at or soon after the beginning of the attack. This information affords much aid in distinguishing organic lesions from each other; while the occurrence of convulsions entirely excludes poisoning. The *age* of the patient should be ascertained, if it is known.

(ii.) In the next place a *careful examination of the patient* must be carried out, noting especially the following particulars:—*a.* The apparent age; general conformation and appearance, whether full and plethoric or the reverse; and if there are marked signs of decay. *b.* The colour of the face, whether indicating congestion or shock. *c.* If any signs of injury about the head can be discovered. *d.* Whether there are any indications that the attack began with convulsions, such as the tongue having been bitten. *e.* The odour of the breath, which may reveal alcohol, opium, or uræmia. *f.* The degree of insensibility, deep coma usually indicating hæmorrhage or poisoning. *g.* The state of the pupils, any inequality showing some cerebral organic lesion; while extreme contraction is a sign of opium-poisoning, though the same condition is now and then observed in cerebral hæmorrhage, and the pupils are greatly dilated at the close in cases of opium-poisoning. *h.* If there are any indications of unilateral motor disorder, looking especially for paralysis; turning of the head and eyes to one side; tremors; or spasmodic movements or rigidity. These afford evidence of some cerebral lesion, though their absence does not exclude this, while marked spasm or rigidity is in favour of plugging of vessels or meningeal hæmorrhage. *i.* The characters of the breathing, slow, laboured, and stertorous respiration being usually only observed in the profound coma of cerebral hæmorrhage or narcotic poisoning. *j.* The state of the pulse. *k.* It is highly important further to examine the heart and vessels. For instance, valvular disease or some other condition likely to give rise to embolism may be thus discovered; cardiac enlargement is often associated with cerebral congestion or hæmorrhage; in cases of thrombosis the heart is usually very weak or fatty; while marked degeneration of the vessels may accompany either hæmorrhage or thrombosis. The urine should also be tested, some of which may be withdrawn by the catheter if necessary. Bright's disease, however, may be associated with uræmia, cerebral hæmorrhage, or thrombosis. The detection of alcohol in the urine has been considered useful in the diagnosis of alcoholic poisoning. If the patient vomits, the matters vomited ought to be examined in any doubtful case, and it may even be desirable to use the stomach-pump.

(iii.) The *progress and termination* of an apoplectic case often give much information as to its nature. Thus, hæmorrhage on a very extensive scale or into certain parts of the brain soon terminates fatally; and so usually does opium-poisoning. In a considerable experience of cases of alcoholic poisoning at the Liverpool Northern Hospital I never knew one prove immediately fatal, even when the coma was very deep. The course of events also affords important help in distinguishing between cerebral congestion, hæmorrhage, and plugging of vessels.

There are some points of practical importance which demand notice. The greatest care should be taken not to pronounce a person merely drunk in whom there are signs that this condition exists, as there may be at the same time some serious injury to the head, or an organic lesion affecting the brain. Grave mistakes have not unfrequently been made in this matter. Alcoholic poisoning may be met with in very young children, even in infants in arms. It is sometimes difficult or impossible to determine whether comatose symptoms are due to some evident injury to the head; or to a sudden cerebral lesion which has caused the patient to fall, and has thus led to the injury.

b. The second group of cases which call for consideration here are characterized by *sudden or rapidly-developed hemiplegia without loss of consciousness*. This indicates either hæmorrhage into the brain; or plugging of vessels, especially from thrombosis. The probability is always in favour of the latter, and the diagnosis is still more certain if the paralysis is not suddenly complete, but increases and extends in a progressive manner.

2. PROGNOSIS.—The immediate prognosis of an *apoplectic seizure* due to cerebral lesion is always doubtful, and a very cautious opinion ought to be given, the case being thoroughly watched. If the coma is merely due to congestion, the patient will soon recover. The chief circumstances which increase the gravity of the immediate prognosis in cases of sanguineous apoplexy are:—advanced age of the patient, with very degenerate vessels; a history of previous attacks; the occurrence of convulsions at the outset, or of marked rigidity or spasmodic movements at an early period; a progressive character of the attack; very deep and prolonged coma, with involuntary passage of urine and fæces; general paralysis; great dilatation and immobility of the pupils, or extreme contraction; a very slow or rapid pulse; signs of profound shock, with feeble circulation, pallor, and cold sweats. As to the ultimate prognosis, supposing consciousness to be restored, this can only be determined by watching the case for some time and noting its progress; and the same is true when hemiplegia sets in without coma. Right hemiplegia is said to be less favourable than left. Anæsthesia affecting any part of the paralyzed limbs is a bad sign, as well as the occurrence of occasional severe pains. If no improvement is evident within a month; if the paralyzed limbs exhibit a marked tendency to permanent rigidity; and if electric irritability becomes impaired or lost, the prognosis is very unfavourable. The leg may recover power while the arm remains permanently paralyzed. After cerebral hæmorrhage the mental faculties are often perfectly restored, even though the paralysis is persistent. It must be borne in mind that a clot in the brain may cause serious inflammation of its substance, and may thus prove fatal some time after the occurrence of the hæmorrhage.

Embolism or thrombosis is not so frequently immediately fatal as cerebral hæmorrhage, but the subsequent history is generally very unfavourable, both as regards the mental condition and the paralysis, especially in cases of thrombosis accompanied with extensive disease of the vessels, such cases often going on rapidly from bad to worse.

3. TREATMENT.—In the treatment of the minor symptoms due to congestion or anæmia of the brain, attention must be paid to the state of the blood and of the circulation. General cerebral anæmia may be obviated by improving the quality of the blood; and stimulating or giving tone to the heart. No special treatment can be directed to

partial anæmia. Cerebral congestion may be relieved by keeping the patient on low diet; administering saline and other *purgatives*; avoiding any cause which is likely to give rise to the condition, such as over-study; or, in appropriate cases, removing blood either locally or generally. The patient must be warned against wearing tight clothing about the neck, hanging down the head, straining at stool, and other causes which are likely to increase cerebral congestion, particularly if there are any indications that the vessels are in a diseased state.

Before describing the treatment of an *apoplectic attack* resulting from cerebral mischief, I would remark that in cases where the diagnosis is uncertain, it is desirable to empty the stomach at once by means of the stomach-pump, lest the symptoms should be due to some poison. In this class of cases the first principle in treatment ought to be not to interfere immediately and actively unless there is some clear indication for such interference. Formerly venesection was at once resorted to, and is now but too often followed as a routine practice. In many cases all that is necessary is to place the patient in the recumbent posture, if possible in bed, with the head high; to loosen all clothing about the neck and chest; to allow plenty of fresh air; and to enjoin perfect quiet. If the attack is merely due to congestion, recovery will soon follow. Should the case be one of hæmorrhage, with obvious signs of marked plethora, unquestionably venesection may be useful, but it is rarely needed; on the other hand, the condition is not uncommonly one of shock, and then stimulant enemata, heat and sinapisms to the extremities, and other measures for rousing the patient are indicated, particularly when the coma is due to plugging of vessels. The practice of placing a drop or two of croton-oil on the tongue is useful in many cases. If the comatose state continues for a considerable time the patient must be supported by enemata; sinapisms may be applied to various parts; and the bladder must be attended to. If consciousness returns, the patient should be kept completely at rest, free from all mental disturbance, and upon low diet, until the period of danger from inflammation has passed. Should this morbid condition be set up, the hair may be cut short and cold applied, or small blisters to the nape of the neck. The subsequent treatment of these cases, as well as those of *sudden hemiplegia* without coma, must depend upon their progress. The main indications are to support the general health, especially by proper diet, attention to hygienic conditions, and the administration of *tonics*; to avoid all forms of mental disturbance; and to treat symptoms, particularly paralysis, which must be managed according to the principles and methods already pointed out. Iodide of potassium and bichloride of mercury have been supposed to aid in the absorption of a clot in the brain. A blister occasionally applied to the nape of the neck may prove serviceable.

---



## CHAPTER XC.

## CHRONIC DISEASES OF THE BRAIN AND ITS MEMBRANES.

## I. CHRONIC MENINGITIS.

**ÆTIOLOGY.**—Chronic meningitis is chiefly met with as the result of former injury to the skull; prolonged mental labour, especially if combined with much anxiety; chronic alcoholism; and irritation by tumours and morbid growths, especially syphilitic. In rare instances it remains after the acute form of the disease. One form of chronic meningitis, named *pachymeningitis*, most frequently comes on spontaneously in aged persons who are the subjects of imbecility or dementia. It is said to have been also observed in cases of chronic alcoholism and chronic phthisis. It is by far most common after middle age, and in males.

**ANATOMICAL CHARACTERS.**—The most frequent *post-mortem* signs of chronic meningitis are thickening and increased firmness of the membranes in different parts, sometimes extreme, with opacity of the arachnoid; adhesion of the membranes to each other, of the dura mater to the skull, or of the pia mater to the brain; increased vascularity, particularly of the pia mater, with permanent enlargement of many vessels; serous effusion into the meshes of the pia mater, there being also sometimes a considerable quantity of turbid fluid in the sub-arachnoid space; exudation, especially in the sulci and around the vessels, often surrounding and pressing upon some of the cranial nerves, and becoming organized and firm; and the presence of excess of clear or flocculent serum in the ventricles, the lining membrane of which becomes thickened and rough. Calcareous or osseous laminae may be formed in the thickened membranes, and the convolutions of the brain are sometimes atrophied. Increase in number and enlargement of the Pacchionian bodies has been considered a result of chronic meningitis.

The form of lesion named *pachymeningitis* usually begins in the region corresponding to the distribution of the middle meningeal artery, and occupies a variable extent. A delicate adherent film forms, which mainly consists of large and thin-walled capillaries, partly of embryonic corpuscles. Gradually new films are developed upon this in succession, until they may attain a considerable thickness. The deeper layers at the same time become firmer, less vascular, and more fibrous. Extravasations of blood often occur, owing to rupture of the delicate vessels, usually small and numerous, but not uncommonly considerable, so that the whole structure may look like a clot. Blood-pigment is frequently precipitated in crystalline and other forms.

**SYMPTOMS.**—These are often very obscure and ill-defined in chronic meningitis, being a combination of symptoms due to excited action and impaired function of the parts involved. The most important clinical indications of this disease are:—1. More or less constant general headache, of dull and heavy character, not severe or attended with exacer-

bations. 2. Persistent vertigo, the patient staggering while walking, as if drunk, but particularly on looking suddenly round over the shoulder. 3. Mental excitability at times, especially in the evenings, with peevishness, irritability, restlessness, and wakefulness; alternating with marked depression, the patient becoming apathetic, gloomy, low-spirited, and apprehensive. 4. In course of time failure of the intellectual faculties, sometimes ending in extreme dementia. 5. Subjective sensations of flashes of light, iridic colours, or tinnitus aurium; with at the same time diplopia or more or less impairment of sight in one eye, or partial deafness. 6. Hyperæsthesia of some parts of the skin, with hypæsthesia or numbness of other portions. 7. Irregular twitchings or clonic spasms of various muscles, especially those of the face and eyeballs, causing grimaces, and often external strabismus; also of the muscles of the limbs, which may present curious spasmodic movements from time to time, or be the seat of rigidity. 8. Irregular motor paralysis, usually incomplete, accompanying and following the above movements, affecting first the cranial nerves on one or both sides, as indicated by partial ptosis, drawing of the face to one side, strabismus or immobility of the eyeball, slight deviation of the tongue and thickness of speech; then extending to the limbs, in some cases only a few fingers or certain muscles being involved, in others the whole arm, one arm and leg, or sometimes all the limbs more or less. Irregular epileptiform attacks not uncommonly occur, but they are not attended with any special cry, or with stoppage of respiration and its consequences, while consciousness is not completely lost. The fit lasts an indefinite and often a considerable time, and is not followed by the comatose state observed in true epilepsy. In most cases some degree of pyrexia is noticed towards evening, with heat of head, flushing of the face, and conjunctival injection. Nausea and vomiting, with obstinate constipation, are not infrequent symptoms. Any excitement tends to increase the symptoms markedly. The ophthalmoscope often reveals optic neuritis or ischaemia.

In *pachymeningitis* the symptoms are very indefinite, being described as headache, vertigo, failure of the mental powers, gradually increasing hemiplegia, and occasional epileptiform or apoplectic attacks. Death usually occurs during one of these attacks.

## II. CHRONIC CEREBRAL SOFTENING.

SYMPTOMS.—The pathology of this disease has been already considered (see p. 910), and it is only requisite to describe here the clinical history of softening which is chronic from the commencement. The chief symptoms of this condition are:—1. Headache, in many cases persistent but not severe; usually of heavy character, and sometimes only amounting to a feeling of weight and heaviness; frontal as a rule, occasionally general, but never unilateral or localized. 2. Mental changes, namely, gradual failure of the intellectual faculties one after another, of which the patient is generally aware at first, which may ultimately end in complete dementia or mania; change in manner, disposition, and temper; various forms and degrees of aphasia, a tendency to repeat the same words several times and on all occasions being considered very characteristic; marked lowness of spirits; emotional disorder, the patient being either apathetic, or the emotions being but little under control, and quasi-hysterical fits

of crying or laughing occurring without cause; sometimes a restless and excited manner at night, or even mild delirium. Occasionally the mind seems unaffected. 3. Sensory disorders, especially superficial and deep pains in various parts of the limbs, hyperæsthesia or dysæsthesia, formication, numbness, and gradual impairment of sensation, seldom amounting to complete anæsthesia; some degree of failure of sight and hearing, but rarely complete blindness or deafness. 4. Motor disturbance, in the direction of paralysis, incomplete in degree, and developed gradually and often in an intermittent manner, usually beginning in either arm or leg, but soon being more or less generally but irregularly distributed, though one side is as a rule more affected than the other, or sometimes confined to special groups of muscles, such as those of the face or part of a limb; frequently tonic rigidity, gradually increasing; tremors or clonic spasms, especially in the paralyzed muscles, with undue irritability on percussion; and sometimes epileptiform convulsions. The subjects of chronic softening commonly present an old or prematurely aged, unhealthy, and cachectic appearance. Degeneration of the vessels, weak heart, and granular kidneys are frequently well-marked. Usually the bowels are obstinately constipated. The duration of the disease is very variable. At last gradual coma sets in, usually with general paralysis and relaxation of the muscles, and involuntary passage of urine and fæces. Speedy and unexpected death may happen from extensive thrombosis or from cerebral hæmorrhage.

### III. ADVENTITIOUS GROWTHS IN CONNECTION WITH THE BRAIN AND ITS MEMBRANES—CEREBRAL TUMOUR.

**ANATOMICAL CHARACTERS.**—The chief adventitious growths or tumours which are met with in connection with the brain or its membranes include:—1. *Cancer*. 2. *Tubercle*. 3. *Syphilitic deposit*. 4. *Sarcoma*. 5. *Myxoma*. 6. *Glioma*. 7. *Cholesteatoma*. 8. *Lipoma*. 9. *Parasitic cysts*, namely, cysticercus cellulosus and hydatids. 10. *Cysts* containing fluid, fat, or hair; or presenting cauliflower excrescences. 11. *Aneurisms*. 12. *Vascular erectile tumours*. 13. *Osseous or calcareous masses*.

**Cancer.**—All forms of malignant disease are met with in the brain, but far most commonly the encephaloid variety, which usually occurs as a more or less round or lobulated tumour, of very variable size, generally single if primary, sometimes numerous if secondary. The growth may be inseparable from the brain-substance; distinctly defined; or surrounded by a cyst. The usual seat of cancer in the brain is the cerebral hemisphere, but it may occupy any part. It often shows signs of degenerative changes in its interior. It must be mentioned that cancer may originate from other structures within the cranium besides the brain itself, and then tends in some cases to make its way outwards; on the other hand, it may begin outside the skull and grow inwards.

**Tubercle** in the brain is described as forming irregularly-roundish masses, yellow and caseous-looking, dry and bloodless, sometimes continuous with the brain-substance through a greyish-white, somewhat translucent border, in other cases separated by a cyst. As a rule there is but one such mass, occasionally two, rarely more. The size generally varies from that of a hemp-seed to a cherry, but it may be equal to a small egg. Tubercle ordinarily occupies the cerebrum or cerebellum,



rarely the pons. It often softens in the centre into a purulent-looking fluid, or an actual cavity may form.

**Syphilitic disease.**—Syphilitic deposit is far more commonly met with in connection with the membranes than in the brain itself. These are matted together, the dura mater being closely adherent to the skull at the seat of disease, and the inner membranes to the cerebral substance, while there is more or less thickening from the accumulation of a tough material, yellow in the centre, but usually presenting a greyish-white border around. This often involves some of the nerves. In the brain syphilis generally causes mere induration from interstitial proliferation of cellular tissue, especially at the surface. Gummata are extremely rare; they assume the form of irregular nodules or tumours, which may attain the size of an egg, their shape being often modified by the part of the brain in which they occur, and they always pass gradually into the surrounding tissue, either through a greyish softer material or an indurated infiltration. A section is yellowish-grey or yellowish and translucent; cheesy or glaucous in consistence; and it often presents signs of decay, in the form of opaque spots scattered over the entire surface, but there is no central softening, such as is observed in tubercle. The more vascular parts of the brain are the usual seats of syphilitic gummata. Generally only a single deposit is found.

**Sarcoma.**—This form of tumour is not uncommon, and may be connected with the membranes, or imbedded in the brain-substance, especially the cerebrum. It is round or lobulated, varying in size from a nut to a good-sized apple, being distinctly defined and frequently enclosed in a vascular capsule, from which it can be turned out. A section is smooth, and dirty-white or greyish-red; and the consistence is generally soft, but may be firm and fibrous. In structure a sarcoma consists mainly of spindle-shaped cells. Calcification may take place in spots; or spaces may be formed, containing fluid.

**Myxoma.**—Usually occurring as a distinct tumour of variable size, occasionally as an infiltration, myxoma is generally observed in the cerebrum. It is very soft, and may be almost gelatinous; somewhat translucent; and has usually a yellowish or reddish colour, but may present extravasations of blood. In structure the material consists of variously-formed cells, imbedded in a homogeneous hyaline substance.

**Glioma.**—Resulting from a local hyperplasia of the neuroglia, this form of growth is never well-defined, but runs into the surrounding brain-substance, and does not pass from the brain to the meninges. It is usually found in a cerebral hemisphere, and may attain a considerable size. The colour of a section varies from yellow to greyish-red, and cut vessels are generally seen. The consistence is usually rather soft. Glioma consists of a finely-reticulated material enclosing roundish nuclei. It may become the seat of degeneration or hæmorrhage. It occurs chiefly in young persons.

**Cholesteatoma** is a very rare growth, consisting of concentric laminae of epithelial-cells. It forms a tumour, surrounded by a delicate membrane, and presenting a pearly lustre on section. It may develop from the brain, the meninges, or the skull.

**Cysticerci** are generally found in the grey substance, being usually numerous when present. **Echinococci** are very rare.

**Aneurism** is also a rare condition in connection with the brain. It generally affects one of the arteries at the base, being in most cases of small size, but may attain the dimensions of an egg.

The other forms of cerebral tumour do not call for any special description.

**SYMPTOMS.**—There is no disease of the brain in which the symptoms are more diverse in their characters and course than in the different kinds of tumour, so much depending on their situation, size, shape, number, and rapidity of growth. Further, the symptoms are not only due to the mechanical effects of the tumour, but those of cerebral softening, hydrocephalus, and chronic meningitis are often superadded after a time.

Occasionally a tumour, even of considerable size, is quite latent throughout; or a sudden apoplectic attack, the result of congestion or hæmorrhage, reveals its existence. The characteristic clinical features of a cerebral tumour, however, are as follows:—1. Headache, at first slight, but by degrees becoming very severe; often localized, though not necessarily over the seat of the tumour; constant, and of dull grinding character, but subject to violent exacerbations, which may be accompanied with obstinate cerebral vomiting, the pain being often increased by any excitement, coughing, sneezing, a deep breath, or strong light. 2. Marked vertigo, or a peculiar dizziness on movement in some cases. 3. Absence of any mental disturbance in uncomplicated cases, unless a tumour is very large or rapid in its growth, or unless there should be several growths scattered through the cortical substance. 4. Signs of irritation followed by gradual paralysis of such of the cranial nerves as the growth implicates, these being generally unilateral. Vision is often affected, terminating in complete blindness, and it is not uncommon for one eye to be involved after the other. Smell and hearing may also be impaired or lost, the latter usually on one side. Severe neuralgic pains, hyperæsthesia, and paræsthesiæ frequently affect the fifth nerve, followed by gradual loss of sensation to complete anæsthesia, and paralysis of its motor portion occasionally. The facial nerve is most commonly implicated, next the third and sixth, and sometimes the fourth, there being first twitchings and spasmodic movements followed by paralysis of the muscles supplied by these nerves; when the paralysis is complete, electric irritability becomes usually quite extinct. Partial paralysis of the eighth and ninth nerves is not uncommon, causing impairment of speech and deglutition, or sometimes disturbance of respiration or cardiac action. 5. Disorders of sensation and motion in the limbs. These are, if present, as a rule of more or less unilateral distribution, and are observed on the side opposite to that on which the cranial nerves are implicated. Rarely these disorders are bilateral, or they may be very limited. At the outset signs of irritation are observed, followed by gradually progressive paralysis, which is accompanied with spasmodic movements or rigidity. Electric irritability is not lost in the limbs. When a tumour occupies the interior of one cerebral hemisphere there may be pure hemiplegia. Epileptiform convulsions are not uncommon, the convulsive movements being frequently localized mainly in some particular part, as has already been described under epilepsy. When a tumour occupies certain portions of the brain, peculiar rotatory and other movements are observed. A special feature in tumours of the cerebellum, more especially of the middle lobe, is said to be a tonic rigidity of the muscles of the back of the neck, with retraction of the head, associated frequently with flexion of the fore-arms and extension of the legs, with pointing of the toes. They also press on the venæ Galeni, and cause dropsy of the ventricles,

with corresponding mental symptoms. The *important ophthalmoscopic signs* of cerebral tumour are those of ischæmia; optic neuritis; or atrophy of the disc. Occasionally a tumour becomes evident externally. In cases of aneurism, it is said that a murmur has been occasionally heard over some part of the skull. The general condition of the patient varies much. The constitution is gravely affected, should there be much suffering, with loss of sleep. Sometimes considerable emaciation and marasmus, or evidences of some cachexia are observed; or cancerous, syphilitic, or tubercular deposits may be discovered in other parts. The mode in which cases of cerebral tumour terminate is very variable. In those which have come under my observation the end was always unexpected, acute symptoms setting in without any obvious cause.

#### IV. CHRONIC HYDROCEPHALUS.

**ÆTIOLOGY AND PATHOLOGY.**—Chronic hydrocephalus is characterized by accumulation of fluid, either in the ventricles; in and beneath the arachnoid; or in both these situations. In the great majority of cases the condition is either congenital, or is revealed within six months after birth, and has then been chiefly attributed to arrest of development of the brain, or to chronic inflammation of the ventricular lining membrane. It may come on as an acquired complaint, however, in older children, or very rarely even in adults, being then the result of closure of the *venæ Galeni*, usually the consequence of pressure by a tumour. Chronic hydrocephalus has no connection with tubercle, but is not uncommon in cases of rickets. Excess of fluid may accumulate in the arachnoid sac in connection with senile or other forms of atrophy of the brain; or after previous hæmorrhage.

**ANATOMICAL CHARACTERS.**—The quantity of fluid in cases of chronic hydrocephalus varies from a few ounces to several pints. It is usually watery, limpid, and colourless; of low specific gravity; and contains but a very small quantity of albumen, with some saline matter. The ventricular lining membrane is often altered in appearance, thickened, granular, and rough. The arachnoid is stretched, and signs of chronic meningitis are often observed about the base. The brain is altered in shape, sometimes unsymmetrical, its convolutions being flattened and spread out; and its texture is in many cases firmer than natural, or, on the other hand, soft and macerated. The optic nerves are usually much stretched. The cranial bones are frequently expanded and thinned, the fontanelles and sutures being considerably widened; sometimes the bones are thickened, but spongy.

**SYMPTOMS.**—Only the signs of chronic hydrocephalus in children need be considered here. The head enlarges, in some cases attaining an enormous size, so that it falls from side to side if not supported, and at the same time assumes a curious shape, becoming round and globe-like, with a very large and prominent forehead, the bones being driven apart, while the orbital plates of the frontal are pushed down, especially behind, the eyeballs being thus pressed forward so as to become very prominent and to look downwards. The fontanelles and sutures are widened to a variable degree, as well as prominent; while frequently distinct fluctuation can be detected. The scalp feels thin, sometimes almost as if it were going to give way, and the bones may be so attenuated as to yield a crackling sensation. The lower part of the



face looks very small, and has a curious worn or stupid expression. It may present a puffy appearance, with enlarged vessels on the cheeks. The nervous symptoms which are liable to be met with are headache, though this is often absent; vertigo; non-development or gradual failure of the mental faculties, even to complete imbecility; disturbed sleep at night, with drowsiness by day; marked peevishness, irritability, or depression of spirits; failure of the special senses, especially that of sight, with signs of ischæmia or atrophy of the disc; restlessness, with general muscular weakness and loss of co-ordinating power, tottering gait, tremors of the limbs, spasmodic movements or convulsions coming on without any obvious cause, strabismus, or laryngismus stridulus. The body is generally much wasted; circulation is feeble; and the child always feels cold. Excessive appetite, vomiting, and constipation with unhealthy stools are common symptoms. The duration of cases of chronic hydrocephalus is variable, but usually death occurs within the first few years of life, chiefly from gradual or sudden coma, exhaustion, convulsions, or laryngismus stridulus.

#### V. HYPERTROPHY OF THE BRAIN.

Only a few remarks are required with reference to the so-called *hypertrophy* of the brain observed in children. This is associated either with rickets or with congenital syphilis; and the increase in size and weight of the organ is probably due either to albuminoid infiltration of the white substance, or to increase in the neuroglia. The tissues become unusually firm, pale, and dry; the convolutions being compressed, flattened, and closely packed. The head expands, but the enlargement is distinguished from that of chronic hydrocephalus by being much less rapid in its progress, never attaining any great size, and by having an elongated form from before back; while the fontanelles and sutures are not at all or but little apart, the former being often depressed, and not yielding fluctuation; and the eyes are sunken. Frequently there are no evident nervous symptoms, but if the head is closed before the brain enlarges, serious symptoms are liable to arise, such as severe headache, vertigo, mental failure, epileptiform attacks, paralysis, or coma.

#### VI. GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. **DIAGNOSIS.**—The principal chronic cerebral diseases between which a diagnosis has usually to be made are *chronic meningitis*; *chronic softening*; and *cerebral tumour*. It must be remembered that these are often associated, under which circumstances their symptoms are more or less combined. The chief points to be considered in the diagnosis are:—1. The *history* of the case, as revealing some local cause of meningitis or a syphilitic taint; or the absence of any such cause. 2. The *age* and *general condition* of the patient, with the state of the main organs and vessels, softening being generally accompanied with signs of marked degeneration, and occurring in old persons or in those prematurely aged. There may be signs of some constitutional taint associated with a cerebral tumour; or of morbid deposits in other parts, especially cancerous, tubercular, or syphilitic. 3. The *seat*, *intensity*,

and characters of *headache*. 4. The *mental condition*, meningitis being chiefly characterized by excitement alternating with depression; softening by gradual and permanent impairment of the mental faculties; while in cases of tumour the mind is often quite unaffected. 5. The character and mode of distribution of *sensory* and *motor* disturbances. These have been already pointed out in the description of the symptoms of each disease, and they are very important. 6. The appearances revealed by the *ophthalmoscope*. The most characteristic signs of cerebral tumour are the headache, cerebral vomiting, and optic neuritis. Occasionally external objective signs of such a tumour are observed. As to the *nature* of a growth in the brain, it is often impossible to come to any certain conclusion. Some indications may be derived from the age of the patient; a history of syphilis; signs of a particular cachexia; or the presence of morbid growths in other parts. The *situation* of a cerebral tumour must be determined by the symptoms present.

Epileptiform seizures may occur in the course of the diseases just considered. These can generally be distinguished from true epileptic fits by their irregular character; and by the existence of definite symptoms indicative of one or other of these morbid states.

The difference between *chronic hydrocephalus* and *hypertrophy of the brain* in children, each of which causes enlargement of the head, have been sufficiently pointed out in their several descriptions.

2. PROGNOSIS.—In chronic brain-affections the prognosis is very uncertain. All that can be definitely stated is that any such affection is always serious; and that at any moment dangerous symptoms are liable to arise, which may end in speedy death. If the disease is of a syphilitic nature, however, much improvement may often be effected by proper treatment. If there are indications of frequent or constant disorder of the cerebral circulation, along with degeneration of the vessels, the danger of the supervention of hæmorrhage or thrombosis should be recognized.

3. TREATMENT.—The principles of treatment applicable to chronic cerebral diseases are very simple, namely:—*a.* To keep the mind free from every possible excitement or anxiety, and to forbid any mental labour; in short, to keep the brain as much at rest as possible. *b.* To support the general health by good food, fresh air, quinine, iron, cod-liver oil, and hypophosphites. *c.* To aid absorption of morbid products. Iodide of potassium, bichloride of mercury, and grey powder are the chief drugs employed in cases of chronic meningitis, but they are especially important in syphilitic disease. Occasional blistering is supposed to promote absorption. *d.* To treat symptoms, especially headache; paralysis; restlessness and sleeplessness, by means of hyoscyamus, cannabis indica, or chloral; and convulsive seizures by bromide of potassium. Acute symptoms may arise calling for active interference. For chronic hydrocephalus *diuretics* are recommended, with the view of aiding absorption of the fluid. Pressure around the head by means of a bandage or strapping; and removal of the fluid by the aid of a fine trochar or the aspirateur, have also been employed as methods of treatment in this condition.

---

## CHAPTER XCI.

## DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

THE knowledge concerning diseases of the spinal cord has been greatly extended during late years, and the subject has now become a very wide one, as well as somewhat intricate. It is only intended in the present chapter to give an account of some of these diseases; certain complaints referred to the cord having already been sufficiently alluded to, while others will be more conveniently discussed in subsequent chapters. It will, however, render the subject clearer if at the outset a brief summary is given of the morbid conditions to which the spinal cord and its meninges are liable, and of the affections which have been associated by different writers with this portion of the nerve-centres.

The division of diseases into *functional* and *organic* deserves special recognition in the case of the spinal cord; but for practical purposes the conditions in which this structure is, or is supposed to be involved, may be conveniently arranged under three groups, as follows:—

1. *Certain definitely-named nervous diseases*, chiefly tetanus, tetany, chorea, hydrophobia, diphtheritic paralysis, lead-paralysis, paralysis agitans, writer's cramp and allied affections, and pseudo-hypertrophic muscular paralysis.

2. *Special functional disorders*.—These include:—*a.* Functional paraplegia, of various kinds. *b.* Spinal irritation, already described under HYSTERIA. *c.* Neurasthenia spinalis.

3. *Special organic diseases*.—Although the organic lesions to which the spinal cord is liable are few in number and easily understood, the individual diseases recognized under this group are very numerous, and their classification is a complicated one. The cord may be involved along with the brain, or with the nerves: When it is affected alone, the lesion may be limited to particular physiological tracts, constituting the group of so-called *system diseases*; or the morbid change is not thus limited, but more or less of the transverse area of the cord is affected, without reference to any special tracts—*indiscriminate diseases*. Again, a morbid condition may implicate the cord at different levels in different cases. And, finally, the disease may be primary in the cord itself; or secondary to, and associated with some extra-medullary mischief, such as caries of the vertebræ, or injury to the spinal column. It will thus be seen that great variety may be anticipated with reference to organic affections of the spinal cord, but the following list includes those which at present demand special recognition:—*a.* Injuries, especially concussion, this being the only injury which is not strictly surgical. *b.* Acute or subacute inflammatory diseases, namely:—(i.) Acute spinal meningitis, simple or tubercular. (ii.) Acute myelitis (diffuse or general, transverse, central, disseminated, &c). (iii.) Polio-myelitis anterior acuta. (iv.) Polio-myelitis anterior subacuta. *c.* Acute ascending paralysis. *d.* Hyperæmia and anæmia of the cord. *e.* Hæmorrhage. *f.* Chronic spinal meningitis. *g.* Chronic myelitis and softening. *h.* Progressive muscular atrophy. *i.* Glosso-labial paralysis. *j.* Diseases due to primary



sclerosis, including :—(i.) Locomotor ataxy. (ii.) Primary lateral sclerosis. (iii.) Amyotrophic lateral sclerosis. (iv.) Multiple or disseminated sclerosis. These will be considered in a separate chapter. *k.* Secondary degenerations, which have already been discussed. *l.* Tumours and new growths.

Having given this summary, I will now proceed to describe briefly the individual diseases of the spinal cord and its membranes which are not discussed in other parts of this work. The signs indicating mischief in different parts of the cord have already been pointed out in the chapter ON THE LOCALIZATION OF NERVOUS DISEASES, and these must be borne in mind in considering its separate affections. Their diagnosis, prognosis, and treatment will be discussed as a whole.

### I. NEURASTHENIA SPINALIS.

ÆTIOLOGY AND PATHOLOGY.—The term *neurasthenia spinalis* has been applied to a class of cases supposed to be due to a functional weakness of the spinal cord; but they have also been referred to a similar condition of the cerebellum. What the real state of the cord is can only be a matter of conjecture, but the symptoms have been attributed to marked anæmia, or to a mere alteration in the molecular conditions and actions of the nerve-elements, and impairment of their nutrition. Neurasthenia occurs chiefly in males, especially those of a neurotic temperament. Its main causes are sexual excess or masturbation, undue physical exertion and fatigue, and excessive mental work, especially when these causes are associated with deficient or much disturbed sleep. Sometimes the condition cannot be traced to any definite cause. It may be here mentioned that the symptoms of neurasthenia are very like those which are noticed during convalescence from a severe acute illness, especially of a febrile character.

SYMPTOMS.—These are indicative of great nervous debility, the most striking symptom being that the patient complains of extreme weakness and fatigue, or even of actual prostration, after the slightest exertion. Usually this is accompanied with coldness or numbness of the extremities; and not uncommonly with aching pains in the muscles of the limbs and back, and tenderness along the spine. There may be mental depression, indisposition or inability to perform mental work, and marked sleeplessness. The symptoms are liable to be aggravated after sexual intercourse, especially if excessive. There are no symptoms of any actual disease of the cord, such as paralysis or other phenomena. Not uncommonly patients who are the subjects of neurasthenia present a tolerably healthy appearance. The complaint varies much in its duration, but recovery may be brought about in most cases under suitable treatment, if sufficiently prolonged.

### II. CONCUSSION OF THE SPINAL CORD.

The description of the various injuries to which the cord is liable does not come within the province of this work; but a few remarks are needed with reference to concussion of this portion of the nervous system, as the effects of such an injury may come under observation in ordinary medical practice, especially as the immediate or remote consequences of

railway accidents, and they are particularly important in relation to actions for damages in such cases. It is difficult to determine the exact nature of the lesions produced in any particular case. Probably in many instances there is no obvious change. In some cases hæmorrhage occurs, either in connection with the meninges, or in the form of minute extravasations into the substance of the cord. Sub-acute localized inflammation may also be set up in the membranes shortly after an accident which leads to concussion.

**SYMPTOMS.**—When the symptoms of concussion of the cord immediately follow the injury, they are usually those of more or less shock and general nervous disorder, with others pointing more definitely to disturbance of the cord itself. There may be great tenderness along the spine, especially opposite the lowest cervical and first two dorsal vertebræ. Only exceptionally is there complete paralysis, but rather a paresis of one or more limbs; or if the paralysis is marked at first, it may pass away in a few days, sometimes suddenly. Twitchings and startings of the extremities are not uncommon. Sensation may be unchanged, exaggerated, or impaired. Constipation is often present; and micturition may be slow and difficult, followed by irritability of the bladder, and incontinence of urine. The symptoms gradually disappear in favourable cases; while in others they become worse, and give evidence of more definite lesions. Again, they may not appear at the time of the accident, but gradually supervene after a variable interval. In dealing with cases of supposed concussion of the cord much caution is necessary, as the symptoms are easily imagined, exaggerated, or pretended. They require to be watched for a considerable time; and to be examined repeatedly by some one thoroughly skilled in the investigation of such cases.

### III. ACUTE INFLAMMATORY DISEASES.

#### I. ACUTE SPINAL MENINGITIS.

In spinal meningitis the pia mater and arachnoid are usually chiefly affected—*leptomeningitis spinalis*; but occasionally the dura mater is most involved—*pachymeningitis spinalis*. The latter is almost always local and chronic; and acute spinal meningitis may be regarded for practical purposes as synonymous with *acute leptomeningitis*.

**ÆTIOLOGY.**—Acute spinal meningitis may result from :—1. Traumatic injury. 2. Caries of the vertebræ. 3. Sacral bed-sores penetrating deeply, and opening the spinal canal by destroying the sacro-coccygeal ligament. 4. Exposure to cold and wet, especially local; to sudden changes of temperature; or to powerful direct heat over the spine. 5. Acute rheumatism very rarely. 6. The bursting of an abscess into the spinal canal. 7. Adventitious deposits and tumours, especially syphilitic growths and tubercle. 8. Tetanus, chorea, or hydrophobia, it is said. 9. Epidemic cerebro-spinal meningitis. 10. Extension from cerebral meningitis.

**ANATOMICAL CHARACTERS.**—The *post-mortem* appearances resemble more or less those of cerebral meningitis. Usually the membranes are extensively affected, the disease being more or less general. The pia mater is very vascular, infiltrated, and thickened. A soft exudation often covers its surface, as well as that of the arachnoid, which is also swollen and velvety; while fluid occupies the sub-arachnoid space, either

turbid and flocculent, or more or less puriform in appearance. This may be so abundant as to distend the dura mater considerably. The latter is frequently reddened, and exudation of pus may accumulate between it and the bones, or it may exhibit signs of local injury or irritation. These conditions are especially seen in connection with caries of the vertebræ, or sacral bed-sores. The pus in the arachnoid cavity may be fetid, dirty-looking, and greenish. The inflammatory products are generally more abundant towards the posterior surface of the cord, on account of gravitation. In *tubercular spinal meningitis* grey granulations are visible, often in considerable numbers, especially on the surface of the arachnoid. The superficial layer of the cord, as well as the roots of the spinal nerves, are very often involved in the inflammatory process. Should recovery take place, absorption and organization ensue, and adhesions form.

**SYMPTOMS.**—Acute spinal meningitis is characterized at the outset by signs of irritation of the roots of the spinal nerves; followed by those of paralysis. The disease often sets in insidiously, and at first may be mistaken for rheumatism. In other cases it is ushered in with chills or a rigor, or with convulsions, followed by a variable degree of irregular pyrexia, with its accompanying symptoms; or it may follow cerebral meningitis. The early symptoms are severe paroxysms of pain felt along the spine, but only coming on when the patient moves; tenderness on deep pressure, though not very marked as a rule, and the pain is not increased by percussion; pains shooting from the spine into the limbs and trunk, but especially into the legs, though sometimes they may be chiefly felt in the arms, or even in one arm if the inflammation is limited; some degree of hyperæsthesia; contraction and rigidity of the muscles of the neck and back, which may cause opisthotonos, usually regarded as being due to tetanic spasm, but also considered as an instinctive act to avoid pain; fits of painful spasm in the limbs, neck, and back, with involuntary startings and jerks, but not the powerful spasms observed in tetanus; some embarrassment of breathing, which may be due to pressure by the anterior muscles of the neck upon the larynx, when the breathing is stridulous, and it becomes considerable if the respiratory muscles are affected with spasm, being then attended with a sense of oppression and suffocation; occasionally some difficulty of mastication and deglutition; and irritability of the bladder, or sometimes retention of urine. The reflex irritability is exaggerated at this time, but not to an extreme degree. The patient is usually very restless, anxious, and sleepless; at the same time keeping instinctively as quiet as possible. There are no prominent head-symptoms, if the cerebral membranes are free. Afterwards tingling, formication, and numbness are not uncommonly complained of; while increasing muscular weakness is observed, beginning below and extending upwards; with partial loss of control over the bladder and rectum, involuntary discharge of urine and fæces consequently taking place. Priapism is rarely observed. At this stage the reflex irritability is impaired. In fatal cases death may result from asphyxia; from asthenia and wasting; from implication of the cerebral membranes; or from the cord being compressed by inflammatory products, or becoming itself inflamed. Cases of spinal meningitis last a very variable time. If they become chronic, complete paraplegia is established, motor and sensory, with paralysis of the sphincters, and bed-sores ultimately supervene. Recovery may take place, even in severe cases.



## 2. ACUTE MYELITIS—INFLAMMATION OF THE CORD.

Under this heading *indiscriminate* lesions of the cord will be dealt with, of an acute inflammatory nature.

**ÆTIOLOGY.**—Acute myelitis may result from caries of the spine; injuries, including also severe strain and concussion; excessive muscular effort; irritation by adventitious growths or clots; cold and wet; or direct exposure of the spine to powerful heat. The disease has been attributed to suppression of perspiration or of chronic discharges; the sudden cure of a chronic skin-disease; or sexual excess. Some of the causes mentioned seem to *predispose* to acute myelitis. The cord becomes more or less inflamed in connection with spinal meningitis. A form of myelitis, usually subacute, is in rare instances associated with acute febrile diseases, such as typhoid, small-pox, or diphtheria. The complaint is most common in youth and early adult life.

**ANATOMICAL CHARACTERS.**—Inflammation of the spinal cord, when primary, usually begins in the central grey matter, and may either extend along this from one end to the other, or be confined to one or more portions of the cord, spreading throughout its entire thickness, especially opposite the lumbar enlargement. According to its distribution, acute myelitis has been divided into several forms, namely, *general*, *central*, *transverse*, *unilateral*, *disseminated*, and *bulbar*, in which the medulla oblongata is also involved. If myelitis follows meningitis the white substance is first involved, and the central grey matter is not implicated for some time. Three stages are described in the morbid changes characteristic of acute myelitis, namely, those of congestion; exudation and softening; absorption and cicatrization. As usually seen, the affected tissue is much softened, being often of cream-like consistence; more or less reddened at first, but afterwards it becomes yellowish; swollen and relaxed, the entire cord sometimes presenting a distended appearance, or being nodulated on the surface, while on section the inflamed tissue rises above the level of the healthy structure. The central grey matter has lost its normal contour. Extravasations of blood are liable to occur; and Charcot believes that hæmorrhage into the substance of the cord is generally, if not always, the result of previous inflammatory softening. Very rarely abscesses form. There is always more or less spinal meningitis. If the third stage is reached, the affected parts of the cord become contracted and sclerosed, the softened materials being absorbed; and rarely a cyst remains. Subsequently secondary descending degeneration of the pyramidal tracts often occurs. In the early stage microscopic examination reveals accumulation of blood in the vessels; leucocytes in the lymphatic sheaths; hypertrophied and swollen axis-cylinders and nerve-cells; and proliferation of connective-tissue elements. A glistening colloid substance has also been described surrounding the vessels, sometimes present in their interior, and infiltrating the tissues, the nerve-cells of the anterior cornua being often greatly swollen and distended. Some authorities, however, regard this material as merely due to the action of spirit in hardening the cord for examination. When softening occurs, the affected parts are more or less disorganized, as revealed by broken-down nerve elements, granular corpuscles, and abundant fat-granules and oil-globules. If sclerosis ensues, the connective-tissue and its cells become increased, and the blood-vessels are sometimes greatly dilated and hypertrophied.

**SYMPTOMS.**—Primary myelitis is characterized by the absence or slight degree and short duration of the signs of irritation observed in meningitis; with the rapid development of those indicative of destruction of the cord. The disease may set in gradually, with premonitory symptoms; or with remarkably acute or even sudden intensity. Its special symptoms are slight pain over the spine, usually circumscribed, not increased by movement or moderate pressure, but intensified by kneading, and especially by applying a hot sponge or ice, which produces a burning sensation at the upper limit of the inflammation; a feeling of constriction around some part of the trunk, as if it were bound by a tight cord—*girdle sensation*; marked paræsthesiæ in the limbs and trunk, but especially in the legs, such as tingling, formication, furriness, numbness, or subjective coldness, speedily followed by hypæsthesia or anæsthesia and analgesia, more especially in the legs, but sometimes more extensively distributed; marked restlessness, followed rapidly by muscular paralysis below the seat of inflammation, therefore usually paraplegic in its distribution; loss of power over the bladder, not uncommonly accompanied with great irritability, the patient desiring to have the catheter passed very frequently; constipation, followed sometimes by involuntary evacuations, from paralysis of the sphincter ani; and constant priapism. In the paralyzed parts electric sensibility and contractility are usually impaired or lost. The condition of the reflexes will depend on the site of the inflammation; so long as the reflex arc of a spinal segment is uninjured, its reflex movements are not affected; if the pyramidal tracts or lateral columns are involved, giving rise to secondary descending degeneration, there is an exaggeration of the reflexes passing through segments lower down. In consequence of the extensive destruction of the grey matter in many cases, the trophic lesions already described as being associated with diseases of the spinal cord are very liable to arise, namely, rapid wasting of muscles, which present the “reaction of degeneration;” the formation of acute bed-sores; and inflammation of the bladder and kidneys. Acute myelitis usually gives rise to little or no fever. The extent and precise character of the symptoms in acute myelitis will vary much, according to the height of the disease in the cord; and its exact distribution. Thus when it involves the upper part of cord, the arms are paralyzed; breathing is more or less impeded, and may be gravely disturbed; the voice is weak; there may be dysphagia or difficulty of speech; hyperpyrexia is often present; and the cardiac action is liable to be disordered. Again the condition of the bladder and rectum will depend much on the seat of the disease. In some instances sensation is only partially affected, as evidenced by inability to localize tactile impressions, slowness of conduction of sensation, anæsthesia dolorosa, or a condition in which a touch on the skin produces a diffuse sensation of vibration and pain in the whole of the extremity. Acute myelitis often soon terminates fatally from asthenia, apnœa, pulmonary complications, cystitis and renal disease, or acute bed-sores; in other cases chronic disease of the cord remains; while in others still improvement takes place, and rarely a complete cure is established.

### 3. POLIO-MYELITIS ANTERIOR ACUTA.

In the disease thus named there is an acute inflammation of the *anterior cornu* of the cord, and its usual clinical form is the so-called *infantile paralysis*; occasionally it occurs in adults, and is known as *adult spinal paralysis*. Each of these forms requires brief consideration.

#### a. Essential Paralysis of Children—Infantile Paralysis.

**ÆTIOLOGY.**—The causation of infantile paralysis is but little known. The complaint occurs in the large majority of cases between 6 months and 3 or 4 years of age, especially in the second year of life, but it may be met with in patients from 2 months to 8 or even 10 years old. Sex and constitutional condition have no influence. It sometimes follows one of the acute exanthemata or other febrile diseases; and has also been attributed to painful dentition; injury to the back; cold from lying on damp ground, or simple exposure to cold and wet; and digestive derangements. Most cases occur during summer and autumn.

**ANATOMICAL CHARACTERS AND PATHOLOGY.**—It seems now to be generally agreed upon by pathologists that infantile paralysis is due to *polio-myelitis anterior acuta*. The inflammation is practically confined to the anterior cornu, and it may occur in different regions, but is usually situated in the cervical and lumbar enlargements, the latter being often alone involved. It may, however, appear in scattered patches, or tolerably uniformly through a considerable vertical extent of the cord. One or both anterior cornua may be affected, but not always symmetrically. There is a difference of opinion as to whether the inflammatory process begins in the neuroglia or in the nerve-elements, but Charcot is of opinion that the latter are first affected, as the inflammation is sometimes limited to them, or only the immediately surrounding neuroglia is implicated. At any rate, more or less of the multipolar nerve-cells become rapidly and completely destroyed; while others are disabled temporarily, but ultimately recover. The involved portions of the cord are softened. The microscope reveals the changes already described as characteristic of acute myelitis. Subsequently the softening is less marked, and finally disappears, the cornua becoming atrophied, shrunken, indurated, and sclerotic, the nerve-elements having to a great extent or entirely disappeared, and the connective tissue being increased. The axis-cylinder processes of the affected nerve-cells, the fibres of the anterior roots of the nerves, and the muscles which they supply undergo secondary atrophy and degeneration, often with great rapidity. The muscles connected with the nerve-cells which are only temporarily disabled are only temporarily paralyzed, and subsequently recover. The wasted nerve-tubules become smaller, and lose their medullary sheath. The involved muscles also shrink rapidly; there is some increase of the cells of the sarcolemma, and, according to some observers, of the intervening connective tissue. Subsequently the muscular fibres lose their transverse striæ, and become atrophied, many undergoing more or less fatty degeneration, often with increase in the connective tissue, and the nuclei being increased in number; sometimes



a large accumulation of fat takes place, causing the muscles to be enlarged.

**SYMPTOMS.**—The invasion of infantile paralysis is usually indicated by some *premonitory* symptoms, especially pyrexia, of variable degree, but generally not very marked and exhibiting remissions, which lasts from 24 to 48 hours; sometimes by convulsions, not involving the face, and unattended with cerebral symptoms. In exceptional cases mental excitement, delirium, or loss of consciousness is noticed at the outset; or paralysis may set in suddenly without any warning. At first the paralysis is often more or less general, affecting both sides, but usually the lower limbs more than the upper, so that the child lies quite helpless; in many cases it is paraplegic in distribution, occasionally monoplegic, very rarely hemiplegic. The paralysis attains its highest degree at once, any subsequent changes being in the direction of improvement. Rarely are all the muscles of an affected limb involved, and parts of muscles may escape. The paralysis attacks muscles or parts of muscles which are functionally related. Very exceptionally muscles supplied by cranial nerves are implicated (Buzzard). The affected muscles are relaxed and flaccid; while some of them become rapidly wasted, and exhibit the "reaction of degeneration." Fibrillar twitchings are frequently seen. The reflexes are either much impaired or abolished. Sensation is not perceptibly affected as a rule, but children who are old enough may complain of pains in the limbs and back, and there may be some numbness, which, however, soon disappears. The sphincters are not involved, or only for a very brief period. In rare instances the paralysis disappears entirely in a few days, and the patient is completely restored. The ordinary course of events, however, is for some of the limbs or muscles to recover in from two or three days to a fortnight, while others remain permanently paralyzed. This permanent paralysis is generally of paraplegic distribution, though one leg is more affected than the other; in exceptional cases it is hemiplegic, or a leg and an arm may be implicated on opposite sides, or the paralysis may be confined to one limb, or even to a part of it. Subsequently the paralyzed parts become atrophied, limp, and stunted in their growth, the bones included; electric irritability is entirely lost; all the tissues undergo degeneration; the local pulse becomes small, and the circulation languid; the temperature falls considerably and permanently; and various deformities and distortions arise, according to the part involved, such as club-foot, flexion of the hips, &c., these being much aided by the great laxity of the ligaments and the mobility of the joints, and being usually due to the changes in the muscles, and to unrestrained action of non-paralyzed muscles. Those who have been subjects of infantile paralysis often live to an advanced age, and many belong to the class of mendicant cripples.

### *b. Adult Spinal Paralysis.*

Adult spinal paralysis has been chiefly described by Duchenne and Charcot, and is supposed to be of the same essential nature as infantile paralysis. It begins with febrile symptoms, and not unfrequently pain in the spine, with forward curvature, and some degree of pain in the limbs; motor paralysis, of variable extent, occurs either from the outset or speedily; cutaneous sensibility is unaffected; there is no loss of power over the bladder or rectum; nor is there any tendency to the

formation of bed-sores. The affected muscles are flaccid, and tend rapidly to waste and to lose their electric contractility. Adult spinal paralysis is said to differ from infantile paralysis in that the cerebral nerves are more frequently affected; headache is often observed at the commencement; and aching pain or occasionally tenderness may be felt in the paralyzed muscles. In one case observed by Dr. Byrom Bramwell there was temporary aphasia. In the subsequent course of the complaint various degrees of improvement take place in the muscles; and deformities do not occur if the bones and joints have reached their full development.

#### 4. POLIO-MYELITIS ANTERIOR SUBACUTA.

ÆTIOLOGY AND PATHOLOGY.—An affection has been thus separately described, of extremely rare occurrence, due to subacute inflammation or degeneration of the anterior cornu, with atrophy of the anterior nerve-roots. It was first described by Duchenne, who named it "*paralysie générale spinale antérieure subaiguë*." In some cases it is chronic throughout. Nothing definite is known about its causation, but Erb has suggested that in some cases the condition is associated with lead-poisoning. It occurs principally in persons between 30 and 50 years of age.

SYMPTOMS.—This complaint comes on insidiously, without any marked fever or other prominent symptoms, but there may be very slight pyrexia, or shooting pains in the back and limbs. Paralysis sets in, usually beginning in the legs, and travelling upwards—*ascending type*; sometimes starting in the arms, beginning in the extremities of the fingers—*descending type*. It progressively increases in degree, the affected muscles being flaccid from the outset. They speedily waste, all the paralyzed muscles undergoing this change simultaneously, so that the limbs shrink markedly. They also exhibit the "reaction of degeneration." The reflexes are first diminished, and subsequently abolished. The skin is apt to become cold and livid. The muscles of the trunk, head, and neck may be afterwards involved; and if the disease is not arrested, the medulla oblongata becomes involved, with the usual consequences. In most cases the paralysis is more marked on one side than the other. Sensation is practically unaffected, but there may be slight numbness. The bladder and rectum perform their functions properly. This disease usually progresses, either continuously or with remissions or intermissions; or temporary improvement may take place; or even complete restoration, either permanent or followed by subsequent relapse. Recovery may occur even after a long duration of the disease. The muscles are restored in the reverse order to which they are attacked. If death occurs, it results usually from implication of the medulla oblongata.

#### IV. PARALYSIS ASCENDENS ACUTA—LANDRY'S PARALYSIS.

This is another very rare and peculiar disease, originally described by Landry, with regard to the pathology and ætiology of which nothing positive is known. *Post-mortem* examination in undoubted cases has not revealed any lesion in the nervous system or muscles. The complaint occurs between 20 and 40 years of age, and chiefly amongst men. It has been attributed to previous acute febrile diseases; syphilis; and exposure to cold and wet.

**SYMPTOMS.**—Acute spinal paralysis sometimes sets in suddenly; more commonly there are premonitory symptoms, such as slight fever, numbness in the limbs, and a sense of heaviness or weakness. The paralysis begins in the toes and feet, rapidly becomes complete, and extends upwards, involving successively the legs and thighs, the upper extremities, beginning in the hands, the trunk, and the muscles of deglutition and respiration. The muscles do not waste much, and do not exhibit the “reaction of degeneration.” The reflexes are soon diminished or abolished. Sensation is but slightly affected; the bladder and rectum seldom suffer; and there are no trophic changes in the skin. Usually there is no fever, but this sometimes occurs. Only a few cases recover, a fatal termination usually resulting from asphyxia in from three or four days to two or three weeks, the average duration being from eight to twelve days.

#### V. SPINAL CONGESTION AND ANÆMIA.

**ÆTIOLOGY AND PATHOLOGY.**—Our knowledge concerning derangements of the supply of blood to the spinal cord is certainly at present very vague and unsettled. *Hyperæmia* of this part is supposed to be *mechanical* or *active*. *Mechanical* congestion is present in cases of cardiac disease obstructing the general venous circulation; and it is believed that it may be local, due to pressure on a particular vein. *Active* hyperæmia has been attributed to vaso-motor paralysis, of reflex origin; and it constitutes the first stage of myelitis. *Anæmia* of the cord may result from general anæmia, embolism, or thrombosis; from local pressure; or, it is said, from vaso-motor irritation, leading to spasmodic contraction of the arteries. This last-mentioned condition has been regarded by Brown-Séquard as the cause of “reflex paraplegia.” Spinal anæmia has also been referred to aortic regurgitation; and to sudden plugging of the abdominal aorta. Dr. Moxon has shown that, from its peculiar mode of blood-supply, the lower end of the cord readily becomes anæmic.

**SYMPTOMS.**—Congestion of the cord has been supposed to be characterized by the sudden onset of incomplete spinal symptoms, which afterwards disappear, but are liable to recur, namely:—some degree of dull aching along the spine, increased by heat, but not by movement or pressure; aching pains in the limbs, with variable paræsthesiæ, such as tingling in the toes and fingers, numbness, or sometimes hyperæsthesia, but no anæsthesia; twitchings in the limbs, with partial loss of power in the legs, or sometimes in the arms, often unequal on the two sides, there being no evident alteration in reflex irritability or in electric irritability or sensibility, or any tendency to wasting or other signs of impaired nutrition. The bladder and rectum are not involved. Slight motor and sensory disturbances in the limbs often accompany persistent mechanical congestion of the cord, such as that which results from chronic heart disease. *Anæmia* of the cord has, as already stated, been made to account for some forms of paraplegia. When it is a part of general anæmia no special symptoms are noticed. Sudden paraplegia appears to follow blocking of the abdominal aorta, as happens when this vessel is tied in animals, the hinder limbs becoming paralyzed; but how far this is due to anæmia of the cord, or to cutting off the blood-supply from the lower extremities, is a matter of doubt.



## VI. SPINAL HÆMORRHAGE OR APOPLEXY.

**ÆTIOLOGY AND PATHOLOGY.**—Hæmorrhage associated with the spinal cord is of extremely rare occurrence. Blood may be extravasated either into the cord itself; or in connection with the membranes, between them or outside the dura mater. By far the most common cause, on the whole, is traumatic injury, but the bleeding then usually takes place outside the cord, and rarely into its substance. Occasionally hæmorrhage into the cord seems to be spontaneous, owing to previous disease of vessels; usually it supervenes upon some other disease, such as acute myelitis, softening, or the rupture of soft growths. Hæmorrhage associated with the membranes may be due to *pachymeningitis hæmorrhagica*; the bursting of an aneurism of the aorta; or purpura, scurvy, and allied conditions. Blood may also escape from the cranial cavity into the spinal canal. Spinal apoplexy is most frequent from 10 to 20 years of age.

**ANATOMICAL CHARACTERS.**—In hæmorrhage into the cord the blood is almost always confined to the central grey matter, and is generally very small in amount. It destroys the involved nerve-structures; and subsequently inflammatory and degenerative changes are set up. The quantity extravasated in connection with the membranes varies much. It is usually clotted and dark; but may be partly fluid.

**SYMPTOMS.**—1. *Into the spinal cord.*—When hæmorrhage into the cord is sudden, this is evidenced by sudden acute pain in the back, with signs of severe shock to the system, the patient being sometimes unconscious for the time; complete and permanent paralysis of motion and sensation in the legs, or more extensively, according to the seat of mischief; paralysis of the bladder and rectum, with the usual consequences; and priapism. The paralysis comes on with great rapidity under such circumstances; but if the hæmorrhage is gradual it may be some hours before paraplegia is fully established. There may be some signs of irritation at the commencement, such as shooting pains in the limbs, hyperæsthesia, and muscular spasms or twitchings, but they are usually absent, and are never marked. The pain in the back soon subsides. At first all reflexes are abolished; and the temperature of the legs occasionally falls. The paralytic symptoms are usually permanent. Rapid atrophy, with the “reaction of degeneration,” is set up in all muscles in immediate relation with the damaged portion of the cord. When the hæmorrhage is in the dorsal or lower cervical region there may be increased temperature in the legs in a day or two, and some of the reflexes may become exaggerated. Trophic changes in the skin are common, giving rise to large bed-sores; and cystitis occurs early. Very rarely hæmorrhage into the cord gives rise to *hemi-paraplegia*.

2. *In connection with the membranes.*—If the hæmorrhage is abundant the symptoms are as above; but indications of severe irritation are first observed, in the way of painful sensations shooting from the spine, hyperæsthesia, shooting pains in the limbs, painful spasmodic movements in the limbs, rigidity, opisthotonos, or even strong convulsive movements, followed by paralytic symptoms. These are seldom very marked, and come on late. The functions of the bladder and rectum are not much interfered with; and bed-sores do not occur. Recovery often takes place, and may be complete.

## VII. CHRONIC SPINAL MENINGITIS.

**ÆTIOLOGY AND PATHOLOGY.**—Several forms of chronic inflammation in connection with the spinal meninges are described, namely:—  
 1. *Chronic leptomeningitis*. 2. *Pachymeningitis externa*. 3. *Pachymeningitis interna hæmorrhagica*. 4. *Pachymeningitis interna hypertrophica*.  
*Chronic leptomeningitis* may remain after the acute disease; or may be gradually set up. The chief causes to which it has been attributed are exposure to cold and wet; injury; syphilitic and other growths; and intra-medullary diseases extending to the surface of the cord. *Pachymeningitis externa* is set up by some local irritation, especially spinal disease, or an abscess or deep bed-sore. *Pachymeningitis interna hæmorrhagica* is usually observed in cases of general paralysis of the insane, being associated with a similar condition inside the skull; it has also been referred to alcoholism and injury.

**ANATOMICAL CHARACTERS.**—All forms of chronic spinal meningitis are usually localized, but sometimes the membranes are extensively involved. *Chronic leptomeningitis* is characterized by dilatation and thickening of the walls of the blood-vessels; increase in amount, and turbidity of the spinal fluid usually; opacity, thickening, induration, and roughness of the membranes; remnants of old inflammatory products; adhesions or bands passing across the sub-arachnoid space; firm adhesions of the pia mater to the cord; and sometimes calcareous deposits. The cord itself may be more or less affected, as evidenced by thickening of its connective tissue septa, and superficial softening or sclerosis. The roots of the nerves may also be compressed, softened, or atrophied. In *pachymeningitis externa* the inflammation begins in the outer part of the dura mater, and in the connective tissue between it and the spine; but it extends inwards and may reach the pia mater. There is a large amount of exudation, and the dura mater is greatly thickened, thus causing much pressure on the nerve-roots and cord. In *pachymeningitis hæmorrhagica* the inner surface of the dura mater presents a thick stratum of exudation; partly soft, partly organized; of rusty or brown colour, from extravasated blood; and enclosing many thin-walled vessels. There may be recent blood visible, or remnants of it, in the form of cysts containing clots. *Pachymeningitis hypertrophica* also affects the inner surface of the dura primarily, but extends to the arachnoid and pia mater. It is observed in connection with the cervical enlargement of the cord, and has been described by Charcot as *pachymeningite cervicale hypertrophique*. The layers developed upon the dura mater differ from those on the cerebral portion of the membrane, in being from the first dense and tough, only slightly vascular, and therefore not inclined to bleed. Enormous thickening is ultimately produced, a cicatricial fibrous tissue being formed. The morbid change usually extends in a ring round the cord, which is slowly compressed, the nerve-roots being first irritated, and then destroyed.

**SYMPTOMS.**—The symptoms of *chronic spinal meningitis* are described as slight pain over some part of the spine; severe pains in the limbs, of a rheumatic character, or shooting; paræsthesiæ or hyperæsthesia in the legs, with gradually increasing hypæsthesia, but not complete anæsthesia; slight spasmodic movements in the limbs, or rigidity, followed

by paralysis, beginning in the lower extremities, and gradually extending upwards, so that the trunk, bladder, rectum, or even the arms may be ultimately involved, the paralysis being at first slight, increasing very slowly, and being persistent but subject to marked variations in its course. The symptoms are usually very gradually developed, and are more localized than in acute spinal meningitis. In the earlier period the muscles are not obviously wasted, and reflexes are maintained; subsequently muscles which are supplied by involved nerves may waste markedly, and their reflexes are diminished or abolished. The bladder and rectum are only affected when the nerves arising from the lower part of the cord are implicated. Ultimately all the signs indicative of destruction of the cord may be developed.

All forms of *pachymeningitis* are characterized by localized pain; accompanied with phenomena corresponding in their distribution to the seat of the lesion, and indicative of irritation followed by compression of the anterior and posterior nerve-roots, as well as of slow compression of the cord. They are often very indefinite. *Pachymeningitis hæmorrhagica* is usually associated with the symptoms of a similar condition in the cerebral membranes; and signs of meningeal hæmorrhage may occur at any time, owing to rupture of the thin-walled vessels in the exudation.

*Pachymeningitis hypertrophica* demands brief special notice. Clinically Charcot divides the disease into two stages, namely (1) that of irritation; and (2) that of paralysis and atrophy.

In the *first stage* the symptoms are acute pains at the back of the neck, shooting to the head and arms, constant, but liable to exacerbations; twitchings, spasms, and rigidity of the muscles, especially of those of the neck, which is kept fixed; hyperæsthesia, formication, and a feeling of weight in the limbs; more or less muscular weakness; and not uncommonly herpetic or bullous eruptions. In the *second stage* the pains in the limbs cease; while the muscles of the arms become gradually paralyzed and wasted, especially those of the forearm. The faradic irritability of the muscles is slowly abolished; and characteristic contractures and deformities supervene. If the lesion involves the upper part of the cervical enlargement the musculo-spiral nerve is chiefly involved, and the corresponding muscles are paralyzed; if it occupies the lower part of the enlargement, the median and ulnar nerves are mainly affected, and a form of "claw-hand" is produced. Patches of anæsthesia also are noticed on the arms and upper part of the trunk, corresponding to the compressed sensory nerve-roots. Subsequently the cord being destroyed throughout at the seat of lesion, and secondary descending degenerations taking place, spastic paraplegia gradually supervenes, with sensory disorders and other phenomena characteristic of these morbid changes. This disease is always very chronic; and occasionally it seems to be arrested, and some degree of improvement may even take place.

### VIII. CHRONIC MYELITIS—WHITE SOFTENING.

**ÆTIOLOGY AND PATHOLOGY**—There has been much controversy as to the nature of chronic "softening" of the spinal cord. Many authorities regard the condition as having two distinct modes of origin, namely, in inflammation, and in degeneration. Others consider that the change is



always of a degenerative kind. It seems certain that softening may remain after an attack of acute or sub-acute myelitis; and there is probably also a chronic form of this disease. *Simple* or *non-inflammatory* softening results in the great majority of cases from deficient supply of blood. This may be due to mere degeneration of the blood-vessels, narrowing their calibre; but sometimes follows embolism or thrombosis. The lower end of the cord is especially liable to become softened from this cause, on account of its peculiar blood-supply (Moxon). Softening is often caused by gradual pressure upon the cord, and is then said to be chiefly inflammatory, and only occasionally simple. It may also follow some injury.

**ANATOMICAL CHARACTERS.**—Softening of the cord is indicated by various degrees of diminished consistence. In chronic myelitis it is said that this is much less marked than in the acute form, and that there is a greater degree of sclerotic change. The softened portion may be quite white, or more or less red or yellowish. Erb distinguishes inflammatory from non-inflammatory softening by the microscopic appearances. In the *inflammatory* form he describes “a large number of cells containing fat-granules, tensely distended blood-vessels, numerous young cells, increase of the interstitial tissue, swollen axis-cylinders, &c.,” in *simple* softening merely “swollen and disintegrated nerve-fibres, ganglion-cells in a state of glassy swelling, a few cellular elements and fat-granule cells, and a small quantity of fatty detritus. It must be noticed that in many cases of chronic myelitis the cord is firmer than natural, on account of considerable increase of connective-tissue elements, and gradual removal of the nerve-tissues. Indeed, by some pathologists the condition termed *sclerosis*, with the particular diseases to which this change gives rise, is believed to be inflammatory in its origin. Chronic myelitis is very variable in its distribution, hence named *transverse*, *disseminated*, *annular*, *general*, &c.

**SYMPTOMS.**—If the cord becomes gradually softened, from whatever cause, the signs of the change are more or less of the following character:—Dull pain or uneasiness over some portion of the spine, increased by pressure, percussion, or the application of a hot sponge or cold, but not by movement; a feeling of tightness round the body; distressing paræsthesiæ, wandering pains or fidgetty sensations in the legs, followed by gradual loss of feeling to complete anæsthesia, this often extending up the body to a variable extent; twitchings, spasmodic movements, and cramps in the legs, with diminished power, dragging of the legs in walking, and a sense of heaviness and fatigue, culminating in paraplegia; frequently a marked tendency to painful contractions and rigidity in the paralyzed limbs, the legs being in many cases drawn up involuntarily if left to themselves, sometimes by jerks, so that the joints become strongly flexed, or one or both limbs being rigidly extended; tendency to wasting of the muscles of the legs, with failure of circulation and nutrition, the skin being often covered copiously with dried epithelium-scales, and bed-sores being very liable to form; paralysis of the bladder, leading to retention and decomposition of urine, and consequent cystitis and renal mischief; paralysis of the rectum, with unconsciousness of the passage of stools; gradual loss of sexual power and inclination, though there is often reflex priapism. In short, the symptoms of chronic myelitis or simple softening are usually those of slowly-developed chronic paraplegia in its most typical form. The condition of the muscles, and the state of the reflexes will depend on the exact seat and

extent of the disease. Often there is an exaggeration of the deep reflexes, but they may be impaired or lost. The clinical history is liable to vary much from the association of softening with other morbid conditions. In rare instances the cord may become so extensively diseased as to give rise to general paralysis, with marked wasting of the muscles, and loss of electric irritability. The general health often remains good; and patients may live for many years. Death may occur from the effects of the cord-lesion; or from intercurrent complications, such as phthisis or pneumonia.

#### IX. ADVENTITIOUS GROWTHS IN THE CORD.

New growths affecting the cord usually originate in connection with the spinal column, the meninges, or the nerve-roots, and very rarely in the substance of the cord itself. The most important are *cancer*, *tubercle*, *syphilitic formations*, *inflammatory growths*, and *sarcoma*. Of the rarer forms may be mentioned glioma, myxoma, fibroma, lipoma, cartilaginous growths, and hydatids. Some of these formations are the manifestation of a special diathesis; or they are secondary to similar deposits elsewhere. In some cases local injury has been set down as the *exciting cause*, but as a rule none can be discovered. Generally a growth connected with the spinal cord is localized and single; occasionally there are separate formations in different parts. The secondary effects which are usually liable to be produced are gradual displacement and atrophy of the nerve-elements; myelitis or simple softening, with subsequent secondary degenerations; chronic meningitis; and pressure upon the nerve-roots and its consequences. Rarely a sudden hæmorrhage takes place, with or without previous changes.

**SYMPTOMS.**—There is much variety presented by the symptoms of a tumour in connection with the spinal cord, depending upon the region it occupies, whether it begins in the substance of the cord or is extra-medullary, its effects upon the cord or nerves, the rapidity of its growth, and other circumstances. As a rule they are very gradual in their onset and progress, but occasionally sudden or acute symptoms occur, due to spinal apoplexy or to acute myelitis. Taking the mass of cases, the more characteristic phenomena may be described as localized pain in the back, especially in connection with cancer; neuralgic pains shooting from this point into the limbs or trunk, due to irritation of the nerves, with hyperæsthesia or paræsthesiæ, followed by anæsthesia if the cord becomes destroyed; muscular disturbance, succeeded by paralysis, in some cases beginning on one side and extending gradually to the other. Objective evidences of a tumour might be discovered on examination of the spinal region. If the growth is in the cord itself, there will generally only be gradual loss of its functions up to the level of the part involved. If the growth affects the cervical enlargement, the upper extremities are implicated. Sometimes one lateral half of the cord is affected, or only its motor or sensory portions. In the case of syphilitic disease, the symptoms often improve greatly or entirely disappear under appropriate treatment, owing to absorption of the deposit. Signs of some cachexia may be present; or there may be indications of morbid growths in other parts. Ultimately all the signs of destruction of the cord may be developed, death occurring from bed-sores, cystitis, paralysis of respiratory muscles, or pulmonary complications.

## X. GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

Although all the diseases of the spinal cord have not been discussed in this chapter, it will be convenient now to offer a few general remarks with reference to their diagnosis, prognosis, and treatment.

1. DIAGNOSIS.—When any case comes under observation in which the symptoms point to the spinal cord, the questions to be determined are:—1. Whether they are real, imaginary, or pretended. 2. Whether they are of functional or organic origin. 3. If due to organic disease, the nature of such disease; and its situation and extent, with reference to whether it is extra- or intra-medullary, the region it occupies, and the tracts or divisions of the cord which it involves. It will be unnecessary to recapitulate the clinical signs of the various complaints, but these must be duly considered in each case, as regards their mode of onset, nature, combinations, and progress; along with the history in all its details, the general state of the patient, and the condition of other parts and organs of the body. Of course appropriate skilled examination, directed to the spinal column, and to the investigation of the nervous system as a whole, is often of essential importance; and in any really difficult case it ought to be conducted by one who is experienced and thoroughly competent, otherwise serious mistakes are liable to be made. In some instances the effects of treatment give valuable diagnostic indications, as in the case of syphilitic disease, the symptoms due to this disease being often greatly improved, or entirely got rid of, by the administration of iodide of potassium. The influence of treatment in curing certain forms of functional paraplegia is also to be borne in mind.

Only a few observations need be made with regard to the diagnosis of the individual diseases of the spinal cord, and it must be remembered that different lesions are often associated together in the same case, or they become developed secondarily. The effects of *concussion* must always be studied with particular care and caution, as they are so easily pretended or imagined, and so much often depends on their correct observation and interpretation. *Neurasthenia spinalis* is generally readily diagnosed. Sudden lesions of the cord, not arising from injury, are due either to some *disorder of the circulation* or to *hæmorrhage*, and the symptoms are usually sufficiently characteristic. *Acute spinal meningitis* may be mistaken for tetanus; spinal congestion; or spinal irritation; but there is rarely any actual difficulty in making a diagnosis between them. *Myelitis* is distinguished from *meningitis*, whether acute or chronic, by the absence or slight degree of symptoms of irritation and severe pain; with rapid development of signs of destruction of the cord, and failure of its functions. *Chronic* affections of the cord leading to paraplegia may be simulated by various forms of *functional paraplegia*. In *reflex* paraplegia some cause can be discovered; the paralysis is in proportion to the intensity of this cause, and is generally partial and incomplete; there is no wasting of muscles; sensibility is usually normal; and the bladder and rectum are but little or not at all affected. The paralysis disappears if the cause is removed. *Chronic softening* of the cord is as a rule easily recognized by the local sensations; and the



permanent paralytic and other symptoms. There are no practical clinical distinctions between *inflammatory* and *simple softening*. The signs of *new growths* are as a rule sufficiently diagnostic. The *system diseases* of the cord already considered present highly characteristic phenomena. The disease due to sclerosis will be individually considered in the next chapter.

2. PROGNOSIS.—Great caution is required in giving an opinion as to the immediate and remote prognosis of cases where the spinal cord is involved, and this must depend in the first instance upon as accurate a diagnosis as possible. It must further be guided by the exact nature, severity, effects and symptoms, rapidity of progress, and direction of extension of the disease; the general condition and circumstances of the patient, as well as his mental state; the presence of complications, and their nature; and the results of treatment. *Functional* affections of the cord are often very difficult to cure, although not in themselves dangerous. *Acute inflammatory diseases* of the cord or its membranes are very grave, and often prove rapidly fatal. At the same time it must be remembered that in some very serious cases recovery takes place; or myelitis may remain as a chronic condition. Once the cord is transversely destroyed by a sudden, acute, or chronic lesion, permanent paralysis is established in the parts below the seat of mischief, but cases of this kind frequently linger on for a long while, and the patient may enjoy good general health, and be able to perform excellent mental work. Bed sores, cystitis, renal disease, and other untoward complications are, however, very liable to arise, and these add to the danger. Symptoms due to *syphilitic disease* of the cord often improve remarkably under appropriate treatment.

3. TREATMENT.—The indications for treatment of affections of the spinal cord lie within a very narrow compass, and it is well in dealing with them not to be too ready to resort to active measures. In the first place, any *cause* that can be discovered must be removed, if practicable, and this is of particular importance in the cure and prevention of functional disorders. Secondly, any *organic lesion* must be attended to, with the view of checking it, removing its effects, and repairing any injury which it has produced; but as a rule little can be done in this direction. Thirdly, *symptoms* must be watched and treated judiciously, especially with the view of influencing the functions of the cord, or of modifying symptoms and conditions depending on abolition or disorder of these functions. Fourthly, it is also very important to guard against *complications*, and to treat them as they arise. Fifthly, *general management* is often of much service in chronic cases; and the *general health* needs to be sustained.

As regards individual diseases, sudden lesions, such as *concussion* or *hæmorrhage*, must be treated on general principles applicable to such conditions. In treating any *acute inflammation* affecting the spinal cord or its membranes the patient should be kept at rest, lying on the side or in a somewhat prone position. Ice may be applied constantly along the spine. In some cases the application of leeches over this region is useful. Medicines are of doubtful value. Dr. Radcliffe recommends iodide of potassium with opium in the treatment of spinal meningitis. When the cord is involved, belladonna, conium, and ergot are believed to exercise a direct beneficial effect upon it. It is particularly important to attend to the bladder and bowels in all cases of disease of the cord; to see that the patient is kept clean and dry; and to guard against bed-

sores, for which object the use of a water-bed or air-bed is very serviceable. This applies more particularly to *chronic* affections, in which all that can be done further is to support the general health by good food, proper hygienic conditions, and the use of *tonics*, especially quinine, iron, phosphorus, or hypophosphites; to promote absorption of morbid products, particularly syphilitic deposits, by the aid of iodide of potassium and bichloride of mercury; to stimulate the functions of the cord by minute doses of strychnia or tincture of cantharides; and to treat paralysis or other symptoms, as well as complications. Electricity is often of great service, but must be carefully employed. Treatment directed to the spinal column is often demanded; and surgical interference may be necessary for deformities, abscesses, and other conditions.

---

## CHAPTER XCII.

### SCLEROSIS OF THE NERVE-CENTRES.

OF late years the morbid condition named *sclerosis*, which involves the nerve-centres, has attracted considerable attention. The change affects different parts of these centres in different cases, and sometimes the peripheral nerves, but the subject can be most conveniently discussed in a comprehensive article, first considering the lesion from a general point of view, as regards its *ætiology*, *pathology*, and *anatomical characters*; and then discussing the individual affections in which sclerosis is the pathological change. Certain diseases believed by some authorities to be of this nature, but the pathology of which is still disputed, will be separately considered. Different writers have adopted different classifications of the affections resulting from sclerosis, but the following list includes the chief varieties which have been recognized, and is sufficient for all practical purposes:—I. **DIFFUSED CEREBRAL SCLEROSIS** (Hammond). II. **SPINAL SCLEROSIS**, including (1) **Locomotor ataxy**; (2) **Primary lateral sclerosis**; (3) **Amyotrophic lateral sclerosis**; (4) **Secondary lateral sclerosis**. III. **DISSEMINATED or MULTIPLE SCLEROSIS**.

**ÆTIOLOGY.**—Age seems to have considerable influence as a *predisposing cause* of the various forms of sclerosis. The *diffused cerebral* form is said to begin during infancy. The *spinal* varieties occur chiefly from 25 to 45 or 50 years of age; while the *disseminated* form usually comes on between 20 and 25, seldom after 30, sometimes at the time of puberty. Males suffer in larger proportion than females, but amyotrophic lateral sclerosis is said to be more common in females; and Charcot affirms the same fact with regard to disseminated sclerosis, but others deny this. Hereditary predisposition is traceable in some cases, and if not to the actual disease, it may be to some other form of nervous disorder; locomotor ataxy occasionally runs distinctly in families, and a congenital variety of this disease and of primary lateral sclerosis have been described. The *exciting causes* of sclerosis are very obscure, and in many cases cannot be made out in the least. When of *cerebral* origin, it has been attributed in different instances to hæmorrhagic cysts; injury to the head;

acute fevers, especially typhoid and scarlatina; rheumatism or syphilis; dissipation; severe emotional disturbance; excessive mental application; or great muscular exertion. The *spinal* varieties have been supposed to be the result of previous inflammation of the cord or its membranes; injuries to the spine, or shock; over-exertion and straining; the constant maintenance of a bent position; sexual excess; exposure to cold and wet; gout, scrofula, or syphilis; acute febrile diseases; or abuse of alcohol. Secondary lateral sclerosis and other secondary forms of the complaint follow some preceding lesion.

**ANATOMICAL CHARACTERS AND PATHOLOGY.**—Sclerosis consists essentially of hyperplasia of the neuroglia, with atrophy and degeneration of the nerve-elements, which may ultimately lead to their complete destruction and disappearance. The process is looked upon by most pathologists as being of a chronic inflammatory nature; but others regard it as a degeneration. In most forms of sclerosis the primary change seems to be in the neuroglia, the nerve-elements being strangled and destroyed by the connective tissue which surrounds them; but in locomotor ataxy it is said to commence in the nerve-tissues themselves. The more obvious characters indicative of the sclerotic change are a greyish, semi-translucent appearance of the affected part; various degrees of increased firmness and induration, ultimately ending in marked hardness and toughness; and at first some tumefaction, but soon passing into contraction and condensation, with consequent diminished bulk and shrinking of the affected part. The colour may finally become greyish-white or yellowish-grey. Usually firm adhesions form with the corresponding pia mater, which also undergoes analogous changes.

As regards the microscopic changes in sclerosis, in the early period embryonic cells appear in the neuroglia and perivascular spaces, along with more or less increase of the amorphous intercellular substance in the neuroglia, and the connective-tissue corpuscles become large and prominent. Subsequently contraction and induration take place; the cells become small and indistinct; the intercellular substance becomes delicately fibrillated; and the walls of the vessels are thickened, with consequent narrowing of their channels, or they are sometimes dilated. Ultimately wavy bundles of connective tissue are seen. At first the nerve-elements present little if any change in most cases. Later on, if sclerosis involves the white substance, the nerve-fibres become more or less separated from each other, most of them diminish in size, though some may be normal or even enlarged, and they may present a moniliform appearance. Finally they become greatly atrophied, on account of the loss of their medullary sheath, but they are rarely entirely destroyed. In the grey matter the nerve-cells are also involved. In some cases Charcot describes in the early period changes in which the nerve-cells become swollen, sometimes being enormously enlarged, finally granular, and opalescent; their processes at the same time appearing more or less thickened and twisted. Usually atrophic changes take place in these cells. They may merely shrink in all directions and dry up; or sometimes a deposit of pigment occurs in them, while they diminish in size, assume a more or less globular shape, their processes become shortened and attenuated, and at last they only form minute roundish collections of pigment, or even disappear altogether. Lockhart Clarke has described irregular disintegrated patches as being occasionally observed in sclerotic tracts, from which all traces of the different tissues and blood-vessels have disappeared. Compound granule corpuscles,



oil-globules and granules, corpora amylacea, and sometimes crystals, supposed to be margarine, are visible under the microscope.

Having thus discussed the general nature of sclerosis, the several varieties of the disease, according to its distribution in different parts of the nervous system, will now be considered. It may be remarked that the lesion shows a peculiar tendency to be confined to certain tracts or regions, rarely passing beyond the limits of these portions of the nerve-centres. Moreover, the ultimate effect of the morbid process is seriously to impair, or even completely to abolish, the functions of the parts involved, though during its progress signs of irritation may be evident.

## I. DIFFUSED CEREBRAL SCLEROSIS.

Dr. Hammond describes a form of diffused cerebral sclerosis, in which a large portion or the whole of a lobe is involved, or sometimes even the entire hemisphere, and the lesion is not circumscribed. It becomes less marked at its circumference, and never invades the grey substance of the brain.

**SYMPTOMS.**—In diffused cerebral sclerosis the mental faculties either remain undeveloped to a variable degree, or become impaired if the disease sets in later in life. The patient never learns to talk, or speech becomes imperfect or lost after it has been acquired. Usually more or less hemiplegia is observed, with arrest of growth, contractions, and distortions of the affected limbs, in which sensation may also be impaired. One or more of the special senses are usually enfeebled or lost. Many of the patients suffering from this complaint belong to the class of idiots or imbeciles, whose habits are filthy, and who pass their excretions involuntarily. Frequent attacks of epileptiform convulsions are not uncommon during the progress of the lesion, with signs of cerebral irritation at an early period. The course of diffused cerebral sclerosis is very chronic, and patients often live to an advanced age.

## II. LOCOMOTOR ATAXY—TABES DORSALIS.

**ANATOMICAL CHARACTERS AND PATHOLOGY.**—This is one of the forms of sclerosis best known and most commonly met with. Many authorities look upon syphilis as a most important factor in the causation of locomotor ataxy, and by some it is even regarded as the sole cause, but this question is not yet settled. The disease involves the posterior columns of the spinal cord, as a rule equally, and often throughout their whole horizontal area, the changes commencing and being most marked in the lower dorsal and lumbar regions, and progressively diminishing upwards. Charcot has shown that the tracts which are essentially concerned in producing the symptoms of locomotor ataxy are two narrow bands of white matter, lying on each side between the inner and posterior aspect of the posterior cornu and nerve-roots on the one hand, and the posterior pyramid on the other—*postero-external columns*; in some cases the pyramids are quite healthy, but generally they become involved. Moreover, in most cases the internal radicular fibres of the posterior roots of the nerves, and the adjoining parts of the posterior cornua, become more or less implicated. Dèjérine has published cases in which chronic neuritis was found in the peripheral nerves,

with changes in the cord; and in some cases only peripheral nerve-changes were found, with nothing in the cord. He thinks tabes may arise from primary changes in the peripheral nerves. In exceptional instances the disease spreads to the lateral columns, or even to the anterior cornua; but, according to Charcot, this extension takes place along the internal radicular fasciculi, and not through the intermediate tissues. In the cervical region the lesion is generally limited to the postero-internal columns, presenting the characters of an ascending degeneration, but may involve the postero-external columns. It can generally be traced through the medulla oblongata. The cranial nerves, especially the optic nerve and disc, may be involved in the sclerotic change.

The spinal cord, in a case of advanced locomotor ataxy, appears flattened antero-posteriorly, while the posterior columns and nerve-roots are obviously wasted and shrunken. The membranes are usually thickened and adherent at the back of the cord. On section the posterior columns are found to be firm, and present a translucent grey appearance.

**SYMPTOMS.**—Locomotor ataxy in the large majority of cases comes on insidiously, and runs a very chronic course. In exceptional instances its characteristic symptoms are developed suddenly or rapidly. Certain so-called *premonitory* symptoms are usually observed, which may last for months or years, but these are really the early symptoms of the disease, constituting its *stage of invasion*. They may be summed up as follows:—1. *Sensory derangements* in the legs and lower part of the body, namely, an unusual feeling of fatigue after slight exertion; painful sensations in various parts of the limbs and about the joints from time to time, supposed to be rheumatic; and also extremely severe neuralgic pains, coming on suddenly and being of momentary duration, described as darting, boring, stabbing, cutting, throbbing, or like an electric shock—the so-called *lightning pains*; sometimes constrictive or girdle-pains, affecting the trunk or occasionally the limbs; hyperæsthesia, hypæsthesia, dysæsthesia, or paræsthesiæ of the skin, or retarded conduction of sensation. The lightning pains differ from ordinary neuralgia in being referred to the deeper structures, and not radiating along the superficial branches of any particular nerve. When the postero-external columns of the cervical portion of the cord are involved, these pains are felt in the arms, and rarely in the head. Hyperæsthesia is usually paroxysmal and fugitive in its characters. 2. *Internal pains*, referred to the bladder, urethra, or rectum; or, more particularly, extremely severe attacks of gastralgia, the pains shooting to the back, around the abdomen, and in other directions, and being accompanied with vomiting, dyspeptic symptoms, faintness, deranged cardiac action, and a feeling of marked illness. These attacks are known as *gastric crises*, and Dr. Buzzard has drawn particular attention to them. They are more frequent in females than in males. 3. *Paralysis of sensory or motor nerves*, sometimes temporary or recurrent, sometimes permanent. 4. *Abolition of the patellar tendon reflex*, which usually occurs at an early stage of locomotor ataxy, but is not an invariable symptom. The condition of the *superficial reflexes* varies, but it is said that in most cases the plantar reflex is impaired at an early period, and finally abolished. 5. *Disorders of vision and hearing*, and objective changes in connection with the eyes, such as dimness of vision at times, or even complete amaurosis; colour-blindness; diplopia; contraction of the field of vision; slight strabismus or ptosis;

atrophy of the disc or chronic neuritis. Extreme contraction of the pupils is often a marked feature, so that they become pin-pointed, and they may be unequal. The "Argyll Robertson phenomenon" is frequently observed, there being loss of the pupil-reflex to light; while the iris may still retain its contractility in connection with efforts of accommodation for near objects. Deafness is not uncommon. 6. *Sexual disturbance*. It is said that sexual desire is usually increased at first. Trousseau observed that there is in the early period a peculiar aptitude for repeating sexual intercourse a great many times within a short period. Soon, however, sexual power and desire become gradually lost. *Spermatorrhœa* is frequently complained of. 7. *Disorders of micturition*. In the early stage of locomotor ataxy the most frequent condition is irritability of the bladder, with painful micturition, and difficulty in retaining the urine, the patient being obliged to obey the call to urinate instantly. Occasionally there is a difficulty or inability to pass water, ending in retention. 8. *Constipation* is of very common occurrence, and may be accompanied with a peculiar painful feeling of over-distension of the rectum.

When locomotor ataxy is fully declared—*stage of full development*—the symptoms are very characteristic, and chiefly point to a *loss of the power of co-ordination in the muscles of the legs*, and of the *muscular sense*. At first the patient feels that he is losing control over the movements of his legs, and that he cannot walk steadily or firmly without support, but slips about and has an uncertain gait. This is particularly noticed in the dark, or when he shuts his eyes, and the patient finds that he has to pay special attention to the movements of his lower extremities, in order to carry them on properly. After a while the signs of impaired co-ordination are very evident when the patient is made to walk, and he presents a characteristic gait. He is very unsteady in his movements; tends to stagger and to advance precipitately; and during progression lifts the foot up to an unnecessary height, then throws it forwards and outwards, and brings down the heel suddenly with a heavy stamp. On turning suddenly he staggers or falls, and the same thing happens if he shuts his eyes when standing. Habitually he walks with sticks, and keeps his eyes fixed on his feet or on the ground in front of him; he takes his steps slowly, deliberately, and at regular intervals, and in uncomplicated cases walks in a straight line. The difficulty in walking, and the characteristic disorder of gait, are greatly aggravated by timidity and other mental causes. There is no paralysis, as is proved by the fact that the legs can be easily moved in all directions in the recumbent posture; and the muscles sometimes retain extraordinary power. At last walking becomes impossible, the legs being thrown hither and thither, without any appearance of design or control when any attempt at progression is made. The muscles do not waste, and retain their tone. The condition of electric irritability is doubtful, some observers affirming that it becomes much impaired, others that it remains normal; in the early stage it is often exalted. Sensation is frequently much altered; the pains in the limbs continue; often there is tingling or numbness in the toes and feet; cutaneous sensibility is impaired, the patient not feeling the ground properly, but having a sensation as if he were treading on wool or sand; and sometimes there are spots of complete *anæsthesia* to all stimuli except heat and cold. Muscular sense is also more or less diminished or even lost in advanced cases, the patient not being aware of the position of his legs when lying



down, unless he is looking at them. The electric sensibility is said to be impaired in those muscles in which the muscular sense is affected. There is no loss of power over the bladder and rectum as a rule, but the former may certainly be much affected, and there may be complete paralysis of this organ, with consequent retention of urine.

In the majority of cases of locomotor ataxy the upper limbs become involved sooner or later. Numbness is noticed in the fingers, generally beginning in the little and ring-fingers, and then often extending to the hand or arm. The movements of the fingers, hands, or arms become clumsy, unduly violent, and uncertain, so that the patient cannot perform any delicate combined movements requiring precision. Moreover, if he shuts his eyes, he cannot judge of the extent or direction of the movements of his upper limbs. The voluntary movements are often executed in a jerky manner. In some instances the muscles of the head, neck, and trunk become involved. Articulation may be impaired; and the different cranial nerves may become implicated permanently. Deglutition and respiration may also be affected. The occurrence of atrophy of the optic disc is stated by Charcot to be due to sclerosis commencing here, and gradually extending backwards along the optic tracts, as far at least as the corpora geniculata. In advanced cases of locomotor ataxy severe and constant aching pains are liable to arise in the head, along the spine, and in the trunk and limbs. Retention or incontinence of urine or fæces may occur; and sexual power and desire are lost. In exceptional cases rigidity, contraction, and wasting of muscles set in, due to the extension of the sclerosis to the lateral columns and anterior cornua. Bed-sores may also be produced.

During the progress of locomotor ataxy trophic lesions are liable to arise, as has been particularly shown by Charcot. Thus *cutaneous eruptions* sometimes supervene, more especially during periods of exacerbation of the disease, and in connection with the lightning pains, these eruptions being not uncommonly limited to the area of distribution of the painful nerve. They include lichen, urticaria, herpes zoster, ecthyma, impetigo, and erythema nodosum. *Joint-affections* are also occasionally met with, usually occurring at the onset of the symptoms of inco-ordination. The knees, elbows, or shoulders are chiefly implicated, there being much effusion into the joints, with very rapid destruction of the articular surfaces, and not unfrequently dislocation occurs. The bones also become very friable, and are liable to fracture spontaneously. These articular and osseous lesions are attended with little or no pain; but they may cause marked deformities. They are said to be more common in women, and Dr. Buzzard has shown that they are apt to be associated with gastric crises. This writer thinks that they are probably due to a lesion of the medulla oblongata.

The *course* of locomotor ataxy is very variable. The limbs may be involved unsymmetrically. Usually the disease is exceedingly chronic, and may last many years before it reaches its full development. In early cases treatment may check its progress, or even lead to improvement or a cure. As a rule locomotor ataxy tends to become worse and worse, perhaps with occasional remissions. The mental faculties generally remain quite clear, but it is affirmed that locomotor ataxy is not uncommonly associated with general paralysis of the insane. Febrile paroxysms sometimes occur; and repeated attacks of bronchitis are observed in exceptional instances. Death generally results from intercurrent disease; but may happen from implication of the muscles of

deglutition or respiration, bronchitis, renal or vesical disease, or bed-sores. The latest period of locomotor ataxy has been ranked as a separate stage, but it is hardly practicable to make any such division.

### III. PRIMARY LATERAL SCLEROSIS—SPASMODIC SPINAL PARALYSIS—SPASTIC PARAPLEGIA.

**PATHOLOGY.**—This form of sclerosis of the cord has been only comparatively recently recognized as a distinct disease, and is of exceedingly rare occurrence. Erb and Charcot predicted the probable pathology of the complaint, and Dr. Dreschfeld has since described the morbid changes in a case in which he had the opportunity of examining the spinal cord. He found a band of sclerosis occupying the greater portion of the lateral columns, throughout the cervical, dorsal, and lumbar regions, but not implicating the grey matter or extending to the surface of the cord, while the anterior and posterior columns were quite healthy. In short, primary lateral sclerosis involves symmetrically the *crossed pyramidal tracts*. The complaint is usually met with in strong, muscular, male adults.

**SYMPTOMS.**—Primary lateral sclerosis is an extremely chronic disease, and sets in very gradually. It has been divided into three stages. The *first stage*, that of *incomplete spastic paraplegia*, begins with a sense of weakness, heaviness, and stiffness in the legs, causing some difficulty in walking. This is sometimes preceded by pain in the back and limbs. On examination slight stiffness and rigidity in the legs may be discovered, with exaggeration of the deep reflexes. The symptoms gradually increase, until a characteristic "spastic" gait is developed. "The patient then walks with two sticks; each step is attended with evident effort; the feet appear to be stuck to the ground, and can only be moved forward by raising the pelvis, and with it the limb as a whole. In this process the back is strongly arched, the chest thrown forward, the patient leans forcibly, first on one stick and then on the other, and appears to aid the elevation of the trunk by movements of his arms. The toes are dragged along the ground with an unpleasant scraping noise, the knees are apt to interlock, and the foot which is being brought forward tends to cross in front of its fellow. In some cases, after the foot leaves the ground, a peculiar hopping movement of the whole body is observed. It is due, according to Erb, to spasmodic contraction of the calf muscles." (Byrom Bramwell.) The legs are generally kept close together, owing to spasmodic contraction of the adductors; and when standing thus, if the patient closes his eyes there is no increased unsteadiness or feeling of vertigo. At the same time the muscles of the lower extremities are affected with spasmodic twitchings, tremors, and rigidity. These phenomena are usually due to some reflex irritation, or to attempts to perform voluntary movements, but may occur spontaneously. The affected muscles are tense and rigid, especially on manipulation, and this or any external irritation may throw the whole limb into a state of tonic spasm. Their nutrition is unimpaired. Their reaction to electricity is usually normal, or somewhat decreased; but some writers state that it is increased. The deep reflexes are greatly exaggerated; ankle-clonus is easily elicited, and when the patient, sitting or standing, presses upon the balls of the toes, this reflex is sometimes spontaneously excited, causing a rhythmical tremor. A blow

upon one patellar tendon may cause a jerk of the opposite leg; and a knee-clonus is sometimes elicited. The superficial reflexes may be normal, increased, diminished, or abolished. The only sensory disturbance observed is increased sensibility to cold. The bladder and bowels are usually unaffected. Erb states that only one leg, or a leg and arm, may be occasionally involved; or still more rarely both arms are first affected.

In the *second stage*, that of *complete spastic paraplegia*, the power of locomotion becomes entirely lost, and the patient is confined to bed, the legs being rigidly extended, the thighs closely approximated on account of spasm of the adductors, and the feet inverted. The arms may ultimately become similarly affected.

The *third stage* results from the implication of the anterior cornua or postero-external columns. In the former case, the muscles slowly waste, while the rigidity decreases; the reflexes become less marked, and are finally abolished. Implication of the postero-external columns is indicated by lightning pains and signs of inco-ordination. Cystitis or bed-sores may ultimately supervene, with death from gradual exhaustion or pyæmia, but usually this event occurs from some intercurrent complaint, such as bronchitis or pneumonia.

#### IV. AMYOTROPHIC LATERAL SCLEROSIS.

**PATHOLOGY.**—This variety of sclerosis was first described by Charcot, and it presents the following distinctive pathological features:—1. In the great majority of cases it commences and is most marked in the cervical enlargement of the cord, and extends gradually downwards. 2. Though it begins in the lateral columns, it quickly spreads to the anterior cornua, involving and destroying their large motor ganglion-cells, so that there is a combined sclerosis of these portions of the cord. 3. The lesion almost always extends upwards also, involving the medulla oblongata, and sometimes passing through the foot of the cerebral peduncle; the internal capsule is usually intact. The nuclei of the facial, hypoglossal, and spinal accessory nerves are generally involved at the close. Exceptionally the change begins in the medulla oblongata, and extends downwards; or it may commence in the lower part of the cord and ascend. After the extension of the disease to the anterior cornua, the anterior roots of the nerves become gradually involved, and the muscles waste.

**SYMPTOMS.**—Three stages are described:—In the *first stage* the arms are affected, presenting weakness gradually increasing to actual paralysis, and soon accompanied with marked muscular atrophy of all the muscles of the arms, fibrillar twitchings, and tremors on movement. Rigidity and contractions also supervene, so that deformities are produced, the arms being fixed closely to the sides, the fore-arms semi-flexed and pronated, and the hands and fingers strongly flexed. In the *second stage*, which sets in in from four to twelve months, the legs become involved, while the symptoms increase in the arms. The lower limbs present at first the signs of spastic paraplegia gradually increasing; while there are no marked sensory disorders, and the bladder and rectum are unaffected. Subsequently the muscles waste; the reflexes diminish; rigidity and spasms gradually decrease; the reaction of degeneration is developed; and fibrillar twitchings occur. There are no bed-sores throughout. In the *third stage*, the upper part of the cord and the medulla oblongata become implicated, and signs of bulbar paralysis appear, involving the lips, tongue, palate, pharynx, and larynx.



The phrenic nerve usually becomes affected, and thus the diaphragm is interfered with. Amyotrophic lateral sclerosis is always fatal, and death generally occurs in from one to three years.

#### V. SECONDARY LATERAL SCLEROSIS—SECONDARY DESCENDING DEGENERATION.

**PATHOLOGY.**—Secondary degeneration of the crossed pyramidal tract follows some primary lesion, situated either in the brain or spinal cord, which severs the connection of its fibres with their trophic centres, namely, the multipolar nerve-cells of the cortex of the brain. This lesion may be of different kinds, such as hæmorrhage or softening, and from it a descending sclerosis proceeds. If it is seated in the brain, the sclerosis extends down along the crus cerebri, through the pons, into the anterior pyramid of the medulla, and along the decussation to the opposite side of the spinal cord, in which it passes down almost entirely along the lateral white column, the superficial portion of which, however, is not involved. In short, it affects the crossed pyramidal tract on the opposite side to the brain-lesion; while it also involves the direct pyramidal tract on the same side. The extent of the change becomes more and more confined in its limits, both relatively and actually, as it proceeds downwards. When the original disease is situated in the spinal cord, such as myelitis or gradual compression, the tracts involved in the degenerative process will differ according to circumstances. Thus a complete transverse lesion will cause degeneration of the direct and crossed pyramidal tracts on both sides; a unilateral transverse lesion will involve both tracts on the same side; and if either tract is alone implicated, the degeneration will be confined to it.

**SYMPTOMS.**—The supervention of secondary degeneration of the tracts of the cord just considered will be indicated clinically by spastic symptoms, muscular rigidity, and exaggerated deep reflexes, as in the other forms of sclerosis of these parts. It is recognized, however, by the fact that these phenomena usually follow, and are super-added to those clearly traceable to the original lesion. Thus, in the case of the spinal cord, rigidity follows distinct paraplegia, usually sensory as well as motor, while the bladder and rectum are affected, and there may be trophic lesions of the skin. These characters are not noticed in primary sclerosis, in which muscular weakness and rigidity advance together, the rigidity being usually in excess. Moreover, secondary degeneration is generally much more rapid in its progress; but, when very chronic, its symptoms are exceedingly like those of primary lateral sclerosis. As regards the brain, there is seldom much difficulty in diagnosis, there being hemiplegia and other characteristic symptoms, to which a condition of spastic hemiplegia is added. There are exceptional cases in which difficulty may arise, from the fact that the phenomena may be unilateral in connection with disease of the spinal cord, and thus a cerebral lesion may be simulated. In cerebral cases, however, there is generally a distinct history of the original lesion; brain-symptoms are usually present; the face and tongue are commonly involved; rigidity and paralysis are more marked in the arms; sensation is unaffected, or anæsthesia, if present, is on the same side as the motor paralysis; and superficial reflexes are diminished or abolished.

## VI. DISSEMINATED OR MULTIPLE SCLEROSIS.

**PATHOLOGY.**—The form of sclerosis thus denominated is also known by various other names, such as *insular sclerosis* (Moxon), *multifocal sclerosis*, *sclérose en plaques disséminées* (Charcot). It is characterized anatomically by the morbid condition being arranged in small roundish patches or nodules, scattered irregularly through the nerve-centres, and sometimes involving also the peripheral nerves. They may be found in different parts of the brain or spinal cord, either separately or together, often occupying several regions at the same time. According to the distribution of the nodules in the nerve-centres, disseminated sclerosis has been divided into three main types, namely:—1. *Spinal*; 2. *Cerebral*; 3. *Cerebro-spinal*, the last being the most common. In the cerebrum the nodules are seen chiefly in the corpus callosum, corpora striata, optic thalami, and septum lucidum; sometimes in the centrum ovale; very rarely in the grey matter of the convolutions. The corpus dentatum is almost the only part of the cerebellum which is involved. Sclerotic patches may also be found in the pons or medulla. In the spinal cord their distribution is extremely irregular. They are said to be mainly confined to the white column, but may involve the grey matter; and are usually indiscriminate, though sometimes symmetrical. The nerves may be studded with patches, or diseased throughout. The nodules are in most cases well-defined, and either project above the surrounding level, or are depressed. Charcot states that they may usually be divided into three zones, indicating successive phases of the disease, the innermost zone being most advanced. They vary in size from a pin's head to a bean or larger: and also considerably in number in different cases. They have a grey translucent appearance, and on exposure to air assume a pink colour. Patches of sclerosis rarely lead to secondary degenerations, either ascending or descending.

**SYMPTOMS.**—It will be readily understood that the precise clinical history of disseminated sclerosis must be variable in different cases, and the symptoms are often exceedingly complex. Charcot aptly calls the disease *polymorphous*. Its invasion is usually extremely gradual and chronic, but occasionally is more or less abrupt or sudden. The reason that any definite clinical history of multiple sclerosis can be given at all is, that it very frequently involves the lateral columns of the cord, the medulla, and the pons, and the associated symptoms are indicative of such a distribution of the lesion. Usually they point at the outset to the spinal cord, but in a certain proportion of cases the brain is first involved, as indicated by headache, vertigo, mental disorder, &c. In general terms they may be described as rhythmical tremors; slow and progressive paralysis, especially of the lower extremities; contraction of the limbs; peculiar vertigo, paroxysmal or almost constant; affections of the eyes; defect of speech, with tremors of the lips and tongue; and marked change in the expression and mental condition. In the course of the disease inco-ordination of the movements of the legs may arise; as well as wasting of certain voluntary muscles; and disorders of deglutition, respiration, and circulation.

For practical purposes it will be sufficient to give a brief account of a typical case of *cerebro-spinal sclerosis*. The clinical history has been divided into three stages. The *first stage* begins with motor disorder in one leg, usually of the nature of paresis, sometimes of ataxy. The

paresis gradually increases to actual paralysis, while the opposite leg becomes similarly affected, and then the arms one after the other. Sensation is either entirely normal, or there may be merely temporary numbness, or a sensation of "pins and needles." Soon a characteristic symptom is developed in the affected limbs, namely, a marked rhythmical jerking, tremor, or shaking, which, however, only occurs on voluntary effort, affecting those muscles or parts of a limb which are called into action, and immediately ceasing when they are at rest. The disordered movements are usually more marked in some parts than in others. They differ in degree and in exact character, giving rise accordingly to fine tremors or shakings, very marked trembling, or violent movements somewhat resembling those of chorea. As a rule the voluntary movements are not intensified when the eyes are closed, as in locomotor ataxia. In well-marked cases any voluntary action at once brings on the tremor, and this is in proportion to the effort made. Later the muscles of the trunk and neck become affected in the same way as those of the limbs, producing nodding of the head; and the condition extends to the face, lips, tongue, eyes, or sometimes to the palate, pharynx, and larynx. In well-marked cases the facial expression becomes dull, vacant, stolid, and stupid. Articulation is more or less affected, as evidenced by monotonous voice; a peculiar slow, hesitating, drawling, and measured speech, each syllable being separately pronounced—*scanning speech*; or a jerky articulation, ultimately becoming thick and blurred; or by the voice being weak or whispering. Deglutition is less frequently disturbed, but may be involved in advanced cases. In connection with the eye, the most common and characteristic symptom is nystagmus. Actual paralysis of the ocular muscles is rare. The pupils may be irregular, or sometimes "pin-pointed;" the "Argyll Robertson phenomenon" is rare. Dimness of vision is frequent, and may be unilateral; it may be due to nystagmus, to atrophy of the optic discs, or to changes in the visual centres. Diplopia is rare; and actual blindness very rare.

In order to observe the tremors in cases of multiple sclerosis, it is necessary to make the patient perform various acts. When he is quietly seated nothing may be observed, or at most only a slight rhythmical jerking of the head. Among the actions which have been particularly recommended to demonstrate the irregular movements are rising from a lying to a sitting or standing posture, elevation of a limb, attempts to grasp some object, drinking a glass of water, writing, putting out the tongue, and walking. In the earliest period there is merely slight unsteadiness when walking is attempted, the only thing noticed being some degree of stiffness in the way in which the neck is held, and slight jerking of the head. By degrees the gait becomes more and more unsteady; "in some cases it resembles that of locomotor ataxy; in others the inco-ordination chiefly affects the muscles of the trunk, and the patient does not walk deliberately in a straight line (as the subject of locomotor ataxy does), but is apt to shoot forcibly from side to side; in other cases again the feet seem to cling to the ground, and the spastic gait is conjoined with a rhythmical shaking tremor of the whole body" (Byrom Bramwell). The cause of the tremors is disputed. Charcot attributes them to irregular conduction through axis-cylinders lying in the midst of sclerosed tissues; others regard them as being due to the presence of sclerotic patches in the pons and parts of the brain in front of it.



The muscles which are involved in multiple sclerosis are not wasted ; and there is no change in their electric excitability. The deep reflexes seem to be, as a rule, distinctly exaggerated, and ankle-clonus may be readily obtained. The general condition is well maintained. The bowels are constipated, but the bladder is seldom interfered with, and there are no bed-sores.

With regard to cerebral symptoms, headache is common, and vertigo may be very pronounced. The mental faculties also become gradually more and more affected, as indicated by hebetude and mental obscurity, irritability and loss of self-control, failure of memory, or actual dementia. Other forms of mental derangement occasionally observed are a sub-acute maniacal condition; delusions of grandeur, as in general paralysis of the insane; or profound melancholy. Apoplectiform or epileptiform attacks occur in some cases from time to time. These attacks do not seem to be attended with any obvious lesions. The epileptiform convulsions are often limited to one side of the body, and may last only for a short time, or for hours or even days, with intermissions. The temperature rises in both forms of attack, and may reach  $104^{\circ}$  or even higher. A fatal result may ensue, but more commonly a temporary hemiplegia remains, which soon disappears; after each attack of this kind, however, the patient is left in a worse condition than previously.

In the *second stage* of cerebro-spinal sclerosis the patient becomes unable to walk or stand, the legs having become more and more paralyzed, and he may be entirely confined to his bed. Rigidity not uncommonly supervenes, the legs being closely drawn together as the patient lies in bed, and rigidly extended, this condition being generally exaggerated when any attempt at movement is made. At first it occurs only at times and in paroxysms, but subsequently becomes permanent. Less frequently the arms are affected in a similar manner to the legs, and they are sometimes rigidly extended and closely drawn to the sides of the trunk. The deep reflexes are now markedly exaggerated, the ankle-clonus being often easily excited, and when set up may cause movements in the opposite leg, or even in the whole body. The tremors are also greatly intensified, and any attempt at movement may cause a violent shaking of the entire frame. Exposure to cold, or various kinds of irritation of the skin, will also often set up a general tremor, which, however, may commonly be made to cease at once by forcible flexion of one of the great toes (Brown-Séquard).

In the *third stage* the patient becomes emaciated; some of the paralyzed muscles may be specially atrophied; the mind is more and more affected; bulbar symptoms arise; the bladder is involved, followed by cystitis and renal complications; and bed-sores may develop.

Multiple sclerosis is usually a very chronic disease, its average duration being from eight to ten years. Death may occur at any time from apoplectiform or epileptiform attacks; or at a late period from exhaustion, interference with respiratory or cardiac functions, pyæmia, inflammatory or other complications. The varieties in the clinical history of the disease not only depend on the parts of the nerve-centres which it may involve at its commencement or in its course, but also on the particular tracts of the cord which the morbid lesion implicates.

## VII. GENERAL DIAGNOSIS, PROGNOSIS, AND TREATMENT.

1. DIAGNOSIS.—The different forms of sclerosis are usually clearly indicated by their clinical history. They are essentially chronic affections, and each variety presents usually certain prominent symptoms, which have been sufficiently pointed out in their several descriptions. In the early stage *locomotor ataxy* is liable to be mistaken for rheumatism or neuralgia, on account of the pains; the gastric attacks are also apt to be regarded as merely dyspeptic, should they occur before the characteristic symptoms of locomotor ataxy are developed. This complaint should always be suspected when amblyopia and atrophy of the optic disc are present without any obvious cause. The abolition of the patellar reflex may afford much aid in diagnosis in the early stage of locomotor ataxy. When the disease is well-established, it is usually easily recognized. The conditions with which it is most likely to be confounded are cerebellar disease, and cerebro-spinal sclerosis. The different forms of *lateral sclerosis* have to be distinguished from each other, but the principal points in diagnosis have already been indicated. The *amyotrophic* variety somewhat resembles wasting palsy at first, but is much more rapid in its progress; the mode of onset and extension of the disease is different; and there is never any rigidity in progressive muscular atrophy. It may also be mistaken for pachymeningitis cervicalis hypertrophica. *Disseminated sclerosis* has to be distinguished from paralysis agitans; locomotor ataxy; cerebellar tumour; chorea; or tremor following hemiplegia due to cerebral hæmorrhage or any other cause.

2. PROGNOSIS.—This is always grave, but in some forms of sclerosis arrest of the disease or even improvement may be effected at an early period, by appropriate treatment. In advanced cases the prognosis is very unfavourable, and sooner or later a fatal issue must always be anticipated.

3. TREATMENT.—It is important in all varieties of sclerosis to maintain the general health by means of good diet, *tonics*, cod-liver oil, strychnine, and such remedies. If the patient cannot swallow, it may be desirable to introduce food by the stomach-pump or by means of enemata. Passive exercise in the open air may be of service, but walking is often injurious, and especially fatigue. Reflex sources of irritation must be avoided, as well as exposure to cold. If any syphilitic taint is suspected, iodide of potassium and bichloride of mercury should have a fair trial. Large doses of iodide of potassium have been found useful in the treatment of locomotor ataxy. Other drugs which have been employed are ergot, nitrate of silver, chloride of barium, and arsenic, but they are rarely of much service. Various baths have been resorted to, but are of questionable use. Warm baths relieve spasmodic rigidity; gaseous thermal waters have also been recommended; and a properly-conducted hydropathic treatment may be of service. Electricity may prove very useful, but this agent must be used judiciously and according to correct principles. In lateral sclerosis it has been recommended to pass the constant current perseveringly through the spinal cord; but no benefit, in fact rather the reverse, is to be obtained by local electrical treatment of the muscles. (Byrom Bramwell.) Galvanization of the cord is also employed in locomotor ataxy. Counter-irritation near the spinal column, by means of the actual cautery, has

been found useful in some cases of sclerosis of the cord. Symptoms must be treated as they arise, and this is often all that can be done. For the lightning pains in locomotor ataxy subcutaneous injection of morphia, full doses of bromide of potassium, cannabis indica, salicylate of soda in 20-grain doses, the constant current, and forcibly stretching the sciatic or other large nerves, have been chiefly recommended. Nerve-stretching does not seem to affect the course of the disease, as was at one time hoped. The gastric symptoms are relieved by morphia, bismuth, and pepsin. Paralysis, rigidity, vesical symptoms, bed-sores, and other complications must be treated on ordinary principles; and every attention must be paid to cleanliness and other hygienic conditions.

---

### CHAPTER XCIII.

#### ON CERTAIN SPECIAL NERVOUS DISEASES.

##### I. PROGRESSIVE MUSCULAR ATROPHY—WASTING PALSY— CRUVEILHIER'S PARALYSIS.

**ÆTIOLOGY AND PATHOLOGY.**—Wasting palsy has been attributed, pathologically to an atrophic and degenerative change beginning in the involved muscles themselves; in the anterior roots of the nerves supplying them; or in the spinal cord. The view which now seems to be generally held is that the primary lesion consists in a gradual destruction of the motor nerve-cells of the anterior cornua of the cord, which some regard as due to a chronic inflammation, others to a degeneration, the nerves and muscles being secondarily affected. The chief supposed *exciting causes* are exposure to cold and wet; a blow or fall on the neck or back, or traumatic injuries of peripheral parts; syphilis; excessive use, with consequent fatigue of the affected muscles; acute fevers; and lead-poisoning. The disease occurs by far most commonly in males; and usually in persons about 30 years of age, though it may be met with at any period from childhood to old age. In some cases it appears to be distinctly hereditary, or to affect several members of the same family.

**ANATOMICAL CHARACTERS.**—The affected muscles in progressive muscular atrophy are wasted more or less, pale and yellowish or fawn-coloured, and soft. The muscles are altered to a very variable degree, and one may be found quite destroyed while the next is unchanged, or healthy bundles of muscular tissue may be seen in the midst of the morbid materials. The upper portions of the muscles are usually most changed. Microscopically it is found that the interstitial connective tissue, and in some cases the fat, are increased; while the muscular fibres have usually undergone simple atrophy, but sometimes they present nuclear proliferation and fatty infiltration. The anterior roots of some spinal nerves and the sympathetic branches joining them have been found atrophied, the nerve-elements being replaced by a finely-granular tissue. The morbid changes in the cord are practically limited to the anterior cornua. The nerve-cells are found in all stages



of atrophy and degeneration; and in some instances this change is simple. In others dilatation and thickening of the blood-vessels have been noticed; and in some cases compound granular corpuscles and oil-globules have been present. The anterior columns and adjacent parts of the cord sometimes exhibit distinct sclerotic characters.

**SYMPTOMS.**—Wasting palsy sets in very insidiously. It usually begins in either shoulder or hand, especially the right, but gradually advances from its starting-point so as to invade other muscles, until finally every voluntary muscle in the body may be involved, except those of the eye-balls and eyelids, and the muscles of mastication. Exceptionally the muscles of the neck, trunk, legs, or face are first implicated. The atrophy seems to begin most frequently in the right hand, involving the thenar eminence, then the hypothenar, and then the interossei. The right deltoid is not uncommonly first affected. There is a failure of muscular power, corresponding in situation, extent, and degree to the wasting, and this may culminate in absolute helplessness, with inability to swallow, speak, or breathe, death then resulting from asphyxia. At the same time there are marked objective signs of the atrophy of the muscles, which are well seen about the shoulders and in the hands, the latter assuming the “claw-hand” shape, or “*main en griffe*,” characterized by deep depressions due to the wasting of the muscles, while the tendons stand out, and the fingers are drawn in towards the palm, being also pushed back; the ball of the thumb is much wasted; the shoulder is flattened or depressed; and the bony prominences seem to stand out. The claw-hand is due, as shown by Duchenne, to the special paralysis of the interossei and lumbricales, the extensors and flexors still retaining their power, at least partially. The tissues have a soft and flabby feel. The face assumes a vacant or idiotic expression when its muscles become affected. During the progress of the wasting the muscles present constant flickering or fibrillar movements, so long as any muscular tissue is left, which are more marked if the skin is exposed to cold or blown upon. The irritability and force of contraction under electricity become diminished in proportion to the waste of tissue, but there is no “reaction of degeneration.” The reflexes may be exaggerated at an early period of the disease, but soon become diminished, and are finally abolished. The temperature of the affected parts is reduced, and the patient is usually very sensitive to cold. The mind is unaffected to the last. Pains are not uncommon in the diseased parts, either myalgic or articular. There is never any loss of power over the bladder or rectum; and the heart is never implicated. Sexual functions are not involved; and there are no trophic skin-lesions. In some cases wasting palsy does not spread to the extent above described, but is arrested in its progress, the patient ultimately recovering, especially when it is due to fatigue of special muscles. Death generally results from extension of the disease to the medulla oblongata, when bulbar symptoms supervene; or to pulmonary complications, a slight bronchitis being very dangerous if the respiratory muscles are involved. Sometimes the fatal result arises from gradual exhaustion. The duration of wasting palsy is very variable.

**DIAGNOSIS.**—The conditions which are liable to be mistaken for progressive muscular atrophy are paralysis from local injury or from disease of a nerve; amyotrophic lateral sclerosis; chronic lead-poisoning; poliomyelitis anterior acuta and subacuta; and general paralysis of the insane. Attention to the history, symptoms, and mode of progress of the complaint will generally make the diagnosis clear.

**PROGNOSIS.**—Improvement can often be effected in wasting palsy by early treatment, but in advanced cases very little can be done, especially if the disease is extensive and rapid in its progress. The prognosis is more favourable when the complaint is due to fatigue; while it is worse if any hereditary tendency can be traced.

**TREATMENT.**—If wasting palsy has arisen from excessive use of certain muscles, these must be allowed to rest. Improvement of the general health is highly important, by means of nutritious diet, *tonics*, change of air, and gentle regular exercise. Arsenic, strychnine, iron, and nitrate of silver are the chief drugs employed; and iodide of potassium if there is any syphilitic taint. Warm or sulphur baths have been recommended, but cold baths should not be used. The chief local methods of treatment advocated are systematic friction, for which some simple liniment may be employed; passive motion; kneading; and electricity. The continuous and interrupted currents are both serviceable, and their persevering use proves often very beneficial. According to Duchenne, "the more a muscle is atrophied and its contractility diminished the longer it should be subjected to the stimulation, the more intense should be the current, and the more rapid its intermissions. When the sensibility is seen to return, it is prudent to diminish the intermissions and abate the intensity of the current." The constant current may also be passed through the affected region of the spinal cord. Pain may be subdued by warm fomentations or baths; or, if it is severe, by the hypodermic injection of morphia. Any hereditary tendency to wasting palsy must be recognized, fatigue of muscles, and exposure to cold or wet being especially avoided under such circumstances.

## II. GLOSSO-LABIO-LARYNGEAL PARALYSIS—BULBAR PARALYSIS.

**PATHOLOGY AND ÆTIOLOGY.**—Bulbar paralysis is in exceptional instances an acute or sudden affection, but it is chiefly known as a chronic form of disease, and is sometimes associated with progressive muscular atrophy. The lesion seems to be of the same nature as progressive muscular atrophy, and to begin in the nerve-elements, ultimately involving the nuclei of origin of the hypoglossal, facial, pneumogastric, and spinal accessory nerves, in the medulla oblongata and upper part of the spinal cord. The motor cells are atrophied and shrunken; their processes are lost; and the intermediate tissue is in a state of degeneration. Afterwards the morbid change implicates the roots, and may extend along the trunks of the nerves, the nerve-fibres being grey, translucent, and degenerated. It may also pass down the spinal cord to a variable extent. The muscles which are affected in this complaint may present a healthy aspect; or they are pale, atrophied, with fat between the fibres, which may further present granular degeneration. No definite causes for bulbar paralysis have been made out, but it has been attributed to mental emotion, exposure to cold and wet, and syphilis. It occurs in adults, and chiefly in females.

**SYMPTOMS.**—The chief clinical phenomena in bulbar paralysis depend upon paralysis of the muscles of the tongue, palate, and pharynx, and of the orbicularis oris. In course of time the larynx and respiratory muscles become involved. In the great majority of cases the tongue is first affected, which is indicated by some embarrassment of speech, and impaired articulation. Special difficulty is experienced in raising the

tip of the tongue to the roof of the mouth, or in bringing it against the upper teeth; hence words beginning with lingual and dental consonants give most trouble. The organ can still be protruded, though perhaps not quite normally. Then dysphagia is experienced, particularly as regards fluids, which are apt to pass into the larynx or through the posterior nares, causing much distress and danger. Consequently saliva accumulates in the mouth, assuming a viscid, glutinous, stringy character, and it flows out instead of being swallowed. Food collects between the gums and cheeks, because the tongue cannot remove it. When the orbicularis oris becomes involved the labial sounds are not properly pronounced; whistling is impossible; and in time the lips remain apart and cannot be closed, so that the teeth are exposed, and the patient presents a peculiar and most unpleasant aspect. Articulation and deglutition become ultimately impossible; the tongue remains at the bottom of the mouth as a sodden inert mass; and the patient has to be fed. General debility and wasting result, in consequence of the interference with nutrition. Subsequently the implication of the respiratory muscles leads to difficulty of breathing, and inability to cough; while, when the larynx is affected voice becomes almost absolutely lost. In some instances the tongue is obviously wasted, wrinkled, and furrowed; but even when it is apparently enlarged, this seems to be due to accumulation of fat, the muscular fibres being atrophied. The lips may also be of normal size or thinned. Electric irritability is usually scarcely altered in the affected muscles. The mind is generally clear to the last, but the emotions are easily excited. If the disease extends down the cord, muscular atrophy or paralysis, with or without rigidity, is observed in the corresponding muscles. Bulbar paralysis always proves fatal, death resulting either from gradual or sudden asphyxia; from exhaustion and asthenia usually; from interference with the cardiac action; or from some intercurrent complaint.

DIAGNOSIS.—*Labio-glosso-laryngeal paralysis* may be confounded with simple paralysis of the tongue; facial paralysis, especially double; general paralysis of the insane; or diphtheritic paralysis.

TREATMENT.—But little can be done for this disease. Electricity to the affected muscles has been employed, in the early stage, with some benefit. Symptoms must be attended to; and it becomes necessary in time to feed the patient by the stomach-pump or enemata.

### III. WRITER'S CRAMP—SCRIVENER'S PALSY—MOGIGRAPHIA.

ÆTIOLOGY AND PATHOLOGY.—The form of nervous disorder thus named is but the most common type of a group of diseases, in which sets of muscles habitually and frequently exercised for certain complicated actions become the seat of peculiar spasmodic movements. Writer's cramp occurs principally among those who write a great deal, such as teachers, merchants, and clerks; but derangements of a similar character are met with in connection with other occupations, such as amongst violinists, violoncello players, pianoforte players, telegraphists, watch-makers, composers, engravers, tailors, sempstresses, milkmaids, shoemakers, bricklayers, and nailsmiths. Over-work of the affected muscles seems to be an important element in the causation of the malady, and it is aggravated by mental worry and anxiety. Among writers the use of a steel-pen, the wearing of a tight coat-sleeve, and an inconvenient and



constrained attitude have been considered as *predisposing causes*. Writer's cramp never occurs under 30 years of age; and is far more frequent among males than females.

The *pathology* of writer's cramp is very doubtful, but the affection has been attributed to some morbid condition or a state of mal-nutrition of the nerve-centres governing the implicated muscles, with consequent diminution in nerve-force, or loss of co-ordinating power; to chronic fatigue of these muscles (Poore); to a reflex neurosis from muscular nerves; or to the transmission of voluntary impressions to other motor nerves besides those which are intended, sympathetic movements being thus excited.

**SYMPTOMS.**—The earliest symptom in most cases of writer's cramp is a sense of fatigue and aching in the hand after writing, especially in the thumb, as well as often in the muscles of the entire upper extremity. The patient finds that he must hold his pen more firmly, and give more direct mental attention to the act, in order to write properly. This only aggravates the mischief, however, and in time control over the muscles concerned in the act becomes diminished or lost, so that irregular spasmodic movements are excited in the fingers and thumb whenever any attempt at writing is made. The thumb may be convulsively flexed, the pen getting over its knuckle; the index-finger is jerked; or the first three fingers exhibit disorderly spasmodic movements. Of course the writing is more or less altered, and ultimately becomes mere illegible scribbling. The patient learns to alter his mode of writing, using the hand, wrist, elbow, and shoulder in succession, but as he does so the corresponding muscles present similar spasmodic movements. Then he takes to writing with the left hand, which becomes affected in the same manner.

The symptoms just mentioned are intensified by mental excitement, and by anxiety on the part of the patient to write properly. They cease immediately the attempt to perform this act is given up, and all other actions, however complicated and delicate, can be carried on without any difficulty or disorder. In some cases a dull pain is experienced in the muscles of the limb, or a feeling of weight and tightness, and tenderness over the nerve-trunks is often present; but ordinary sensation, electric irritability, and nutrition are in no way impaired. In exceptional cases headache, vertigo, mental dullness, occasional tremors, clonic spasms of some or of all the muscles of the arm, and other nervous symptoms are observed. The general health is usually good; but the patient is liable to be depressed in spirits, and may become actually melancholic. There are some cases in which the impairment of writing power does not depend on any excessive use of the muscles; these seem to be less severe, and Dr. Poore states that the trouble is then not so strictly limited to the act of writing. In the case of the other occupations mentioned, the peculiar character of the disorder will vary with the action which each habitually involves.

**DIAGNOSIS.**—There is but little difficulty in recognizing the class of diseases now under consideration, if attention is paid to the occupation of the patient, and the peculiar course of symptoms above described. Writer's cramp might possibly be mistaken for wasting palsy; or for the effects of chronic lead-poisoning.

**PROGNOSIS.**—If the condition has only existed for a short time, a cure may be expected under appropriate treatment; but in cases of long duration the prognosis is most unfavourable.

**TREATMENT.**—All attempts to check the progress of writer's cramp by using quill pens, altering the mode of writing, employing douches and friction, and such measures, are quite ineffectual, and an essential part of the treatment consists in absolute and prolonged rest from the particular employment which is the cause of the complaint, or, if possible, it should be given up altogether. The regular use of the continuous current has proved serviceable, applied to the muscles and nerves of the arm, and along the spine. Dr. Poore employs this agent along with voluntary rhythmical movements of the muscles. Subcutaneous injection of atropine has also been found useful. When nothing can be done in the way of improvement patients can sometimes manage to write by using some special apparatus, such as a pen which brings into use the extensors of the fingers, and not the flexors, as recommended by Nussbaum.

#### IV. PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS—DUCHENNE'S PARALYSIS.

**ÆTIOLOGY AND PATHOLOGY.**—The *exciting causes* of this peculiar affection are quite obscure. Pathologically it has been attributed by some writers to a lesion affecting the motor nerve-cells in the spinal cord, and has been considered to be a disease of the same nature as progressive muscular atrophy; others, however, regard it as a disease of the muscles. It commences almost always during early life, and is by far most common in boys. In exceptional cases the disease begins in adults. Hereditary predisposition is said to be present in a considerable number of cases, but the disease is almost exclusively transmitted through the female line, and it attacks several boys in one family. Dr. Gowers thinks that the complaint is more common among the better classes. It sometimes follows an acute febrile attack, such as scarlatina.

**ANATOMICAL CHARACTERS.**—The more obvious changes in Duchenne's paralysis are associated with those voluntary muscles which are affected, these being increased in size and very firm; while they present important structural alterations, the muscular fibres having to a great extent disappeared, many of those which remain being atrophied or in a state of degeneration, while the great mass of the apparently enlarged muscles is made up of fat and fibrous tissue, the latter being partly the remains of the sheaths of the muscular fibres, partly the result of increase of the interstitial tissue. In advanced cases other muscles are simply atrophied. Many eminent observers have been unable to detect any morbid condition of the spinal cord in pseudo-hypertrophic muscular paralysis; but marked changes have been described by Lockhart Clark and Gowers, and more recently by Drummond and Byrom Bramwell.

**SYMPTOMS.**—Pseudo-hypertrophic muscular paralysis is a very chronic disease, and its progress has been divided into certain stages, the duration of which presents much variation. The muscles first affected are those of the legs and back, especially those of the calves, back of the thighs and gluteal regions, and the erector spinæ. At the outset these are merely weak, the weakness being noticed when the child begins to walk. The legs are unsteady, movements are clumsy, and the child often stumbles or falls. Subsequently the muscles increase in size, and then the characteristic symptoms become evident. These are:—1. Enlargement and unnatural firmness of the calves, back of the thighs, and lumbar

region. 2. Peculiarities in the *attitude*. When standing the patient is evidently unsteady, and keeps the legs wide apart, with the heels raised; the shoulders are thrown back, and the antero-posterior curve of the spine is much exaggerated, so that the abdomen appears peculiarly prominent, but this prominence subsides in the sitting posture. In extreme cases a vertical line from between the shoulders falls behind the sacrum. The hands are extended by the sides, and are used to balance the body. The slightest push will make the patient fall. 3. Peculiarities affecting the *mode of progression and movements*. In walking the legs are also much separated; the patient supports himself almost on tiptoe; and the body is balanced, first on one leg then on the other, with a kind of waddling or oscillating movement. There is evident difficulty in bending the thigh and bringing the foot forwards. The advance made in each step is very small. The patient easily stumbles or falls, especially on attempting to walk rapidly, and soon becomes tired. Stooping is easily effected, but it is very difficult then to gain the erect posture, except when the patient is sitting down. He has great difficulty in raising himself from the recumbent or sitting position, and in advanced cases is quite unable to do so. When he has nothing to take hold of he goes through a series of characteristic movements when told to rise from the recumbent posture. When on "all-fours," he has first to get the legs straight, and then climb up his legs with his hands.

In course of time the muscles of the upper part of the trunk, of the arms, or even those of the face become involved. These may be also evidently enlarged, but more commonly wasting of the upper part of the body is observed, contrasting markedly with the enlargement of the lower portion. Gowers lays stress upon wasting of the latissimus dorsi and sterno-costal portion of the pectoralis major. The paralysis becomes more marked and extensive, and ultimately the patient lies in a completely helpless condition, while at the same time the muscles formerly hypertrophied undergo wasting. Contraction of the affected muscles occurs, producing talipes equinus, &c. The mental faculties may become impaired; and headache, disorders of vision, and other evidences of cerebral disturbance may be noticed before the close. Death results either from gradual exhaustion; from implication of the respiratory muscles or heart; or from some intercurrent malady, very often from bronchitis.

Very different statements have been made as to the electric contractility of the affected muscles in this complaint. It is probably usually impaired to the induced current, but has been found increased to the primary current. The reflex movements of the involved muscles are first impaired, and then abolished. The patella tendon reflex is abolished in advanced cases. During the active stage of the disease the temperature of the affected parts may be raised. Portions of the diseased tissues may be removed for examination during life, by means of Duchenne's or Leech's trochar.

**DIAGNOSIS.**—In a well-marked case there is no difficulty in recognizing pseudo-hypertrophic muscular paralysis. It might possibly be mistaken for true muscular hypertrophy; or for spinal disease.

**PROGNOSIS.**—This is usually very unfavourable. Instances of recovery in the early stage have been reported, and improvement has been effected in more advanced cases, but these are exceptions. The duration is very variable, but the complaint is usually exceedingly chronic; it is said to run a more rapid course in boys, and when it begins soon after birth.



**TREATMENT.**—No drug has any direct influence upon Duchenne's paralysis, but arsenic and phosphorus have been recommended. The only local measures from which any benefit can be expected are shampooing and kneading; cold douching; and the use of electricity. Local faradization of the affected muscles is of much use; and the application of the primary current along the spine and sympathetic nerve has also been recommended. The general health must be maintained by good food, fresh air, systematic exercise, and *tonics*, if required. The patient must be carefully protected against cold. He must be encouraged to walk as long as possible, and mechanical appliances and supports may help him to do this. Contractions and deformities must be prevented by passive movements, and by section of tendons, if necessary.

## V. PARALYSIS AGITANS—SHAKING PALSY.

**ÆTIOLOGY AND PATHOLOGY.**—Paralysis agitans must be regarded as a *functional* disease of the nerve-centres, for, although various organic lesions have been found, there are none having any definite relation to this complaint. It has been mainly attributed to violent emotion; long-continued anxiety or grief; exposure to cold and wet; continuous or severe exertion; injury of nerves; and exhausting diseases. Special varieties of the complaint have been described as *hysterical*, which occurs in hysterical persons; *reflex*, due to some reflex irritation (worms, wounds, &c.); and *toxic*, resulting from the action of some poison upon the system (mercury, alcohol, tobacco, tea, or coffee). True paralysis agitans is rarely met with under 40 years of age, and becomes progressively frequent as life advances. It seems to be more common in males than females.

**SYMPTOMS.**—Paralysis agitans is in the great majority of cases an insidious disease, but at the same time progressive. It is characterized chiefly by tremors of the limbs, which are independent of voluntary movements; muscular rigidity; and a tendency to impairment of equilibrium in walking. The complaint begins usually with irregular attacks of tremors, coming on without evident cause, of variable duration, and affecting the hand or foot or the thumb. They become gradually more frequent and severe, and also extend, until finally all the limbs are usually involved, and the tremors are constant. The head and neck remain as a rule entirely free from tremors, but if the lower limbs are affected, and especially on standing, the tremors involve the body. They are made up of fine and rapid oscillations; are subject to exacerbations, especially from mental excitement, fatigue, and other disturbing influences; can sometimes be checked temporarily by voluntary effort; are often very violent when the patient is in other respects perfectly at rest; and cease during sleep. A peculiar rigidity of the muscles usually follows the tremors, but occasionally precedes them; at first more or less intermittent, it subsequently becomes constant, and involves not only the muscles of the extremities, but also those of the trunk, and of the head and neck; the flexor muscles are most affected. The rigidity is often accompanied with cramp-like pains. The difficulty in maintaining equilibrium in walking is not wholly dependent upon the tremors and rigidity, being observed in some cases at a very early period.

In a well-marked case of paralysis agitans the appearance, attitude, and gait of the patient are highly characteristic. The limbs present a combination of tremors and rigidity. The thumbs are generally extended,

and the fingers flexed upon them ; while movements are carried on as if bread were being crumbled. The arms are held out slightly from the sides ; the wrists and elbow-joints are a little bent ; the hands are tilted towards the ulnar side, resting on the abdomen at or near the waist ; and the finger-joints are flexed or distorted. When the patient stands or walks the body is inclined forwards, the knees are slightly bent, and the ankles extended, so that he rests on his toes. The head and neck present a striking appearance, being thrown forwards, and rigidly fixed, while the features are motionless and devoid of expression. The patient rises from his seat with some difficulty, and hesitates before he begins to walk. He starts carefully, but his steps soon become short and rapid, and he cannot prevent himself from running forwards, and will probably fall if not prevented. In some cases the tendency is to run backwards. These movements may often be reversed or altered by a sudden pull at the clothes.

In paralysis agitans the involved muscles are generally the seat of a feeling of marked weariness, especially after a paroxysm of tremors, or after exertion. They are usually stronger than those which are unaffected. The patient becomes irritable and fidgety, and may experience an uncomfortable feeling of heat, especially in the epigastrium and back. There is no giddiness. Articulation becomes in time slow and difficult ; and the tongue may be tremulous. Deglutition also becomes affected in a similar manner. In some rare cases all the above symptoms are present, with the exception of tremor, which is absent.

Paralysis agitans is of slow and often irregular progress. Finally the patient is confined to his bed ; the muscles atrophy ; the tremors are usually extreme, but occasionally cease ; the mental faculties suffer ; and bed-sores may form. Death may result from asthenia ; or from some intercurrent affection, especially pneumonia.

DIAGNOSIS.—Paralysis agitans is most likely to be mistaken for multiple sclerosis and mercurial tremors. Due attention to the ætiology and symptoms will generally make the diagnosis quite clear.

PROGNOSIS.—Paralysis agitans must be regarded as practically incurable. Exceptional instances of recovery in the early stage have been recorded. It is, however, a disease of very slow progress.

TREATMENT.—The indications in the treatment of paralysis agitans are to remove the cause ; to give good diet, and attend to hygienic conditions ; to avoid fatigue and mental excitement ; to improve the general health and condition of the nervous system by strychnia, iron, phosphorus, arsenic, zinc, and similar remedies ; to administer *sedatives*, such as bromide of potassium, hyoscyamus, opium, conium, or cannabis indica ; and to apply the constant current along the affected muscles and over the spine. The systematic use of baths and friction has sometimes proved of service.

## VI. SPASMODIC WRY-NECK, OR TORTICOLLIS.

ÆTIOLOGY AND PATHOLOGY.—Wry-neck has already been alluded to as a form of muscular rheumatism. It may also be a congenital condition, due either to faulty development of the muscles of one side of the neck, or to paralysis, which may result from injury during parturition. The complaint now under consideration, however, is a peculiar nervous affection, attended with spasm of the muscles of one side of the neck,

especially the sterno-mastoid. It generally occurs in persons about middle life, who are apparently in good health. It is regarded as a neurosis, allied to writer's cramp and histrionic spasm, and is not associated with any definite lesion of the nervous system. It is in many instances due to an irritable state of the spinal accessory nerve. In some cases a weakened and fatigued condition of the muscles of one side of the neck seems to excite irregular contraction in their antagonists. Spasmodic wry-neck is sometimes associated with the hysterical state.

**SYMPTOMS.**—The spasm in torticollis is usually clonic, rarely tonic. The implication of the sterno-mastoid is indicated by turning of the head to the opposite side; while the occiput is drawn a little downwards, and the chin slightly raised. The trapezius, scaleni, and splenius being not uncommonly involved, a lateral downward bending is noticed, and elevation of the shoulder. The disorder is slight at first, but progressive, and may ultimately become extreme. The spasms are generally remittent; are increased by any mental exertion; and cease during sleep. The electric irritability of the affected muscles is usually greatly increased. The spasmodic movements may be so severe as to produce a sore on the shoulder. Dysphagia, and swelling of the arm from pressure on the subclavian vein, have been observed in exceptional instances. Spasmodic wry-neck is occasionally associated with spinal irritation, histrionic spasm, or spasm of the limbs; and it has also followed writer's cramp. The complaint is very chronic, but generally incurable after it has become well-marked.

**DIAGNOSIS.**—Spasmodic wry-neck is usually easily recognized. The diseases from which it has chiefly to be distinguished are caries of the spine, and some rare cases of cerebral disease.

**TREATMENT.**—The general treatment applicable to this class of affections must be carried out in cases of spasmodic wry-neck. Large doses of succus conii, and subcutaneous injection of morphia have been found useful by Dr. John Harley. Electricity in various forms has also been employed. Dr. Poore has used with success the continuous galvanic current, combined with rhythmical exercise of the affected muscles. In another case he faradized the antagonist muscles. Mechanical supports and surgical operations have also been resorted to, but without much success.

---

## CHAPTER XCIV.

### DISEASES OF THE SKIN.

**GENERAL CONSIDERATIONS.**—The term "skin disease" may be taken to apply to every deviation from the normal condition of the structure or functions of the skin and its appendages—the glands, hair, and nails. In the following pages, however, it is not intended to discuss those affections that have, by tradition and custom, been unreservedly given up to the charge of the surgeon, and for an account of these the reader may refer to surgical works. There are also many affections, for example, the acute specific diseases, in which a morbid state of the skin plays only a very unimportant rôle in comparison with other symptoms, and these



diseases have already been dealt with in another part of this work. The *study of skin diseases* has been in times past bewildering, partly on account of the paucity of workers in this field, and partly by reason of the infinite variety of form and aspect of diseased skin, so unlike, apparently, morbid changes in other structures, and this has led to a chaotic and overburdened nomenclature. Rapid strides have, however, been made in the direction of simplification, and there is no reason now-a-days why any one should fail to acquire a sound knowledge of the subject, provided a sufficient time is devoted to *practical acquaintance* with it. In the *character of the morbid processes* taking place in the skin there is nothing essentially special, beyond those differences of detail which arise from the situation of the skin on the exterior of the body, and from peculiarities of structure. The facts of general pathology therefore apply here.

*Lesions of the skin* are made manifest to our senses by alteration in its colour, by depression of the surface or elevation by new growths, by the exudation of cells and fluid from the blood-vessels, and by various other indications of the morbid processes. It is necessary at the outset to have a clear perception of the several types of the forms of cutaneous eruption (*erumpere*, to break out) or lesions of the skin. They are divided into *primary* and *secondary* lesions. The primary lesions are distinguished as maculæ, papulæ, tubercula, phymata, pomphi, vesiculæ, bullæ, and pustulæ. By the term *maculæ* is meant any alteration in the colour of a circumscribed area of the skin, whether accompanied or not by structural change, so long as there is not very marked elevation or depression. Tiny points are called *punctæ*, and widely diffused stains or blushes *discolorations*. *Maculæ* may be caused by simple staining or chemical changes, for example, from iodine or silver nitrate; by extravasations of blood, as in purpura and scurvy; by increased deposition of pigment, as in one phase of leprosy, in Addison's disease, in ephelides or freckles, in chloasma uterinum et gravidarum; by irregular deposition (both removal and deposition going on side by side), as in leucoderma; by the growth of vegetable parasites in the skin, as in *tinea versicolor*; by new growths or chronic inflammations of the corium, as in *nævi* or *morphæa*; or lastly, by slight congestions or superficial inflammations, as in erythema, and the erythematous phases of lupus, syphilis, and leprosy. A *papule* or *pimple* is a solid elevation of the skin, whatever may be its cause, whether from congestion about one or more follicles, as in *miliaria*; the accumulation of exudation from inflammation, as in *eczema papulosum*; new growth, as in *lupus*; overgrowth of the epidermis, as in *psoriasis* and some warts; new growths of blood- or lymph-vessels; or blocking or stuffing of the ducts or glands by epithelial *exuvæ* or secretion, as in *comedones* or *milium*. Papules vary in size from a pin's head (*miliary papules*) to a sixpence or more, as in syphilis; and they may be rounded (*papular erythema*), or quadrangular (*lichen planus*), conical, dome-shaped, disc-like (*nummular*), or flat (*lenticular*.) When the morbid process giving rise to the papule extends deeply, the term *tubercle* or *nodule* is usually applied, and hence the expressions *tubercular lupus*, *leprosy*, and *syphilis*; but it is not intended to convey the idea of any association with tuberculosis. *Phymata* or *tumours* are solid formations in the skin, exceeding a walnut in size, as in *erythema nodosum* and *fibroma molluscum*. They may be prominent or deep set, sessile or pedunculate. *Pomphi* or *wheals* are rounded or oval evanescent swell-

ings, accompanied by heat and tingling, due to the sudden and temporary dilatation of a bunch of vessels under nervous influence, causing transient acute hyperæmia and œdema of localized areas. They are typically seen in the effects of nettle-stings. The œdema is generally sufficient in the central parts to obscure the pink colour resulting from the hyperæmia, so that a wheal usually presents a white centre and a pink halo. Wheals may be of all sizes, from a split pea upwards, and sometimes cover very large and irregular tracts by confluence. *Vesicles* are small dome-shaped or acuminate elevations of the skin, formed by the accumulation of fluid, or by the dilatation and projection of lymph (varicose lymphatics) or blood-radicles (vesicular degeneration of nævi). The accumulated fluid is frequently inflammatory, as in eczema and herpes, and exudes from the blood-vessels to collect within and between the rete cells, or between the cuticle and rete (pemphigus). These inflammatory vesicles are often compound, and quickly rupture or collapse, and as they are frequently only an advanced stage of the inflammatory papule, so in their turn they may give place to the pustule. In sudamina the sweat passes out of the duct, to accumulate between the strata of the cuticle. The name *bulla* or *bleb* is applied when the collection of fluid is large, for example, greater than a split pea. Bullæ are formed in the same way as vesicles, and are specially characteristic of pemphigus, but may occur less prominently in a number of other affections, such as erysipelas, scabies, erythema, herpes iris, syphilis, leprosy, and also by the coalescence of vesicles in dysidrosis. *Pustules* are circumscribed inflammatory elevations of the surface, of moderate size, formed by the collection of pus in the skin. They usually have an inflamed areola, and frequently end in ulceration and scarring. A pustule may be such from the first, or be the late stage of a vesicle or papule, and has a similar mode of formation. Papules and vesicles in the strumous and in children have a strong tendency to this termination. The collection of pus may be superficial, as in pustular eczema; or deep-seated around a follicle or gland, as in acne and sycosis. The secondary lesions, namely, squamæ or scales, crustæ or crusts, ulcera or ulcers, cicatrices or scars, excoriations, and rimæ or fissures and chaps, are so called because they arise out of the primary lesions. *Squamæ* or *scales* are formed by the separation of an increased quantity of imperfect dry epidermic scales, and are a usual consequence of the interference with the nutrition of the skin produced by inflammation. Such desquamation may be very fine, or larger and branny (furfuraceous), or very large, as in pityriasis versicolor (membranaceous). *Crusts* differ from scales in being composed mostly of serous and cellular discharge, or blood which has escaped on the surface. They may also be formed of concreted sebum, as in seborrhœa, or of masses of fungus elements, as in favus. *Ulceration* results when the epithelial layers and more or less of the corium are destroyed or removed. It commonly follows suppurative inflammation, but may result also from necrosis of a new growth or gangrene. *Cicatrices* or *scars* are formed by the substitution of connective tissue, covered by a layer of epithelium, for the normal epidermis and dermis with its appendages, which have been destroyed. Scars are nearly always the consequence of ulceration, but occasionally result from a simple atrophic process. *Excoriations* are due to removal of the cuticle or more or less of the rete, generally by scratching or rubbing. Scarring does not follow, though a crust may form. *Primæ* or *chaps* or *fissures* are formed by the cleaving of the skin, which has lost its elas-

ticity and suppleness, from unusual dryness or inflammatory infiltration. In addition it may be mentioned that pigmentation and hæmorrhage are frequently secondary, the former resulting from continued hyperæmia, and the latter occurring sometimes after various kinds of primary lesions.

**ÆTIOLOGY.**—Diseases of the skin are due (1) to congenital aberrations of nutrition or imperfect development, for example, nævi, ichthyosis; (2) to external causes, or those acting from without directly on the skin; (3) to internal causes, or those acting from within, whether arising primarily in the economy or not; (4) to an innate disposition in the skin-tissues themselves to take on diseased action. Amongst *external* causes may be enumerated local irritants of all kinds, such as cold, heat, friction, scratching, pressure; the irritant juices of plants, such as the rhus family and the nettle; substances used or met with in handicrafts and occupations, such as lime, sugar, flour, soda, tar, paraffin, arsenic, oxalic acid, cyanide of potassium, and dyes; medicinal applications, as mustard, croton oil, turpentine, arnica, &c.; animal and vegetable parasites, as ticks, fleas, sand-fleas, bugs, and fungi; want of cleanliness or functional disease, leading to blocking and malnutrition of the follicles and glands. Many of these agencies only cause mischief when the system generally is debilitated and disordered, such as the skin-inflammation in bricklayers, washerwomen, grocers, &c. An *innate disposition* to take on diseased action, involving a plus or minus state of perversion of nutrition, may exist in a special structure, as the skin, apart from any general nutritional defect, as in rodent ulcer, warty growths, fibroma, keloid, and psoriasis probably. The skin disease may, however, be only a local manifestation of a general diathetic state, as in struma. This innate disposition is often hereditary. The chief *internal causes* are ( $\alpha$ ) the introduction of special poisons or harmful substances from without, as in some acute specific diseases, syphilis, and probably leprosy, in which skin-lesions are a prominent, but only a single symptom; ( $\beta$ ) the introduction of animal parasites through the stomach, probably in drinking water, as in elephantiasis arabum and guinea-worm disease; ( $\gamma$ ) the circulation in the blood of certain medicinal substances, as the iodide and bromide of potassium, copaiba, arsenic, which set up morbid changes under certain conditions not well understood as yet, though we know idiosyncrasy plays a great part; (9) the accumulation of morbid products from functional or organic disease of internal organs, which renders tissues prone to inflammation from any slight exciting cause, or actually themselves set up mischief, as in diabetes, gout, rheumatism; ( $\epsilon$ ) a general lowering of nutrition in which the skin participates, from over-work, starvation, depraved habits, climatic conditions, &c.; ( $\eta$ ) nerve-disturbance, either direct or reflex, and primary or secondary, by which the blood-supply to a part is disordered, as in urticaria, or morbid tissue-changes are set up, as in dystrophia and herpes, or the skin nutrition is lowered, so that morbid changes are easily excited. As a rule these causes do not operate in a solitary or individual way, but act in combination, so that it is of great practical importance to distinguish predisposing and exciting, producing and intensifying causes. Age, sex, and temperament have also an important influence.

**DIAGNOSIS.**—The method of examination in cases of skin-disease should be thorough and complete, both with regard to the history of the affection, and the character of the eruption in the several parts of the body. Some diseases with special and constant characters may no doubt be made out at a glance, but the habit of a partial inspection is very



prone to lead to error, since many skin-diseases present considerable differences of aspect, according to the site on which they occur, the intensity of the morbid process, and the stage of the affection, while some run through different stages, and the several stages of quite different diseases may closely simulate each other. Therefore the whole and not a portion only of the eruption must be examined, and the primary lesion must be sought out in its newest developments, as on the edge of a patch, and the transitional relationship of the stages made out. The history of the lesions and of the disease generally from its beginning, and the general course of the eruption up to the time of observation, must further be carefully traced. Any modifications or complications must also be distinguished from the primary mischief; and the diagnosis should be made both by the positive characters, and by the exclusion of other affections. Lastly, the observer must notice as of great value the extent and sites of the eruption; the degree of symmetry and method of grouping; the mode of extension, whether serpigiously or centrifugally, &c.; the existence of special subjective sensations, such as itching, pain, tingling, or of anæsthesia; the colour of the eruption; the formation of scars; and the age and sex of the patient.

CLASSIFICATION.—A great many attempts have been made to reduce skin-diseases to some orderly arrangement of groups and divisions, but in such a matter, where the causes and pathology of many affections are yet obscure, and where we are progressing from day to day, any classification at present must be unsatisfactory and temporary. Still some classification is very useful. We may classify skin-diseases, according to a prevalent custom, on the anatomical conditions found associated with certain symptoms, or according to their supposed ætiology; or we may arrange each disease around a chosen type, or “as an assemblage of symptoms conveniently arranged for diagnosis,” or according to a mixed plan. The following mixed plan, suggested by the late Dr. Tilbury Fox, is useful clinically:—

1. *The eruptions of the acute specific diseases*, already described.
2. *Inflammations*, comprising:—*a.* The *erythematous*, as erythema, roseola, urticaria. *b.* *Catarrhal*, as eczema, dermatitis. *c.* *Bullous*, as herpes, pemphigus. *d.* *Suppurative*, as ecthyma, impetigo contagiosa, furunculus. *e.* *Papular or plastic*, as lichen, prurigo. *f.* *Squamous*, as psoriasis, and pityriasis rubra.
3. *Diathetic affections*, due to some constitutional change or disposition, as struma, syphilis, leprosy.
4. *Hypertrophies*, as ichthyosis, corns, warts and papillary tumours, keloid, fibroma, scleroderma and morphœa.
5. *Atrophies*, as general or local atrophy of the skin or its appendages, senile decay, alopecia areata.
6. *New formations*, as lupus, rodent ulcer, cancer, melanotic sarcoma.
7. *Hæmorrhages*, as purpura (already described).
8. *Neuroses*, as hyperæsthesia, anæsthesia, pruritus.
9. *Pigmentary changes*.—*a.* *Increased*, moles, melasma, chloasma, lentigines. *b.* *Diminished*, albinism. *c.* *Irregular*, leucoderma.
10. *Parasitic diseases*.—*a.* *Dermatozoic*, as scabies, phthiriasis, guinea-worm disease, elephantiasis arabum. *b.* *Dermatophytic*, as tinea favosa, tinea trichophytina, tinea versicolor.
11. *Diseases of the glands and appendages*.—*a.* *Sweat-glands*, as miliaria (lichen tropicus), sudamina, dysidrosis, hyperidrosis, anidrosis, chromidrosis. *b.* *Sebaceous glands*, seborrhœa, comedo, acne, milium, molluscum

contagiosum, steatoma, lichen pilaris. *c. Hairs and their follicles*, sycosis, alopecia, calvities, fragilitas, hirsuties. *d. Nails*, atrophy, hypertrophy, and onychia.

### I. ERYTHEMATOUS INFLAMMATIONS.

These are characterized by hyperæmia and the exudation of serum and wandering cells, mostly into the papillary layer, but in severe cases to a greater depth. The inflammation is chiefly superficial, not very intense, tends to extend at the edge, is generally symmetrical, does not lead to deep suppuration, and after lasting a few days fades away, leaving perhaps some pigmentation and branny desquamation. The exudation is sufficient generally to cause more or less swelling, and in extreme cases raises the uppermost layers of epidermis so as to form large vesicles or bullæ. The following are the chief varieties:—

**Roseola** is a term applied in a very indiscriminate way to any non-contagious, fugitive erythematous blotches, arising in young children (*R. infantilis*), associated with gastro-intestinal disturbance and dentition, but apart from this it is a definite affection, presenting, however, much diversity of appearance. It may be *symptomatic*, as in rheumatism, vaccinia, (about seventh day), cholera, variola, and cerebro-spinal meningitis; or *idiopathic*, and it then seems to be mostly excited by climatic changes. Idiopathic roseola is characterized by a *rosy* eruption, mostly affecting the trunk, but in very marked cases extending to the limbs. It may be scarlatiniform or morbilliform, but is always coarser than scarlatina, and not crescentic or mulberry-coloured. In other cases the eruption occurs as well-marked rosy macules, or very slightly marked papules, which extend at their edges, form rings which coalesce, often assume an urticarial character, and extend over the whole body. It is very fugitive. There is hardly any general disturbance; occasionally very slight coryza, and injection of the eyes and throat; but never any marked ill-health.

**Erythema multiforme** is often ushered in by general malaise, rheumatic symptoms, and febrile reaction; and is characterized by the outbreak of circumscribed erythematous eruptions of various sizes and degrees of swelling, from a mere blush (*E. simplex*) to a papule (*E. papulatum*), or nodosity (*E. tuberculatum* or *nodosum*). The macular eruptions especially tend to enlarge at their periphery, and to clear in the centre to form rings (*E. annulare*), and these rings may coalesce to form patterns (*E. gyratum*). The eruptions also, though generally discrete, may be grouped in rings; and lastly the exudation may be so excessive as to form vesicles and bullæ on the surface (*vesicating erythema*—*herpes iris*). A feeling of heat, pain, or itching may be present. The eruption usually lasts only a few days, and then declines, but occasionally it is far more chronic, and may recur apparently under the influence of the seasons. The exact constitutional disturbance present is not known, but frequently appears to be of a rheumatic nature. As the eruptions subside, a peculiarity of these erythematous inflammations becomes apparent, in that the pigment resulting from the disintegration of exuded red blood-corpuscles goes through a change of colours. The sites affected, which tend to be symmetrically placed, are the extensor aspects of the hands, the forearms and legs, the face, and occasionally the trunk and other parts.

**Erythema nodosum** is included by some under erythema multiforme, but as it presents some constant peculiarities it will be well to describe

it separately. In this affection the constitutional symptoms are much the same as in erythema multiforme, but it rarely recurs, and tends to affect children and young adults, especially females. The painful ovoid swellings, ranging from  $\frac{1}{4}$  to  $1\frac{1}{2}$  inch across, are arranged along the tibiæ, with their long axes parallel to the bone. They evolve in crops, and the affection lasts one or two weeks. From the occasional distribution of the eruption along the lymphatics, and the circumstance that the latter vessels are sometimes implicated in the neighbourhood, it has been thought that the ovoid swellings are due to lymphatic inflammation, whilst others consider them to arise from capillary emboli.

**Peliosis rheumatica**, called also *purpura rheumatica*, is a very peculiar affection, which may be mentioned here, though its exact nature is as yet not defined. Accompanied by some malaise, swelling or pain in some of the large joints occurs (? rheumatic), and with its subsidence crops of a peculiar eruption appear, mostly on the limbs. This occurs time after time, perhaps each afternoon, and thus the affection may be kept up for a considerable period, and a person may have a recurrence after a long interval. The peculiarity of the eruption is that some is purpuric, and some consists of erythematous papules, or it is mixed.

**Urticaria** is an eruption with very special attributes, which is usually included under the erythematous inflammations, though with doubtful propriety, as it seems to be more a hyperæmia with serous exudation than a true inflammation. The wheals so characteristic of it, and typified in the effects on the human skin of the common stinging-nettle, result from acute oedema of the cutis, probably from the sudden complete dilatation of a bunch of capillaries. The exudation may be so little that only a rosy circumscribed elevation is produced, or it may collect in quantity sufficient to mask the central portion of the hyperæmia, so that a white centre is seen, with a pink halo. In rare cases a bulla is produced. The wheal is intensely itchy and tingling; and a striking characteristic is its sudden evolution, and rapid disappearance as the vessels regain their tone. The temporary paresis is brought about either by local irritation, or reflexly from internal functional disturbance, especially of the gastro-intestinal canal. In either case, with the exception of the effects of some special irritants, as the nettle, it is probable that there is a peculiar idiosyncrasy of the spinal nervous system. Wheals vary in area from a split pea to the palm of the hand or larger. Usually only the papillary layer is involved, but occasionally the deeper cutis and even the subcutaneous connective tissue may be affected. Urticaria may be acute or chronic. The acute forms are mostly due to the ingestion of some article of diet, such as mussels, tinned fish or meat, or lobster; and it is remarkable that some persons cannot partake of a certain article of diet, harmless to the generality of people, without inducing urticaria. In acute attacks the fever may run high, and the depression be great. Chronic urticaria is very common, especially in women, being almost invariably associated with gastro-intestinal disorder, and occasionally with uterine disturbance. Now and then chronic urticaria is distinctly neurotic, and recurs with much persistency at certain periods. There are two varieties seen in children to be mentioned, namely, the common *urticaria papulosa* or *lichen urticatus*, and *urticaria pigmentosa*. *U. papulosa* is so called because in infants and young children the wheals on subsidence leave little papules behind them, the size of a pin's head. From the fact that a multiform eruption may exist, consisting of wheals, papules, excoriations, and superficial inflammations, scabies is closely



simulated. *U. pigmentosa* is a very rare affection, commencing in early infancy, and characterized by two things, namely, the long persistence of the wheals, and the rapid deposition of pigment, so that, even after the wheals have gone, dark stains remain for months or years. The successive evolution of wheals may continue the affection for many years.

**Erythema pernio** is the name given to the condition known as a chilblain, which may occur on the hands and feet, nose, ears, and other parts. The exudation and inflammation in chilblains are induced by the action of cold on parts distant from the heart, in persons with a bad circulation. The redness that is sometimes seen on the hot, tense, fissured skin of œdematous legs is termed **Erythema læve**.

**TREATMENT.**—Roseola soon disappears spontaneously, but any derangement of the system must be remedied, special attention being paid to the gastro-intestinal functions. Locally a soothing and astringent lotion of calamine, oxide of zinc, subacetate of lead, or carbonate or bicarbonate of soda may be applied. *Erythema multiforme* and *nodosum*, and *peliosis rheumatica* require a cool regimen, careful dieting, rest, and tonic aperient medicines, such as quinine with sulphate of iron, sulphuric acid, and sulphate of magnesia. If rheumatic symptoms are marked, anti-rheumatic remedies must of course be exhibited. Locally the above-mentioned lotions are also useful, and in *E. nodosum* hot or belladonna fomentations. Acute urticaria requires the removal, if possible, by an emetic of the injurious article of diet, a purge, and then sedative remedies for the stomach, as bismuth with hydrocyanic acid, or a febrifuge. In chronic urticaria any dyspepsia, uterine disturbance, or plethoric condition must be remedied; and then the general health, and especially the nervous system, be braced up by mineral acids, bitters, ferruginous tonics, strychnia, phosphoric acid, and similar drugs. Locally in urticaria soda lotions are, perhaps, the most useful. In chilblains local stimulation to promote good circulation is indicated.

## II. CATARRHAL INFLAMMATIONS.

This type of inflammation is the analogue of the catarrh of mucous membranes, and is characterized by the infiltration into the skin-textures of migratory cells and inflammatory serous exudation, which stiffens and stains linen, and if it escapes on the surface, and is not so abundant as to run away, it forms crusts. This exudation may be so slight that no visible signs of its presence are produced; or it may simply cause a congestive redness, with subsequent desquamation, simulating erythema; or it may project on the surface as papules or vesicles; or escape copiously from the abraded skin; or deeply infiltrate the parts, causing much swelling, especially where the connective tissue is loose. The exudation may further become purulent, and form pustules and thick crusts. Any one of these stages may form the predominant feature, or a mixture of them may be present. Burning heat in the acute stages, and itching in the chronic, are striking characteristics. It is very necessary to understand how the eruption comes to be so frequently multi-form in character, and how it may assume an erythematous aspect at one time, a papular, vesicular, or pustular and crusted condition at later stages, and finally an erythematous and desquamating state in its decline. Two phases of catarrhal inflammation may be described.

**Eczema** is a very common catarrhal inflammation, with the characters mentioned above. We may consider it as a special disease with

important constitutional relations. The slightest form, that of congestive redness, is called *E. erythematosum*, and is rare as the sole condition present, but occasionally attacks the head and neck. Soft, red papules, the size of millet seeds (*E. papulosum*), are frequently met with, and this condition must be distinguished from the lichens and prurigo, which are essentially papular diseases throughout their course. Many of the papules may subsequently become vesicles, or the latter may stud a surface from the outset (*E. vesiculosum*). The contents of these vesicles in children and in strumous subjects become puriform (*E. pustulosum*), and then crusts form. The term *impetigo* was formerly applied to this condition. Frequently the fluid is sufficiently copious to abrade the cuticle, and run from the surface, for instance, about the legs (*E. madidans vel rubrum*). The decline of these stages is marked by an infiltrated reddened scaly skin, called *E. squamosum*; and the loss of elasticity allows the movements of the part to tear open cracks or fissures (*E. rimosum*). The latter state of things is that generally assumed in the palms and soles, where papules and vesicles are rarely formed. Lastly, in very chronic eczemas a warty outgrowth may supervene (*E. verrucosum*). Eczema may be acute, sub-acute, or chronic. Acute eczema is not very common, and when attacking the face may simulate erysipelas, from the swelling of the lax tissues, and the intense burning sensation accompanying the evolution of vesicles, and the attack may subside, or become chronic or recur. In sub-acute eczema the inflammation is less intense, and papules are seen mixed with the vesicles. Chronicity is a prominent feature of eczema in most cases, and the patches do not extend at the periphery like erythematous inflammation, but the papules and vesicles tend to evolve repeatedly over the same area. It occurs at all ages, and attacks all classes. It may be localized to a single patch, or be widespread, and it tends to evolve symmetrically on the body. In infants and young children the scalp and face are the regions nearly always attacked, though the inflammation may be more extensive, and, as already mentioned, the exudation is usually puriform. In adults the face and extensor aspects of the limbs are frequently involved together, and in another phase the large flexures. The squamous form is met with on the poll of women, and on the palms and soles. Occasionally the disease is very widespread, and the genitals are not unfrequently attacked. The constitutional origin of eczema is not yet placed on a sound footing, but the lowering of the nutrition of the skin leading to it is always associated with some form of debility referable to one of three conditions, namely, ( $\alpha$ ) a continued imperfect assimilation and elaboration and sub-oxidation of the food, with imperfect removal of effete products, leading up to the gouty state; ( $\beta$ ) neurasthenia, as seen in people of nervous temperament who are prostrated by nervous strain; and ( $\gamma$ ) perhaps struma.

**Dermatitis** is a term applied to catarrhal inflammations of the skin, other than true eczema. They are indistinguishable to the eye from eczema generally, but have a different causation, and are no more to be considered eczema than all the serous effusions in the knee-joint are to be put down as rheumatism. Thus dermatitis may be set up by croton oil, mustard, chrysophanic acid, many plants, as the rhus or thapsia, poisonous dyes, as aniline, and other irritants; the rubbing together of moist secreting folds of skin about the mammae, axillæ, abdomen, or nates; the action of the solar rays, and so on. The itch insect also excites an inflammation indistinguishable in the main from eczema, but this will

be considered under parasitic diseases. In bakers, grocers, bricklayers, and washerwomen the substances they respectively handle set up a similar inflammation. These conditions usually cease with the removal of the cause; but it is, on the other hand, apparent that for these noxious agents to excite an inflammation it is first necessary that the nutrition of the skin should be lowered by internal functional disturbance. In this place, also, it will be convenient to mention under the term *dermatitis medicamentosa* the inflammations of the skin excited in people of special idiosyncrasies by the ingestion of certain drugs. Many of these eruptions are very definite and constant, and recur each time the drug is exhibited. *Arsenic* is said to induce a brown pigmentation, urticaria, and erythematous blotches; *belladonna*, *stramonium*, and *hyoscyamus* a more or less widespread scarlatiniform or morbilliform rash. The *bromides* excite acneiform eruptions, which may be confluent and crustitial, or ulcerative; and also furuncles. *Chloral* may bring about a widespread scarlatiniform or morbilliform rash, accompanied by much dyspnoea and palpitation, and immediately excited by a meal or alcohol. There is not usually any fever. *Copaiba* excites a coarse erythematous eruption; often with an urticarial element about it. The *iodides* produce an acneiform eruption, rarely confluent, furuncles, bullæ, ecthymatous pustules, petechial purpuric spots, and urticaria; *opium* and *morphia*, a scarlatiniform rash; *quinine*, a rather coarser erythematous eruption, accompanied by considerable general disturbance; and *salicylic acid* produces a similar effect.

**TREATMENT.**—In dermatitis of local or internal origin the obvious indication is to remove the cause. In eczema a careful investigation should be made for the existence of any imperfect assimilation of food, associated with imperfect digestion and elaboration, plethora of the chylo-poietic viscera, and deficient excretion. The urine must be searched for evidence on these points, the bowels attended to, and then directions given as to irregularities in the quantity and quality of the diet; or the existence of any debilitating influence, such as sedentary habits or anxiety, must be sought for and remedied. Diuretics are useful to relieve the congested skin, and purgatives at the outset of an attack, but the latter must not be long continued and overdone. Alkaline remedies are needed where the urine is high-coloured and loaded with lithates; and in gouty conditions colchicum is desirable for a time. When the functions are in good order the system must be toned up by quinine, strychnine, phosphorus, ferruginous tonics, and cod-liver oil. Arsenic is very useful in many cases, especially in the chronic scaly forms, but it should not be given in the acute stages. In selecting a local remedy we must recognise the stage and intensity of the inflammation, and clearly understand what is required. Thus in acute and subacute stages we must soothe. If there is much weeping, we may dry up the discharge by absorbent, astringent, and antiseptic dusting powders, such as starch and oxide of zinc, with a little finely powdered salicylic or boracic acid. If the weeping does not trouble us, we may dab on a calamine and oxide of zinc lotion; or if that is too drying, and consequently not soothing enough, we may use benzoated oxide of zinc, or oleate of zinc, or bismuth ointment. Even in chronic stages we must always keep the skin supple. According to the amount of the infiltration, and the chronicity, we may select a resolvent and stimulant remedy, such as a mild mercurial salve, carbolic ointment or oil, or tarry applications; or in inveterate cases chrysophanic acid ointment,



or liquor potassæ applications. The irritation present often taxes all our resources, and we must alternately soothe, resolve the infiltration, and lubricate the skin. Scratching must be prevented, especially in children, by masking the parts and padding the hands.

### III. BULLOUS INFLAMMATIONS.

In this group are placed those diseases which are characterized by a superficial inflammation of the skin, and the rapid exudation of a quantity of serum, which quickly collects on the surface in large vesicles and blebs or bullæ. The contents of the bullæ are usually serous, but may become puriform or sanguinolent, and then crusting results, and more or less ulceration may occur as an exceptional circumstance. The bullæ themselves are short-lived, and quickly collapse or rupture. They may be of all sizes, from that of an eczema vesicle to that of the palm of the hand, and as a rule they rise directly from the surface without any antecedent papule or swelling. There is not the same general infiltration of the skin as in eczema, and usually not the same irritation. Moreover, facts point rather to a neurotic causation for these diseases, and the evidence is very strong at any rate for one member, namely, herpes. It must, however, be borne in mind that we exclude from this group some other inflammations giving rise to bullæ, for instance, the extreme forms of erythema, itch, and syphilis.

**Herpes** is an acute non-contagious disease, running a definite course, in which groups of vesicles, rather larger than those of eczema, appear on a congested patch of skin, one or more inches in diameter. There are usually several of such patches which evolve successively. Two main varieties exist, namely, *H. facialis et preputialis*, and *H. zoster*. Herpes facialis is a symptomatic eruption, which has a great tendency to recur in persons of a nervous temperament, about the mouth, face, and ears, when the system is febrile, or in any way upset by a cold, dyspepsia, and so on. The hard and soft palates may be involved, and the nasal mucous membrane, and this is more especially the case in the rare affection known as *H. iris* of the back of the hands, ankles, and knees, which is probably only an outlying member of the vesicating erythemata. It is also met with, and is then regarded as a favourable prognostic sign, in pneumonia. Similarly *H. preputialis* recurs about the penis in gouty and neurotic subjects, and may be confounded with a chancre. Herpes zoster, or *the shingles*, is the variety which is more distinctly associated with nerve-distribution, for the patches of vesicles evolve, for instance, around one side of the trunk, in the course of distribution of an intercostal nerve (*H. zoster intercostalis*). Though most common in this situation, it may occur in association with any nerve, as over the distribution of the first division of the fifth (*H. zoster ophthalmicus*), and then may be associated with kerato-iritis, conjunctivitis, and other lesions. Several nerves, generally those in juxtaposition, may be involved simultaneously. The eruption is often ushered in by malaise and some pain; it runs a definite course of ten days to several weeks; and disappears spontaneously, but is apt in old people to leave a neuralgia. It differs from *H. facialis et preputialis* in that it very rarely recurs. It is met with at all ages, equally on either side of the body, and is said to be more frequent in spring and autumn. The evidence for its dependence on nerve-irritation is very strong, namely, its distribution; its occasional complication with disordered sensation, paresis, or amyotrophy; and the demonstration *post-mortem* of congestion and neuritis, sometimes in the ganglia, sometimes in the periphery of the nerve be-

yond the spinal ganglia, and also in the posterior spinal roots. It is commonly an inflammation of peripheric origin, arising either from chill or some not well-ascertained cause, or from pressure or irritation by an aneurism, carious vertebrae, or a tumour. Now and then it occurs in cerebral affections; and rather more frequently in such cord-lesions as myelitis and locomotor ataxy.

**TREATMENT.**—All that is needed is to dust the parts with starch and zinc powder, or to prevent any ulceration and consequent scarring by a little zinc ointment, whilst the parts are protected from rubbing by cotton wool or spongiopiline. Some practitioners attempt to abort the eruption by such drugs as phosphide of zinc. The neuralgia left after herpes is sometimes very obstinate, and if local sedatives prove futile, hypodermic morphia injections are occasionally necessary. Quinine and other tonics are often of service.

**Pemphigus** is a somewhat rare, non-contagious disease, mostly of a definite character, characterized by the formation of isolated bullae arising rapidly on the skin, generally without any antecedent congestive swelling. The blebs are mostly dome-shaped and tense, and after one or more days collapse, and the wall of the bulla separates as a scale. The contents are usually clear at first, but may become opaque and puriform, or sanguinolent. In size the bullae vary greatly, even in the same patient, from that of an eczema vesicle to a pigeon's or hen's egg, but the tiny vesicles are rare, and the bullae commonly range from a pea to a pigeon's egg in size. They may evolve singly (*P. solitarius*) or in successive crops, and then there may be a certain periodicity observed; and in distribution they may recur about a certain region or be disseminated widely, even on the buccal and vaginal mucous membrane, and irregularly or in corymbose groups or crescents. In some rare cases of universal development the bullae tend to be abortive and coalesce, and there is a marked exfoliation of scales (*P. foliaceus*). As a rule there is not a great deal of local irritation, but in some rare chronic forms with small eruption, the bullae arise from and alternate with intensely itchy papules (*P. pruriginosus*). The affection occurs at all ages. The general symptoms are often little marked, but occasionally there is febrile disturbance, with evident debility and anæmia, and the disease may recur with some marked condition, such as pregnancy. In chronic cases there is a general wasting and prostration, and a fatal termination may ensue. The precise causes of pemphigus are obscure, but some evidence is forthcoming in support of the generally received opinion of its neurotic causation. No gross lesions have, however, been found in the nervous system, excepting, it is said, some parenchymatous neuritis of the nerves in immediate relation to the bullae. Some difference of opinion exists as to the occurrence of an acute pemphigus, and though now and again a case may prove more or less rapidly fatal, the great majority of cases are very chronic. As a rule but little difficulty can exist in the diagnosis, but it should be remembered that bullae occur in erysipelas, the vesicating form of erythema, from the use of iodide of potassium, and in hereditary infantile syphilis.

**TREATMENT.**—Locally the bullae should be punctured, and a starch and zinc powder or zinc ointment be applied to the excoriated surface. In very widespread cases a more or less continuous bath is required, or, at any rate, a thorough lubrication with some bland antiseptic oily lotion. Internally arsenic acts like a charm in a great many cases, though not in all; where debility or cachexia exists ferruginous and mineral acid tonics, cod-liver oil, and similar remedies are called for.

## IV. PUSTULAR INFLAMMATIONS.

Pustular skin-diseases include those in which the inflammation proceeds to visible pus-formation as an essential phenomenon. We have already seen that in eczema the exuded fluid may produce a vesicle, which *may* become puriform by the accumulation of pus-cells, and, indeed, all inflammations may be attended with a certain degree of pus-formation. It is to these pustular phases of eczema and dermatitis that the old term *impetigo*, or *eczema impetiginodes*, has been applied. We may regard these affections as essentially suppurative inflammation.

**Impetigo contagiosa** (Tilbury Fox) is an affection especially prone to attack children, though it may occur in adults brought into contact with such children, and subject to the same influences. It is usually sporadic, but occasionally apparently epidemic, in schools, streets, or houses. The eruption consists at first of a number of discrete vesicopustules, from a split pea to a fourpenny-piece in size, rapidly becoming more purulent and often confluent, and in about a week or ten days, if not scratched, drying up into flat yellowish crusts, looking as if stuck on. The eruption evolves spontaneously in crops or by auto-inoculation, and the affection may last several weeks, but always seems to disappear of its own accord. There may be some antecedent febrile reaction and malaise, but it is usually little marked. The site specially attacked is the face, but from this part the eruption may extend about the ears, scalp, hands, mucous membrane of mouth and eyes, and occasionally is widespread over the body, something like varicella. The inoculability of this affection has been ascribed to vegetable organisms, but it must be remembered that many kinds of pus when inoculated give rise to pustules; for example, the pustular inflammation set up by pediculi in the head, the purulent catarrh of the ears and nose of children, or the pus of the vaccine eruption, and such eruption must be carefully eliminated in making the diagnosis. Such a compound group has been denoted as *porrigo* or *impetigo contagiosa* at the Blackfriars Skin Hospital.

**TREATMENT.**—All that is required is to cleanse away the crusts, and anoint the sore places with the astringent weak ammoniated mercury ointment. The eruption then rapidly dies out.

**Ecthyma** is characterized by the evolution of discrete broad flat pustules, the size of a fourpenny-piece to a shilling, seated on a reddened, elevated, infiltrated base. The inflammation is superficial, and but rarely leaves scars. The contents dry into brownish crusts, which fall off in from ten to fourteen days, and the red stains disclosed gradually die away. A *primary* or *idiopathic* ecthyma is not very common, but is occasionally seen in children and some cachectic adults. The legs are the parts specially affected. There is usually more or less debility; and the course of the complaint tends to be chronic. *Secondary* ecthymatous pustules are more common, and occur in syphilis, leaving scars and stains, in scabies, urticaria papulosa, and phtheiriasis, and these causes must be eliminated in making the diagnosis of idiopathic ecthyma.

**TREATMENT.**—The scabs must be removed, and the sores dressed with an astringent ointment, that of acetate or carbonate of lead, or zinc. Occasionally in cachectic cases, where the reparative process is sluggish, a mild iodoform or balsam of peru salve is useful. Internally, good food, tonics, and cod-liver oil are necessary. The treatment of the several forms of secondary ecthyma is discussed in their respective sections.



## V. PAPULAR INFLAMMATIONS.

In the diseases included under this heading the inflammation is of a chronic character, and the eruption preserves the papular form throughout, with the exception that in prurigo there is often some attempt at pustulation. *Prurigo* and various forms of *lichen* are the diseases included here. It is necessary to say an explanatory word with regard to the term *lichen*. This was formerly applied indiscriminately to a variety of cutaneous diseases, inflammatory and of other kinds, in which the papule was the predominating feature. In the more orderly arrangement adopted at present these affections have been relegated to their proper positions. Thus *L. agrius* and *L. simplex* are now described as papular eczema, *L. tropicus* as miliaria, *L. urticatus* as urticaria papulosa, *L. pilaris* as keratosis pilaris. There remain three chronic inflammations to which the term is still applied.

**Lichen circumscriptus vel circinatus** is described as characterized by the formation of persistent, somewhat itchy, small papules, which evolve in round groups or rings. These rings have a red marked border and clear or stained centre, they spread centrifugally, and may intersect. They are situated mostly on the back, from the shoulders downwards, and often on the chest. The disease must be distinguished from ringed forms of *tinea versicolor*.

**Lichen scrofulosorum** is a chronic disease described by Hebra, but by no means yet recognized fully in this country or America, and is uncommon here. It is characterized by the development of pale yellowish or red papules, the size of pins' heads, which have a great tendency to a ringed or rounded grouping. Some papules may become crowned by a little scale, and some may inflame and take on an acneiform aspect. There is usually but little itching. The eruption is generally limited to the trunk, but may extend to the upper parts of the extremities. It is a disease of childhood, and is rare after puberty. A feature to be noticed is its very general association with such manifestations of struma as glandular and spinal or hip disease, or phthisis. It must not be confounded with the lichen circinatus or the annular miliary syphilide.

**Lichen planus** (Wilson) is a somewhat rare, non-contagious disease, marked by the development of discrete papules, accompanied by marked itching; in colour from lilac to deep purple; in shape flattened, slightly raised, smooth and shiny, or covered with a slight micaceous scale, well-defined, of angular outline, often with a central umbilication; in diameter from one to three lines; tending to become aggregated into irregular patches and bands, by the development of new papules amongst the persistent older ones; evolving, with a marked tendency to symmetry, chiefly over the anterior aspect of the forearms just above the wrist, round the waist and flanks, over the hips and about the lower end of the vastus internus, but occurring anywhere; and frequently leaving on their disappearance a melasmic staining like a syphilide. The papules persist as such, and show no disposition to become eczematous; there is never any marked scaling, and no bleeding points are exposed by removing the scales, as in psoriasis; nor, as in the latter disease, do the papules continue to enlarge peripherally and form rings. Further, like psoriasis and the papular syphilides, from which the diagnosis must be made, lichen planus tends to recur. In structure the papules approach those of psoriasis, so far that they are mostly formed by a hyperplasia of the

epithelium about the hair-follicles and sweat-ducts. In the usual form of *L. planus* here described a comparatively limited number of papules are seen, but Hebra described an affection called *L. exsudativus ruber*, in which large tracts of skin are involved with sheets of coalesced, slightly scaly papules. In these severe, widespread, and often acutely-developed phases, the papules lose somewhat the typical features described above, and become miliary and more or less acuminate. The nails may be involved; the itching may become intolerable; and marasmus may set in. The causes are unknown, but some attribute the complaint to a neurosis.

**TREATMENT.**—Arsenic is almost a specific for *L. circinatus*. The itching should be alleviated by frequent applications of a soda and glycerine or borax lotion. For *L. scrofulosorum* Hebra recommended cod-liver oil internally and externally. Arsenic should likewise be tried in *L. planus* and *exsudativus ruber*, but is not always successful; and then or simultaneously a toning up of the system should be carried out, by ferruginous preparations, mineral acids, and other tonics. Rest and change for an exhausted nervous system is, however, what is really required. Locally the very chronic forms may be stimulated, and the itching relieved, by tarry preparations, carbolic acid, and allied remedies, but alkaline baths and lotions, acid lotions, and lubricating salves, such as vaseline, are often useful.

**Prurigo** is a fairly common disease, characterized by the development of discrete rounded papules, the size of hemp-seeds, with a reddish tinge of colour, or of the same tint as the surrounding skin, and therefore often better felt than seen. The papules are widely disseminated over the trunk and limbs, and reach the sides of the face and scalp, but they mostly leave the great flexures free. The itching is very marked, and the tops of the papules are excoriated by scratching, whilst the related lymphatic glands, for example, those in the groin, become indurated. This disease usually commences in childhood, and persists with some intermissions, especially in summer-time, throughout the greater part of life. Consequently, in old standing cases the diagnosis is not so much from scrofula as it is in milder cases, but from chronic eczema, because the skin gets indurated and harsh, and thickly sown with an excoriated and slightly encrusted eruption, especially about the legs. Some papules may develop into abortive pustules, more particularly in cachectic subjects. The cause of prurigo is obscure, but the subjects of it are usually pallid and often debilitated. The inflammatory papules, which are rarely red and soft like those in eczema, are formed by a moderate exudation of cells and fluid into the papillary layer, with some secondary hyperplasia of the related epidermic structures.

**TREATMENT.**—Much relief may be afforded, and the disease held in check, by means of alkaline baths and the inunction of bland lubricants, as vaseline, and stimulating salves, such as those of tar, carbolic acid, or naphthol. Occasionally in children the eruption disappears under treatment, especially for a time, but many cases seem quite incurable. As regards internal remedies, the great object is to keep the general health as perfect as possible.

## VI. SQUAMOUS INFLAMMATIONS.

In this class are grouped two dissimilar affections, *psoriasis* and *pityriasis rubra*, which are brought together by reason of one marked feature which they have in common, namely, the shedding of much epidermis. Both are characterized by an inflammatory congestion of the skin; in psoriasis, in circumscribed spots and patches, and probably *secondary* to epidermic hyperplasia; in pityriasis rubra, primary and diffuse. Both are liable to recur, especially psoriasis.

**Psoriasis** is a chronic, bilateral eruption, frequently met with, and characterized by the evolution, more or less widely disseminated, of dull red, flat, slightly raised, rounded, and somewhat itchy papules (not polished and angular as in lichen planus), surmounted by well-marked silvery scales, which are easily detached by the nail, disclosing hyperæmic points. The eruption may evolve acutely and disappear spontaneously; but usually pursues a chronic course, by the persistence of the old papules and the appearance of new ones. The papules appear as dull red circumscribed elevations, the size of pins' heads, tipped with a scale (*P. punctata*). These spots enlarge centrifugally, and the scales increase, so that the eruption at this stage has been compared to drops of dried mortar (*P. guttata*). As they continue to enlarge to the size of a sixpence or shilling, they frequently clear up in the centre (*P. nummularis*), and finally form large rings (*P. annularis vel circinata*), which may intersect to form patterns (*P. gyrata vel figurata*). If the spreading eruption does not clear in the centre, a large thickened scaly patch is formed, often closely simulating a chronic squamous eczema (*P. diffusa*), especially when inveterate and fissured and weeping from external irritation (*P. inveterata* and *eczemateux*). Lastly, in some children and strumous subjects pus-cells mingle with the scales, and form crusts to simulate rupia (*P. rupiodes*). Many of these phases may co-exist on the same patient. Certain sites are especially prone to attack, such as the elbows and knees, the buttocks and scalp, the extensor aspects of the forearms and legs, but patches may occur also widely over the trunk. Though usually widespread, an isolated patch may exist, which makes the diagnosis more difficult. Psoriasis occurs in both sexes, in all ranks of life, and at any age, from childhood upwards. It is rare in infancy, although heredity is sometimes strongly marked. The papules are formed by a primary hyperplasia of the rete, with enlargement of the papillæ and secondary congestion, and increase of imperfectly formed cuticle. The disease is probably due to a peculiar morbid tendency in the skin itself; and the eruption in predisposed subjects is excited by almost any departure from a state of health, such as gout, struma, dyspepsia, debility from lactation, or pregnancy. The seasons of spring and autumn seem to favour its outbreak, and it is especially prone to recur. The diagnosis of psoriasis has mainly to be made from the papular syphilides and lichen planus, and this will be considered under syphilis.

**TREATMENT.**—In acute stages of psoriasis, with active congestion of the skin, soothing applications, such as watery compresses, oily applications, or alkaline baths answer best, combined with diuretics and laxatives internally. In ordinary chronic cases the general rule is to carefully sift out any departure from health, should such exist, and correct it; and then to exhibit arsenic, which has a remarkable influence



over a great number of cases. This drug should be given in increasing doses, carefully watching its effects, and continued some time after the disappearance of the eruption. The papules may be got rid of by the brisk infriktion of various stimulant applications, chosen according to the site involved, the sex and age of the patient, the chronicity of the eruption, and the extent of the disease. It should always be borne in mind that these applications may be absorbed when applied over a large surface. We may enumerate in order of increasing efficacy mercurial salves, thymol, carbolic acid, naphthol, tar, and chrysophanic acid.

**Pityriasis rubra**, or **general exfoliative dermatitis**, is a very rare affection, which commences as a reddened scaly patch, and as a rule *rapidly* involves the whole surface, so that in well-marked cases a striking appearance is produced, for the whole skin is of deep red hue, congested, and covered with large membranous scales, which are cast in great quantities. The disease is a chronic one, as a rule, though occasionally tending to be acute. Patients may quite recover, or the disease may end fatally, and be complicated by remittent fever, albuminuria, pulmonary œdema, or marasmus. They are extremely sensitive to cold. Very exceptional cases of lichen ruber, psoriasis and eczema, and pemphigus with abortive bullæ, may be universal, and are then, with very great difficulty, distinguished from pityriasis rubra.

**TREATMENT.**—Locally bland emollient preparations are called for, such as vaseline, glycerine of starch, or linimentum calcis applied after a bath taken for cleanliness. Internally the general health must be steadily toned up by arsenic, large doses of iron, and such remedies.

## VII. DIATHETIC DISEASES.

By this term we understand diseases that are dependent on certain persistent morbid dispositions of the body, either acquired or inherited, which modify to a greater or less extent all the nutritive processes. We shall here notice only the skin-manifestations of *syphilis* and *scrofulosis*, as their other symptoms and lesions have already been described earlier in this work.

**Syphilides** or **syphilitic skin-eruptions** vary in character somewhat, according as the patient is the subject of hereditary or acquired syphilis.

**HEREDITARY SYPHILIS.**—A *bullous syphilide*, or so-called *syphilitic pemphigus*, is the most precocious skin-manifestation, being frequently developed in the last months of intra-uterine life, and therefore to be seen on the child at birth, or at any rate it is of very exceptional occurrence after the seventh to the eighteenth day (Parrot). The bullæ are developed on a more or less infiltrated base, and hence ecthyma is often simulated. The contents are sero-purulent and thick, and on separation of the crusts unhealthy ulceration may be seen. The bullæ are of all shapes and sizes, from a varicella-vesicle upwards; and either discrete, confluent, or in crescents. The sites selected are especially the palms and soles, the backs of the fingers and toes, and neighbouring parts of the limbs. Only rarely are the bullæ of wider distribution.

The *macular syphilide* is the commonest eruption, and usually develops about the second month of life about the nates, backs of the thighs, and genitalia, as pinkish or coppery macules, rounded or irregular

in shape, circumscribed or more or less diffuse, and desquamating. These patches may extend widely over the body, face, and upper extremities, leaving untouched, however, the axillæ and lateral parts of the trunk. Mixed with these macules, or developing from them by the infiltration of the cutis, we find raised papules of various kinds, as in acquired syphilis, either small and lenticular, or larger and flattened, or discoid (nummular). The papules may relapse, and they now and again assume the ringed or crescentic forms, simulating a chronic psoriasis. As in the adult also, these papules may become variously modified by the site; thus on the scalp they may be eroded and crustitial; about the anus, vulva, corners of the mouth, and other moist situations, condylomatous; and so on. In strumous and cachectic subjects they ulcerate. Parrot describes a late *lenticular syphilide*, appearing at six months old or later.

The *vesicular and pustular syphilide* is more frequently met with, perhaps, in hereditary than acquired syphilis, and is as a rule a comparatively late manifestation. The eruption may closely resemble that of varicella, varioloid, variola, and the general pustular eruption that occasionally shows itself about the eighth day after vaccination. The fluid is often collected on a shotty, well-marked base; and the eruption is often multiform. As for other syphilides, Mr. Hutchinson, in speaking of the rarity of late symmetrical syphilides, remarks that, "after the first year, indeed, the subjects of inherited taint but rarely exhibit any kind of skin disease whatever; but, if they do, it is almost always some form of ulceration of a rupial, lupoid, serpiginous, or phagedænic character."

**ACQUIRED SYPHILIS.**—The skin is a structure especially prone to be attacked by the syphilitic virus, and the eruptions induced are of various types, corresponding pretty closely to the roseolous, papular, squamous, vesicular, pustular, and bullous conditions. Syphilides, as a rule, tend to evolve slowly, to *gradually* die out spontaneously, and to recur either according to a similar or different type. Each of these types, it must be pointed out, has its proper time of appearance in the natural evolution of the disease, though the regular order of things may be occasionally altered by various circumstances. The skin eruptions seen early in the disease tend to be widely and copiously disseminated over the body, to involve the skin superficially, and to be symmetrical; whilst subsequent eruptions get less copious, and more and more localized and asymmetrical, until in the tertiary stages the syphilitic growth involves the deeper layers of the skin, is localized, is present in greater mass and consequently ulcerates frequently, and there is but little tendency to symmetry, though both sides of the body may be attacked. The *macular or erythematous syphilide* has its time of evolution first after the induration of the glands, and it has a characteristic site about the sides of the abdomen and thorax, or all over the trunk, and perhaps the upper parts of the extremities. The eruption consists of delicate rosy macules, the size of the finger-nail, which evolve in crops, remain out from a few days to several weeks, and then fade away, leaving tawny stains. It itches only when of unusually acute development. It may be confounded with any macular erythematous eruption, such as measles, simple roseola, erythema multiforme, or especially copaiba eruption, but corroborative signs of syphilis are usually present in the throat or elsewhere. The *papular syphilides* present a wide diversity of appearance. They evolve in point of time after the roseolous type, or as the latter is

declining, and they may continue to relapse year by year, becoming more localized and far less copious. The chief forms are as follows:—The *small or miliary papular syphilide*, or so-called *syphilitic lichen*, is rare and precocious, and is characterized by collections of conical, shotty papules, the size of a pin's-head, in consequence of the development of the syphilitic tissue especially around the follicles. They tend to collect in groups. Large papules may be present, and many of the miliary papules may become tipped with pus (see *pustular syphilides*). The eruption is indistinguishable in appearance from a simple or scrofulous lichen. The *large or lenticular papular syphilide*, or so-called *syphilitic psoriasis*, is very common, and consists of circular or oval, flattened, dull red, indolent papules, usually discrete, but occasionally fused into patches, with very few scales as a rule, and in size from a split pea to a sixpence. Occasionally the papules are disc-like (*nummular papular syphilide*) or circinate, especially about the face; or moderately scaly, and then more closely resembling ordinary psoriasis. The site on which they occur modifies their aspect; for example, about the forehead and hairy parts they become eroded and slightly crusted (*papulo-crustial syphilide*) in hairy parts often warty (*vegetating* or *frambæsioid*); on vascular mucous or muco-cutaneous and other moist situations, as beneath folds of skin, in flexures of joints, interdigital clefts, about the prepuce and labia, or perinæum and anus, they become soft, auto-inoculable masses, exuding a viscid secretion (*mucous tubercles* or *condylomata lata*); about the corners of the mouth and between the toes they get fissured and ulcerated; in the mouth they are soddened and silvery-looking (*mucous patches*); and, lastly, in the thick skin of the palms and soles the outlines of the papules are often lost, and the eruption closely simulates chronic eczema or psoriasis. In diagnosing these papular syphilides from psoriasis, it must be remembered that syphilis is almost invariably acquired in adult life, and in the relapses the eruption is less symmetrical and copious and more localized, and may change its type; whereas psoriasis may evolve for the first time at any age, but often begins in childhood and recurs throughout life. Psoriasis is also often hereditary, whilst the papular eruptions inherited do not occur after the first eighteen months. Psoriasis affects especially the extensor surfaces of the limbs, such as the elbows and knees, also the scalp and buttocks; papular syphilides favour rather the flexor surfaces of the limbs; in both, however, the eruption may encircle the limb or stud the trunk. Psoriasis papules, arising as they do from papillary hypertrophy, are, as a rule, rather the more elevated, in consequence of the notable accumulation of silvery scales, which, when detached, expose bleeding points; syphilitic papules, which are formed by deposition of special cell-growth, rarely have very many scales, and there are no bleeding points to be seen unless the surface is forcibly injured. Syphilitic papules frequently have a dull-red, coppery, or raw-ham tint, distinguishable in many cases from the less sombre hue of psoriasis; and again they often present a multiformity of aspect in different parts of the body. It must also be remembered that relapsing papular syphilides are sometimes annular. In making a diagnosis, then, too much stress must not be laid on any one character, but the features and history of the eruption must be considered in their totality. On the palms and soles a papular or relapsing syphilide may occur asymmetrically; whilst eczema and psoriasis in these regions are symmetrical, and almost invariably associated with disease elsewhere. The papules of lichen planus are



frequently mistaken for syphilis by those not acquainted with their peculiar characteristics. The *vesicular syphilide* in the acquired disease is only a curiosity. *Pustular syphilides* are more common, but still rather rare, and they are conveniently arranged as follows:—The *acnei-form syphilide* is the pustular development of the miliary papular eruption, and frequently co-exists with it. The shotty, coppery, conical papules are surmounted by a little collection of pus, which dries into a crust or scale, and some pit-like scars result. They are wide-spread at first, but more localized in relapses, for example, about the forehead. The eruption has to be distinguished from acne cachecticorum, acne varioliformis, and potassium iodide acne. The *varicelliform* and *varioli-form syphilides* have a somewhat greater proportion of pus present, and must be distinguished from varioloid and variola, and some are pemphigoid eruptions. The *impetiginous* and *ecthymatous syphilides* are larger, and simulate a discrete pustular dermatitis, as from scabies and phtheiri-asis. A coppery infiltrated base is to be made out, and in cachectic subjects considerable scarring may result. The early forms are superficial, but the relapsing later phases affect the skin more deeply, and much crusting results, so that as the ulceration extends peripherally, adding fresh layers of crust from below, a limpet-like compound crust is built up, of very characteristic appearance. To this the term *rupia* is applied. An eruption like ordinary pemphigus has been described in acquired syphilis, but is very rare indeed. Most bullous forms are rather of an ecthymatous type. There is further a rare *pigmentary syphilide*, closely simulating a small-patterned leucoderma, occurring about the neck of women in the secondary period. *Tubercular* or *nodular syphilides* are common between the second and seventh years, but after that get less frequent. There is usually a marked cachexia present. They occur as more or less rounded, circumscribed, firm or hard, painless and indolent, coppery or livid nodules, involving the skin pretty deeply, and in size from a pea to a large bean. They may be smooth and shining, or occasionally slightly scaly or crusted over, and they evolve in crops in the neighbourhood of pre-existing ones, which resolve so that they appear to wander. They may be discrete, or grouped in highly characteristic crescentic serpiginous forms. They affect chiefly the face, the bottom of the neck, and behind the sternal and gluteal regions. The nodules may break down into ulceration, and in cachectic subjects this is deep, and may destroy the nose, for instance. In other parts deforming cicatricial bands result. In mass they may closely simulate lupus vulgaris, and the name *syphilitic lupus* has been applied. The diagnosis is sometimes almost impossible; but as a rule lupus nodules are a brighter red; the apple-jelly-like appearances is detected in the smaller ones; and the grouping does not assume the reniform outline. *Gummata* rarely occur before the third year, and usually much later. Their favourite sites are the scalp, face, and extremities about the joints. Usually they are few in number. They are isolated, elastic, freely movable swellings, and circumscribed, unless when involving a part, such as the lip, where the connective tissue is loose. The superficial parts of the skin may become gradually implicated; and an ulcer, with a foul uneven base, and steep punched-out edges, results. Such multiple ulcers about the upper third of the leg are not uncommon.

With regard to the diagnosis of the syphilides generally, it must be pointed out that, though they present such a diversity of form, they have certain family characteristics. Their slow evolution and indolent

character is associated with the absence of marked itching as a rule. A dull reddish-brown, raw-ham, or coppery colour usually characterizes them. The eruption tends to be grouped in a circular or crescentic manner, more especially in relapses; but it must not be forgotten that the ringed form of eruption is seen in many other skin-affections. Syphilides exhibit a polymorphism, that is, the coincidence of several phases of eruption. They also have their favourite sites.

TREATMENT.—The constitutional treatment for syphilis has been already considered in this work, and we only refer here to the local measures required. The erythematous and widespread papular eruptions may be treated by the application of dusting powders, or a lotion of calamine, oxide of zinc, and black wash. To localized non-ulcerating syphilides mild mercurial salves may be applied. Where ulceration exists, the crusts must be removed, and the surface cleansed and healed by iodide of starch paste, iodoform applications, mercurial plaster, simple healing remedies, or local mercurial fumigation. Condylomata require great cleanliness, and dusting with calomel, mixed with magnesia or oxide of zinc and starch.

### VIII. HYPERTROPHIES AND ATROPHIES.

In this class are included those diseases which are characterized by an increase or diminution in the size, or in the quantity, of the normal elements of the skin. We have to consider *xeroderma* and *ichthyosis*, *keloid*, *fibroma* and *dermatolysis*, *morphœa* and *scleroderma*, *callositas*, *clavus* or *corn*, *verruca* or *wart*, *atrophia cutis*, and *striae et maculae atrophicæ*.

**Ichthyosis** is a congenital structural and functional disease of the skin; presenting much diversity of appearance according to its severity; involving the greater part of the body, except, as a rule, the great flexures, face, genitals, and palms and soles. Though to be considered congenital, it does not become apparent in the great majority of cases till after the first few months of life. In the mildest forms the extensor aspects of the thighs and upper arms are roughened by the plugging of all the follicles by exuviae—an appearance very similar to that seen in *keratosis pilaris*. In another very mild phase, known as *xeroderma*, the skin generally is dry and harsh, shiny or roughened, and dirty-looking from the absence of sebaceous and sweat secretions. This condition is worse in cold weather, and when inflamed simulates an eczema. In a somewhat exaggerated condition of this form the cuticular scales are parcelled out in little lozenge-shaped areas, and these plates show a tendency to adhere only by their central parts. In more severe cases the skin gets thickened, and the plates more and more deeply marked out and like mother-o'-pearl (*ichthyosis nacrée vel nitida*). When the papillæ are much enlarged there is a considerable accumulation of ragged epithelium, and probably entangled inspissated sebum and much dirt, so that large horny or mudlike masses may project from the skin, and to these various phases the terms *I. cornea*, *hystrix*, and *hystricimus* have been applied. Most fanciful names have been applied in popular language, such as man-fish, porcupine-man, serpent-skin, &c. Ichthyosis is often hereditary; it persists throughout life; and is incurable. It may, however, be much ameliorated, and kept in check.

**TREATMENT.**—In mild forms of ichthyosis the inunction of lubricants, such as vaseline, glycerine of starch, or glycerine and water, suffices. In cases where much epithelial and other *débris* accumulate, the masses must be removed by prolonged alkaline baths or strong alkaline lotions, and then oily or mild stimulants applied beneath.

**Fibroma** or **Molluscum fibrosum** is an affection of the skin in which there is a special tendency for the connective tissue to hypertrophy and outgrow. There is some doubt at present whether the connective tissue around the sebaceous glands or the nerves is not specially involved. The growths commence as little soft tumours, and as they increase assume all sorts of shapes, and grow to a large size. They may be sessile or pedunculated, smooth or corrugated, flaccid or tense and elastic, normal in colour, or livid or darkly pigmented. Old growths may ulcerate. When single they usually come under the care of the surgeon; but occasionally they develop in crops and in large numbers over the body. Cases of the latter class begin early in life and progress, and the subjects often have a stunted mental and physical development. Fibroma has been noticed in several members and in several generations of a family. The tumours consist of fibrous tissue in various states of active development and condensation.

**TREATMENT.**—The evolution of these growths cannot be prevented; but if a tumour is a source of annoyance it can be removed by the knife or by ligature.

**Dermatolysis** is a closely allied affection, in which, however, the whole thickness of the skin hypertrophies and hangs in folds.

**Scleroderma** and **Morphœa** are here classed together, because in this country they are believed to be respectively the diffuse and the circumscribed form of the same affection. To understand the different phases of the disease, it is necessary to appreciate the fact that a process of atrophy of the connective tissue and other structures may take place from the outset, or may follow varying degrees of hyperplasia. These changes disturb the vascular supply, the pigmentation, and the nerve functions of the parts. *Morphœa*, in its most typical aspect, commences as a circumscribed pigmented or rosy macule, in size from that of a finger-nail to the palm of the hand. The skin of this patch becomes gradually denser from the increase and condensation of connective tissue; often somewhat depressed, until it cannot be pinched up; and paler, whilst enlarged venules are seen to course here and there, and the white alabaster-looking centre is surrounded by a delicate lilac halo of congestion (*M. lardacea*). This condensation of tissue interferes further with the already-disordered nutrition of the part, and the cuticle sometimes tends to separate. There is more or less anæsthesia, with cessation of sweat and sebaceous secretion, and blanched, or absence of, hair. Occasionally the hyperplasia of connective tissue is sufficient to raise the part up into nodules and large lumps (*M. tuberosa*). These hypertrophic phases may be succeeded by an atrophic process (*M. atrophica*); or the atrophy may be the chief feature from the first, and then a shiny, smooth, shrunken, circumscribed area of skin presents itself. Very considerable pigment-disorder often exists. There may be only a single morphœa patch; or several or considerable numbers arising in succession over the body, and more or less confluent, thus approaching the sclerodermatous condition. They tend to assume the rounded form, but often are irregular in shape, or in bands. The sites mostly affected are the base of the neck and adjoining portions of the thorax, the sub-



mammary regions and abdomen, the forehead, and the proximal segments of the extremities. Morphœa occurs mostly in the young or in early middle life, and in females. Often no cause can be made out, but many cases are associated with marked debility, and various circumstances point to a neurotic origin; for instance, the occasionally marked relation between the distribution of the patches and cutaneous nerves, as in morphœa and herpes of the first division of the fifth nerve. *Scleroderma*, or “the hide-bound disease,” is still rarer than morphœa, and differs from the latter in being characterized by the *diffuse* hardening and stiffening of a large surface, so that the skin feels cold and shrunken and tightened on the underlying parts. It is sought in England to connect the morphœa, in which one circumscribed patch appears, with scleroderma, in which the whole body is involved. In the diffuse forms the atrophic process is predominant. Scleroderma may set in acutely after a chill or an attack of rheumatism, and rapidly involve the greater part of the body, or only a limited region, as the hands and forearms or lower extremities; but at other times it has an insidious onset and chronic course. It usually begins in the situations just mentioned, or about the nape of the neck. As in morphœa, the subcutaneous tissues and even the bones may be involved in the disturbance of nutrition. Much deformity may result from the drawing down and fixing of the eyelids and lips, and the impairment of motion about the joints. Scleroderma occurs most frequently in young and middle-aged women; and tends as a rule to disappear spontaneously, like morphœa, after a number of years. *Sclerema neonatorum* is a somewhat similar-looking, though probably distinct generalized affection, occurring in newly-born children, and ending fatally as a rule. It seems to be due to a setting of the fat, and a peculiar kind of œdema.

**TREATMENT.**—The patient should be made to live under the healthiest possible conditions. Internally cod-liver oil, ferruginous and mineral acid tonics, phosphorus and strychnine, are called for; whilst externally warm bathing, with shampooing, the infriktion of bland oils, and the continuous current do most good in restoring the nutrition.

**Atrophia cutis** occurs as a secondary condition in many affections, such as syphilis, and alopecia areata; but, with the exception of senile atrophy, in which the loss of the subcutaneous fat brings about a wrinkled shrivelled condition, it is rare as a primary change. It would seem to be primary, however, in scleroderma and some cases of morphœa. There is, further, a rare affection occurring in lanceolate stripes and rounded spots, closely simulating the lines of pregnancy, and named *linear* and *macular atrophy*. It occurs in groups and parallel lines, chiefly about the hips and thighs, the axillæ and upper arms, and above the knees. The affected parts are depressed, livid or like mother-o'-pearl, glistening and smooth, or reticulated. A primary hyperæmic stage has been described. The cause of the condition is unknown.

## IX. NEW FORMATIONS.

Under this heading are included the heterologous neoplasmata of the skin, namely, *cancer*, *rodent ulcer*, *sarcoma*, *lymphadenoma*, and *lupus*. These diseases are all chronic, and the newly-formed tissue has a constant tendency to invade fresh regions, to break down into ulceration, and in most cases ultimately to destroy life. We shall only discuss here lupus, as the others come within the province of surgical works.

**Lupus** is a non-contagious, chronic, probably non-hereditary disease of the skin and some adjoining mucous membranes, characterized by the formation of a new small-celled growth in the meshes of the cutis, resembling that of syphilis and leprosy, tending to spread peripherally, but not very deeply, by infection of neighbouring cells, and to undergo an atrophic or destructive process, leaving scars. Two groups of lupus are described, namely, *L. erythematosus*, which Kaposi regards rather as an inflammatory affection; and *L. vulgaris*. Lupus most frequently attacks females; is associated with evident signs of struma in a great many cases; and generally the subjects of it have an enfeebled constitution and a bad circulation. Lupus vulgaris develops, as a rule, in childhood or youth; whilst the rarer *L. erythematosus* commences later, namely, in early adult life. *Lupus erythematosus* and *L. sebaceus* or *acneiform lupus* may have an insidious beginning as a local seborrhœa, or apparently a patch of erythema. The peculiar feature is that the red, raised, spreading border is studded with the dilated openings of the sebaceous ducts; which in their turn are plugged with epithelial debris and inspissated sebum; whilst the older central parts of the patch atrophy and leave faint scars. Such patches may be few in number, reach a considerable size, and fuse together to cover a large area. In rarer cases smaller *disc*-like patches develop, often in great numbers, and sometimes acutely. *L. erythematosus* occurs with especial frequency about the face, ears, scalp, hands, and genitals; more rarely on the feet; and occasionally more widely. Over the cheeks and nose a very characteristic fusing of patches takes place, to form what is known as "butterfly lupus." *L. vulgaris* presents itself in many aspects. The mildest form is a chronic erythema-like spreading eruption, which has a dry, gelatinous aspect, and atrophies in the centre. It occurs on regions similar to those in *L. erythematosus*, and is almost indistinguishable from the latter disease, save for the absence of the implication of the glands. *L. vulgaris* more commonly makes its appearance, however, as little, semi-transparent, grey, glistening, *soft* nodules, imbedded in the skin. They have a close resemblance, in appearance and structure, to true miliary tubercles, but do not undergo the same early caseation. The "tubercles" grow in size, or become confluent, so as to form larger red nodules, and then the jelly-like aspect is generally masked by the congestion present. The surface then becomes glazed, wrinkled, and it exfoliates (*L. exfoliativus*). In chronic cases the skin is occupied by much lumpy growth, which projects very considerably above the surface, or the patch, like other chronically congested areas, is the seat of papillary hypertrophy (*L. hypertrophicus*). Where the new growth is in any quantity it tends to break down into ulceration (*L. exulcerans* or *exedens*, as distinguished from *L. non-exedens*). The ulceration does not tend to go deeply like rodent ulcer, except in rare cachectic cases, but in severe cases the cartilages of the nose and ears may be destroyed, very extensive and deforming cicatrices being left. Lupus spreads in a characteristic chronic way, by the formation of fresh nodules of new growth at the periphery, whilst the central older parts atrophy, often without ulceration, and leave a scar. Consequently the serpiginous form assumed reminds one strongly of the tubercular syphilides. Where ulceration occurs in these spreading forms the ulcer is covered by a crust, and advances *pari passu* with the new growth. The erythema-like forms of lupus manifestly have to be distinguished from chronic forms of erythema, and mild inflammations,

such as *tinea circinata*, *seborrhœa*, &c. The implication of the sebaceous glands, and the atrophic process, however, are very characteristic. *Lupus vulgaris* has to be carefully distinguished from tubercular syphilis, but the diagnosis is not difficult as a rule.

**TREATMENT.**—Soothing applications, such as vaseline, oxide of zinc, and calamine lotion, or oleate of zinc or bismuth, should be employed in *lupus erythematosus*, especially when it is actively spreading. If these fail, collodion or liquor plumbi may be painted on, and when quiescent, many practitioners recommend the application of resolvents and stimulating remedies, such as iodine, mercurial plaster, soft soap, or tar. Finally, scarification is resorted to occasionally. In *lupus vulgaris* also soothing measures are called for in irritable and active phases of the disease; but when quiet the soft nodules should be removed or destroyed by means of the sharp spoon, gouging with nitrate of silver points, or the cautery. A variety of caustics and resolvents are also used. If there is much foul ulceration it may be cleansed with iodide of starch paste. Internally cod-liver oil often effects wonders, and efforts must be made to build up the health in every way possible.

#### X. PIGMENTARY CHANGES.

**Melanoderma**, **melanopathia**, or **melasma**, are names used at one time or another to denote a state of increase of pigment in the skin. An increased deposition of pigment occurs in a variety of conditions. Thus it very commonly follows on any continued hyperæmia of the skin, as after long exposure to the sun or a fire, and is especially prone to ensue in the erythematous inflammations, in syphilides, and in lichen planus. It occurs physiologically in pregnancy and during menstruation; and in cachectic individuals suffering from phthisis and cancer. It is seen about the forehead and temples of debilitated women (*chloasma uterinum et gravidarum*), and follows occasionally a persistent phtheiriasis (so-called "vagabond's disease"). The well-known *ephilides* or *freckles* are usually excited by the sun's rays, but not invariably. In some affections, in which the nerves are involved, pigmentation is seen, as in leprosy and the circumscribed and diffuse forms of scleroderma. This brings us to notice that cases are on record in which intense pigmentation has followed a great fright and mental trouble. Other cases of widespread or universal pigmentation occur without any assignable cause. In such cases a careful study must be made, to exclude, if possible, diseases of the suprarenal capsules and neighbouring sympathetic nerves (see Addison's disease).

**Leucoderma** or **leucopathia** is another disturbance of pigmentation of unknown causation. Here there is not only increased deposition of pigment, but simultaneously a removal of pigment from circumscribed well-defined oval areas, producing a very striking appearance. It occurs at almost any age, from advanced childhood upwards, and in either sex, and is usually very widespread, with a tendency to symmetry. The skin itself is structurally unaffected, and its functions are probably normal. Brunettes are specially subject to these changes, which are seen in spare and often debilitated people.



## XI. NEUROTIC AFFECTIONS.

Hyperæsthesia and anæsthesia have been already discussed, and we need only refer here to *pruritus*.

**Pruritus**, or the sensation of **itching**, must not be confounded with the disease *prurigo*. It is a frequent accompaniment of skin-diseases, especially where there is infiltration about the nerves, as in eczema; or external irritation, as from lice or the itch insect. It is met with also in a general and most intense form in some neurotic states not well understood; and locally about the vulva or anus, from irritating discharges (diabetes), thread-worms, or venous congestion (hæmorrhoids).

**TREATMENT.**—This will be as various as the cause of the itching, which must be removed. Lubricants to soften harsh skin; sedatives, as hydrocyanic acid; stimulants to resolve infiltration; and alkalies are especially useful in relieving this symptom.

## XII. PARASITIC DISEASES.

This class includes all the affections produced by the various animal and vegetable parasites that infest the human skin. We shall therefore have to describe firstly *dermatozoic* affections, namely, *phtheiriasis*, *scabies*, and *bug* and *flea eruptions*; and secondly *dermatophytic* diseases, including *tinea favosa*, *tinea trichophytina*, and *tinea versicolor*.

I. DERMATOZOIC.—**Phtheiriasis**, **pediculosis**, or **lousiness** is the condition due to the presence of pediculi or lice about the body. Three different species infest the human subject—the *P. capitis*, the *P. vestimenti vel corporis*, and the *P. pubis*. These creatures are furnished with powerful legs, and a proboscis by which they suck up blood from the skin, and so set up irritation and various forms of eruption. The *P. capitis* inhabits the hairy scalp, especially the occipital region in children and young females, and sets up a pustular dermatitis, formerly called impetigo, with consequent enlargement of the related glands. The *P. pubis* or crab-louse is mostly localized to the pubic hair, but in some long-standing cases may be found clinging to the hair of the armpits, trunk, or limbs, and even to the eyelashes and eyebrows. They cause unbearable irritation, but little inflammation as a rule. The *P. vestimenti vel corporis* infests the trunk and clothes of persons, especially of those somewhat advanced in years and of debauched habits. The real habitat of the parasite is the clothes, especially the seams and folds, so that the irritation will always be found worse on those parts of the body where the clothes press, as about the shoulders and waist. The louse inserts its proboscis deep into a follicle, and thence a little blood wells up to form a tiny crust, whilst a transient hyperæmic papule or urticarial wheal forms around. The irritation induces determined scratching, so that many papules are excoriated, and broad linear excoriations are frequent. In cachectic subjects ecthymatous pustules form, and pigmentation may follow in old-standing cases. This disease, once called *prurigo senilis*, must not be confounded with true *prurigo*.

**TREATMENT.**—Locally mercurial powders, lotions, or ointment, sassailla, cocculus indicus, staphisagria, sulphur, and carbolic acid are very effectual. For the hairy scalp crude kerosene oil, or two grains of perchloride of mercury to the ounce of dilute acetic acid destroys the lice and the nits also. When this is done soothing salves must be

applied to allay any inflammation. Infected clothes must be heated to at least 200° F.

**Scabies**, or the **Itch**, is a disease due to the presence on and in the skin of a minute parasite, known as the *acarus scabiei* or *itch-mite*, which is just visible to the naked eye as a white speck. The male is smaller, and is a wanderer on the surface; whilst the larger impregnated female tunnels her way between the epidermic layers, and there lays at intervals twenty or more eggs, and finally ensconces herself beneath a little eminence or vesicle at the end of the tunnel or *cuniculus*, which looks like a miniature mole-run. These cuniculi are linear tortuous elevations, a few lines long, and present black dots at intervals. The eggs hatch in from five to fourteen days, and the young repeat their parents' history. Besides these pathognomonic cuniculi, the itch insect excites intense itching, increased at night, both at the seat of burrowing and sympathetically over a wider area; as well as various degrees of dermatitis indistinguishable from eczema, ecthyma, boils, and urticaria. The amount and severity of these eruptions depend on the age and state of health of the patient, and the duration of the disease. The *acarus* favours the buttocks and feet of babies; the thighs, abdomen, and hands (if not constantly washed) of adults, and in addition the region about the mammæ and axillæ of females. The face is very rarely involved. The complaint is of course highly contagious. It must be distinguished from chronic eczema, prurigo, and phtheiriasis at all ages; and in children also from urticaria papulosa.

**TREATMENT.**—The first thing to do is to order a prolonged warm bath, with the thorough application of soap, in order to open up all the tunnels, and then the *whole body* should be dressed night and morning, for three or four days, with some parasiticide ointment, such as a mild sulphur ointment. After a second bath the patient will be cured. In hospital practice a stronger parasiticide is often used, and the cure completed in one visit. The balsam of Peru and styrax, and mild mercurial salves are very efficient, and any of these may be used without a preliminary bath. When the acari and their eggs are killed and removed, soothing measures should be adopted, in order to control any inflammation present.

**II. DERMATOPHYTIC.**—The natural history of the several moulds or fungi which flourish on the human skin, and their relationship to one another, is not yet placed on a certain basis, but the three here considered seem to be distinct.

**Tinea favosa** or **Favus** is very rare now in England, and when seen is generally met with in foreigners. It is, however, somewhat commoner in Ireland and Glasgow. It is a disease of dirty, ill-nourished children; but as it is very inveterate, it is sometimes met with in adults.

Favus is characterized by the development of a fungus (*Achorion Schönleinii*), rather larger on the average than the trichophyton, composed of spores and branched and unbranched mycelium tubes, terminating in moniliform chains, which ramify in the epidermic layers, and especially about the hair-follicles. The mass of the fungus and the disintegrated epithelium and sebum form small, sulphur-yellow, discoid crusts or cups, with a peculiar mousy swell, and very characteristic appearance. They leave scars eventually, and may at an early stage be mistaken for pustules.

The hairy scalp is the most frequent site attacked in tinea favosa, but the skin is sometimes implicated, and then the crusts are preceded by an appearance simulating *T. circinata*, to be presently described.

The nails may also be involved, and rendered opaque and brittle. The complaint may be acquired directly from animals much subject to it, namely, mice, rats, cats, and canaries.

**Tinea trichophytina**, or **Ringworm**, is due to the growth in the skin and its appendages of a somewhat different fungus called the *trichophyton*, which consists of wavy, smooth-margined, transparent mycelial tubes, jointed or unjointed, and usually unbranched, terminating in bead-like chains, and of oval or round spores, about half the diameter of a red blood-corpuscle. It is occasionally contracted from animals. The affection differs in appearance as it attacks the hairy scalp (*T. trichophytina tonsurans*), the general body surface (*T. t. circinata* and so-called *eczema marginatum*), the hairy parts of the face (*T. sycosis*), and the nails (*T. t. unguium*). In hair spores, and amongst the epidermic cells of the nails and skin mycelial threads predominate. *T. circinata* is first noticed as a small, circular, reddish, elevated spot, of the size of a split pea, or larger, which becomes slightly desquamative, and is attended with great itching. As the spot extends at its periphery, by the shooting out of the fungus amongst the cuticular and upper rete cells, the centre clears up, as is observed in the fairy rings on grass, and the patch becomes a ring, which may increase to a diameter of three, four, or more inches. *T. circinata* may occur on any part of the body, but is more common in those parts which are more freely exposed to the contact of the fungus, as the face, neck, backs of the hands or wrist. The hairs may be to some extent affected. The irritation caused by the growth of the fungus may not be confined to the production of a hyperæmic patch, but the margin sometimes becomes studded with papules, vesicles, or pustules, and then crusts form. This brings us to mention that occasionally, in adults mostly, the inflammatory patch looks like a chronic eczema, especially about the upper part of the thighs, the buttocks and genital regions (so-called *eczema marginatum*). In warm climates, where the fungus flourishes more luxuriantly, this is not uncommon. *Tinea tonsurans* is met with almost exclusively in children, and in those who are pallid and "lymphatic." It may begin on the general surface, but when the patient is brought for advice there are usually several isolated or confluent, rounded, ashy-grey, scurfy patches, over which are studded swollen, opaque, and exceedingly brittle stumps of hairs full of spores. Occasionally the diseased hairs may be disseminated singly or in little groups about the scalp. The fungus does not excite, as a rule, the same amount of inflammation in the scalp, but a general seborrhœa or eczema may supervene and mask the primary mischief, or a boggy, infiltrated condition, known as *Kerion* may occur, exuding a viscid fluid, and turning out all the hairs. Ringworm of the hairy scalp is an inveterate malady, but tends to die out towards puberty. *Tinea sycosis* is rare in England, and usually begins as *T. circinata*, and as the hairs become affected they assume an appearance identical with that seen in the scalp. The only point to be mentioned is that a pustular folliculitis may be excited, and occasionally severe boil-like inflammations. When the fungus attacks the nails, as it now and then does, they become opaque, brittle, and broken up—*T. unguium*. Usually one or two nails only are affected, and on one hand.

**Tinea versicolor**, or **Pityriasis versicolor** (formerly called **Chloasma**), is caused by the growth in the cuticle of the *microsporon furfur* fungus, which has some peculiarities, inasmuch as the mycelial threads end in aggregations or balls of spores, which are very charac-



teristic. The fungus especially selects the skin of the chest and shoulders, but in long-standing or exceptional cases spreads further over the trunk, and to the upper segments of the limbs. The eruption consists of little shiny or furfuraceous spots or discs, very faintly elevated as a rule, of a characteristic fawn or darker colour, starting about the hair-follicles, and slowly enlarging to join other spots in an extensive sheet enclosing islands of normal skin. *Tinea versicolor* is essentially a disease of the middle period of life, and is said to be most frequent in phthisical subjects.

**TREATMENT.**—The main object is to kill the fungus by parasitocides, when all secondary inflammations and other morbid processes cease. On the general surface this is easily done, because the fungus is superficially placed, but in the nails, and especially the hair-follicles, the fungus reaches so deeply that it is most difficult to get any parasitocides to penetrate. Mere antiseptics are useless, and active parasitocides must be used, such as mercurials, carbolic acid, sulphur compounds, thymol, or chrysophanic acid. The mechanical removal of the upper layers of the skin by blistering or caustic applications is sometimes useful, but the thorough and persistent inunction of milder remedies, properly adjusted to the age of the patient and the state of nutrition of the skin, is generally effective. The hairs should be extracted if sufficiently loose in the follicles. Proper directions must be given to ensure that the disease is not kept up or propagated by infected towels, brushes, linen, &c. In inveterate cases which are *localized to small patches*, kerion may be excited, and the hairs so removed. A hyposulphite of soda solution (3j to Aq. 3j), with thorough soap-washing, is usually sufficient in *tinea versicolor*.

### XIII. AFFECTIONS OF THE GLANDS AND APPENDAGES OF THE SKIN.

The glands and their ducts are frequently involved in the skin-diseases which have already been described, and either share in a general atrophy or hypertrophy, or become the special seat of inflammatory mischief, by reason of the rich vascular plexuses surrounding them. Likewise the hairs in their follicles and the nails may become implicated. We intend here, however, to direct attention only to primary disturbances of these structures.

**Affections of the sweat-apparatus.**—The sweat may be diminished in quantity (*anidrosis*), as in belladonna-poisoning and ichthyosis; but as an isolated symptom this is rarely met with. On the other hand, excessive sweating (*hyperidrosis*) is common, and affects the body generally in the sweating stage of ague, in rheumatic fever and pyæmia, in debilitating conditions, such as phthisis and cancer, and in the crises of some fevers. Localized sweating occurs in some paralyses and other nerve-lesions; and is also met with about the genitals, the axillæ, and palms and soles of certain people, causing great annoyance on account of the saturation of the clothes, the irritating effects of the decomposing sweat, and the frequent development of a penetrating disagreeable odour (*bromidrosis*). If the sweating be sudden and violent, or the mouths of the follicles be closed, the retained sweat collects temporarily under the cuticle in tiny pellucid vesicles (*sudamina* or *miliaria alba*), which must not be confounded with rare cases in which discrete eczema vesicles stud an erythematous surface. If the follicle be congested at the same time,

the fluid is often less clear and alkaline, and the prominent feature will be a red soft papule (*miliaria rubra*, or *prickly heat*). On the palms and soles of debilitated and neurotic people, the sweat sometimes collects in vesicles imbedded in the thick skin, accompanied by heat and painful tension; these may become raised and confluent into multilocular bullæ, while the skin becomes macerated, but the affection may be distinguished from eczema by the absence of a raw weeping dermis. This *dysidrosis* often recurs, and is occasionally chronic. There is a difference of opinion as to whether it is really a sweat affection, a pemphigus, or an eczema. Lastly, the sweat under some conditions becomes coloured (*chromidrosis*) red or blue or black, but very many of such cases are impositions.

**TREATMENT.**—Belladonna, externally and internally, has the power of controlling excessive sweating; and occasionally an astringent, such as alum or oxide of zinc, combined with an absorbent (starch powder), and an antiseptic, such as boracic or salicylic acid, is very useful locally, especially where a strong odour exists. It is found in the latter case that the smell comes from the decomposing sweat that has soaked into the wearing apparel, and this must be prevented by antiseptics and frequent change. Where debility exists, ferruginous and mineral acid tonics are called for. A calamine and oxide of zinc lotion is pleasant in miliaria; and in dysidrosis belladonna fomentations afford relief in the early stages, and bland lubricant applications later on.

**Affections of the sebaceous glands.**—The secretion from the sebaceous glands, and its passage from the ducts, may be sluggish from general debility or other causes, and then the follicles become plugged and congested, so as to form little conical papules. Such a condition is seen in one form of ichthyosis; and on the outside of the upper arms and the thighs, and occasionally in other parts, in children of a phthisical habit (*keratosis pilaris*). In adults, too, after pityriasis rubra or idiosyncratically, a similar state of things is met with, and then the rough file-like projections are tipped with a black speck of dirt (*lichen pilaris*).

**TREATMENT.**—Improve the general health where necessary; remove the plugs by alkaline baths and applications; and excite to healthier action by stimulants.

**Acne** is an extremely common disease, which begins with a very similar plugging of the sebaceous follicles (*comedones*), but then acne is a definite affection associated with the development of the body at puberty and some years afterwards. In those disposed to the eruption it is frequently to be traced at this period of life to dyspepsia, debility, and derangement of the generative organs. It has its favourite sites on the face, and about the shoulders and chest. The sebaceous plug stimulates the excitable surrounding vascular plexus, and an inflammatory papule is produced (*A. papulosa*), which may pass on to suppuration (*A. pustulosa*) and leave scars, or large chronic boutons or nodules form (*A. indurata*). There are also one or two other acneiform affections to be noticed, in addition to *acneiform syphilides*. Thus an eruption indistinguishable from acne, with the exception that there are no comedones, occurs about the forehead and anterior portion of the scalp of adults (*A. varioliformis*). Deep pits are formed, into which the little crusts sink. It is thought by some to be only a form of relapsing acneiform syphilide. Workers in tar sometimes acquire a more or less general acneiform eruption; as well as persons taking iodide and bromide of potassium. There is finally a general acne met with in very cachectic individuals (*A. cachecticorum*).

**TREATMENT.**—Where the affection can be traced to a drug, or to tar, or to general debility, such cause must be removed. In the acne of puberty any dyspepsia, constipation, menstrual irregularity, or debility, must be looked to; and locally the comedones should be extracted, and the glands stimulated to healthier action by sulphur salves and lotions. If there is much inflammation, the temporary employment of a soothing calamine lotion is necessary.

**Rosacea** or **Acne rosacea** is an affection of middle life, especially of women, and in the latter is much influenced by the catamenia and climacteric. It is, however, almost invariably associated with irritative dyspepsia. The face flushes from any emotion, change of temperature, strong wind, contact of food with the stomach, or sexual excitement, until the flushing becomes almost continuous. The skin gets more and more thickened and inflamed, and acneiform papules and pustules, with dilated veins, form about the cheeks, nose, chin, and other parts.

**TREATMENT.**—If the eruption is very angry, a calamine lotion may be used; but if comparatively quiet, a stimulating sulphur salve or lotion may be applied, to resolve infiltration, and give a better tone to the vessels. We have already suggested the directions in which to look for the selection of internal remedies.

**Seborrhœa.**—When the sebaceous glands themselves are actively inflamed, there is an increased production of sebum, mixed with some inflammatory products. This discharge collects on the surface of the skin, and if there be much stearin or margarin, flakes and crusts form, but if olein be in excess the discharge is more fluid and greasy. Such a condition may extend over a considerable area, such as the face, scalp, or genitals, or be localized in patches. The surface of such areas is generally reddened, and so eczema is simulated, especially if the skin become excoriated. On the scalp the concreted sebum usually adheres to groups of hairs in asbestos-like sheaths, or forms substantial fatty scales on an ashy-grey ground.

**TREATMENT.**—Constitutional remedies (iron and cod-liver oil) are required, as it is found that seborrhœa is more frequent in people of phthisical habit. Locally all exudation must be removed from time to time by alkaline or oily applications, such as an alcoholic solution of soap, or olive oil, and in the intervals mild stimulants (mercurials, sulphur, carbolic oil), or astringents may be inuncted. If there is much inflammation bland applications are called for.

**Milium** is a term used to denote the plugging of the glands whilst the ducts remain free. It occurs as little white papules, the size of pins' heads, about the eyes, and the skin must be pricked with a lancet to remove the contents.

**Molluscum sebaceum vel contagiosum.**—This is a disease of the children of crowded courts and alleys, and more rarely of adults brought in contact with them. From the latter circumstance, and because several children of a street or house or family *may* be affected in succession, it is thought by many to be contagious, though by what agency is obscure. The little growths are sessile or later pedunculated, rounded, with a semi-transparent look, and an aperture on the summit, through which the milky or inspissated contents may be extruded. They grow from the size of pins' points to a large pea or bean, or even much larger, and they may drop off or wither away, or necrose. They come out in crops over a long period; grow very slowly; and tend to disappear spontaneously. In adults they have been of universal distribution in rare cases.



The old idea, from their resemblance to a compound racemose gland, was that they are due to a hyperplasia of the sebaceous glands, but recent investigations point to their origin from an overgrowth of the rete.

**TREATMENT.**—All that is necessary is to thoroughly enucleate the growth with the finger-nails.

**Sycosis** consists in a simple inflammation of the hair-follicles and related parts of the hairy portions of the face, and must be distinguished from the rare tinea sycosis. Pustules are formed around the hairs, and sometimes there is considerable confluence and induration. The disease is very obstinate, and pursues a chronic course. Its causes are obscure, but the subjects of it are usually deteriorated in health.

**TREATMENT.**—The hair should be kept cut close by scissors; all crusts removed; and soothing applications, such as the oleates of zinc and bismuth and calamine lotion, be kept constantly smeared on. Where much induration exists resolvents are sometimes useful. Sometimes benefit results from the extraction of the hairs. Any impairment of the general health must be remedied, and tonics given.

**Xanthelasma** is a disease which was formerly considered to be due to an affection of the sebaceous glands, but now is known to be a chronic inflammation of the cutis, and, therefore, properly to be removed from this section. It is characterized by the formation of circumscribed, smooth, soft streaks or patches (*X. planum*), or papules (*X. papulatum*), or nodules, composed of confluent papules (*X. tuberosum*), presenting a remarkable lemon, cream, or buff-yellow colour. The plane patches look like chamois leather embedded in the skin, but the nodules are firmer, and may project considerably. Clinically there are several phases. *X. palpebrarum* is a fairly common affection of the middle and advanced periods of life, occurring mostly in women, and sometimes running in families. It is limited to the eyelids, and begins as a rule about the left inner canthus, then the right, and eventually involves both lids. The subjects of this complaint suffer often from sick-headaches and functional liver-disturbance. *X. multiplex* is a rarer affection, and here the patches form in the folds and creases of the palms, face, neck, ears, scrotum, penis, soles, abdomen, cleft of nates, and back. The eyelids may be affected or not. Macules have also been found in the mucous membrane of the mouth, lips, tongue, palate, trachea, bile-ducts, &c. This general form has been preceded almost invariably by persistent jaundice from an organic cause, or by diabetes. Several cases are on record, however, in which there was no jaundice, but the disease began in infancy or childhood; and in the case of two brothers and a sister it was congenital, and probably hereditary. The corium is the seat of a chronic inflammatory change, in which the young cells become distended with oil. In chronic cases the cells and intercellular matrix become organized into a new growth of connective tissue, so as to form the tuberoso eruption.

**TREATMENT.**—No satisfactory method of treatment is known, though any very disfiguring patches may be removed by the surgeon.

**Diseases of the hair.**—We meet with an excessive growth, a deficiency in the production, or abnormal falling of the hair, and with the latter condition is usually associated structural degeneration. *Hyper-trichosis*, or excessive growth, may be acquired on naturally hairy parts, or on regions usually free from growth (as the face of women); or be congenital (hairy moles). *Alopecia* signifies all forms of deficiency of hair or baldness, whether congenital or acquired. The baldness of

old age (*A. senilis*), commencing at the junction of the scalp with the forehead, and on the vertex, but never touching the temples, is associated with atrophy of the skin and glands. *Alopecia prematura* comprises a number of conditions, and may be brought about by an ill-nourishment of the hair-papillæ from any general lowering of nutrition, as in syphilis, fevers, the puerperal state, &c. ; or from local disease, such as lupus, morphœa, eczema, psoriasis, and seborrhœa. There is also a common affection (*A. areata vel circumscripta*), seen most frequently in female children, but met with at all ages. The scalp is generally the seat of the loss of hair, though other, especially hairy parts, may be involved, and even the whole body. It is thought to be due to a tropho-neurosis, and commences by the more or less sudden formation of one or several blanched, smooth and glossy, sharply-defined patches, either absolutely bald, or studded, especially about the margins, with a few stumps, which must not be confounded with ring-worm hairs. These stumps are very characteristic, with an atrophied root, and a club-shaped free end. The bald patches may coalesce, recover, and recur, and the affection as a whole pursues a very chronic course. It is seen occasionally in more than one member of a family. *Canities*, or a state in which grey hair is formed, may be hereditary or a senile change, or follow great mental distress, neuralgia and other nerve-lesions; and it is seen in tufts in leucoderma, and in alopecia areata.

**TREATMENT.**—The bald patches of alopecia areata are to be stimulated continually by lotions or ointments, containing either cantharides, rectified spirits, spirit of nutmeg, iodine, capsicum, or similar remedies. Internal treatment is often most unsatisfactory, but nerve tonics are usually given.

**Diseases of the nails.**—The nails become implicated in any general mal-nutrition, and white specks and patches form from imperfect cohesion; also striation and transverse grooving may arise from cessation of growth during fevers, and other causes. The clubbing from persistent pulmonary obstruction has been referred to elsewhere. The nails may be shed in pemphigus, and in post-scarlatinal desquamation. Eruptions may form under the nail in psoriasis, lichen planus, syphilis, struma, and variola, and in these and other diseases, such as eczema, pityriasis rubra, and ichthyosis, where the fold whence the nail grows is implicated, the latter becomes imperfectly formed and loosely constructed, dirty, pitted, striated, and opaque. In peripheral and central nerve-lesions also the nails may shed, or take on an abnormal growth. Fungus may attack a nail, as already described (*onychomycosis*). Apart from all these conditions, there occurs a curious idiopathic affection, which tends to attack all the nails of both hands and feet. The nails get discoloured, pitted, brittle, and disintegrated, and raised at the free end from their beds by a mass of accumulated epithelium. Paronychia, in-growing toe-nail, and hypertrophied toe-nail will be found discussed in surgical works.

## APPENDIX.

---

DURING the progress of this Edition through the press, two therapeutic agents have come into prominent use, which I have deemed of sufficient importance to consider briefly in an *Appendix*.

1. *Antipyrin*. This drug has been studied by several observers, and is now regarded as a valuable *antipyretic*. It is soluble in water, much less bitter than quinine, and without the nauseous taste of kairin. It has been given in doses of gr. xv-xxx every hour, or in a full dose of  $\mathfrak{z}\text{i}$ ; and has also been used subcutaneously. The effects of antipyrin thus administered are to lower the temperature considerably; to diminish the pulse usually; and to cause profuse perspiration. It produces no disagreeable symptoms usually, but vomiting occurs in exceptional instances. The drug has been employed and advocated in various diseases, especially typhus and typhoid fevers, scarlatina, erysipelas, intermittent fever, pleurisy, pneumonia, phthisis, and children's febrile diseases.

2. *Cocaine*. Although known for many years as the alkaloid derived from the leaves of the coca plant (*Erythroxylon Coca*), and although its properties had been to some extent experimentally determined, it is only recently that this agent has suddenly sprung up into the remarkably prominent position which it now occupies, its effects having been first demonstrated by Dr. Koller, of Vienna. Cocaine is chiefly used as the hydrochlorate, and the chief action which it is necessary to notice here is that the drug is a powerful *local anæsthetic*, especially when applied in connection with mucous surfaces. It produces this effect upon the conjunctiva, mouth, tongue, pharynx, larynx, trachea, nose, ear, urethra, rectum, vagina, and uterus; as well as upon ulcers and cut surfaces. Its solutions are but little absorbed by the skin, but hypodermic injection of cocaine produces marked local anæsthesia. Innumerable workers have been experimenting with this alkaloid, and testing its value as a therapeutic agent, but, while recognizing it as a most important addition to our list of remedies, it is at present premature to give a definite statement as to the conditions in which it will be found really useful. Apart from its employment as a local anæsthetic in surgical practice, for the performance of minor operations, and in connection with ophthalmic practice, in which it has proved of the greatest service, in medical cases it has been found useful in relieving painful affections of mucous surfaces; in connection with the throat and larynx, for deadening the sensibility of the parts in making a laryngoscopic examination, for relieving cough and irritation associated with local diseases, such as laryngeal phthisis, and in the removal of growths; in the nasal cavity, in the treatment of acute coryza, polypus, and hay-fever; and for neuralgic affections. A 10 or 20 per cent. solution in oil of cloves, rubbed into the part, is said to produce almost immediate relief in cases of supra-orbital neuralgia. The drug is also used subcutaneously in treating neuralgia. Internally cocaine seems to be chiefly a nerve stimulant, and it rapidly increases and sustains the physical powers, without producing any injurious effects. It has been recommended in cases of great exhaustion, as after loss of blood, sun-stroke, or diarrhœa; as a cure for morphia and alcohol craving; and as a stomachic in atonic dyspepsia and other conditions.



# INDEX.

---

- Abdomen, general and local enlargements of the, 578.  
 — physical examination of the, 575.
- Abdominal aneurism, 724.  
 — perforations and ruptures, 587.  
 — reflex, 804.  
 — typhus. *See* Typhoid fever.  
 — walls, rheumatism of, 250.
- Abrasion, 49.
- Abscess, 48; cerebral, 899; hepatic, 680; pulmonary, 409; renal, 757; retro-pharyngeal, 334.
- Absorbent system, diseases of the, 786.
- Acephalocyst, 684.
- Achorion Schönleini, 991.
- Achromatopsia, 867.
- Acne, 995.
- Acupuncture, 35.
- Addison's disease, 720.
- Adenie, 791.
- Adenitis, 787, 789.
- Adenoid cancer, 299.
- Adherent pericardium, 517.
- Adhesive phlebitis, 571.
- Adynamic fever, 82.
- Æsthesiometer, 803.
- Ætiology, 2, 4.
- Aglobulism, 562.
- Ague, 225; symp., 225; description of a fit, *ib.*; temp., 226; urine, *ib.*; varieties, 227; treat., *ib.*
- Albumen, tests for, 730.
- Albuminoid disease, 74.  
 — of arteries, 553.  
 — of glands, 793.  
 — of intestines, 648.  
 — kidney, 773.  
 — liver, 683.  
 — spleen, 715.
- Albuminuria, 746.
- Alcoholism, 881.
- Algide cholera, 194.
- Alopecia, 995.
- Alveolar cancer, 298.
- Amaurosis in lead-poisoning, 887.
- Amnesia, 841.
- Amœboid leucocytes, 45.
- Amphoric breathing, 362.  
 — echo, 368.  
 — râles, 365.  
 — resonance, 367.
- Amygdalitis, 327.
- Amyloid degeneration, 74.
- Anæmia, 12, 563.  
 — cerebral, 905; treat., 914.  
 — of the disc, 808.  
 — of the pancreas, 718.  
 — spinal, 935.
- Anæsthesia, 801, 834.
- Analgesia, 801.
- Anasarca, 28.
- Anatomy, morbid or pathological, 2.
- Aneurism, abdominal, 724.  
 — of arteries, 553.  
 — cardiac, 539.  
 — of brain, 921.  
 — thoracic, 555.
- Angioloecitis, 787.
- Angina pectoris, 504; pseudo, 507.  
 — maligna, 330.  
 — simplex, 326.
- Anidrosis, 993.
- Ankle clonus, 804, 833.
- Anorexia, 593.
- Anthrax, 207.
- Antipyrin, 998.
- Anuria, 727.
- Aortic aneurism, thoracic, 555; abdominal, 724.  
 — murmurs, 495.  
 — obstruction, 526.  
 — regurgitation, 527.
- Aortitis, 551.
- Aphasia, 801, 841.
- Aphemia, 841.
- Aphonia, hysterical, 390.
- Apneumatosis, 432.
- Apnoea, 13, 369.
- Apoplectic diseases, 906; diag. of, 914; prog., 916; treat., *ib.*  
 — fit or seizure, 817.
- Apoplexy, 35, pulmonary, 404; cerebral, 904; spinal, 936.
- Apthous stomatitis, 316.
- Appetite, disorders of, 593.
- Arteries, clinical phenomena connected with, 481; diseases of, 551; exam. of, 498.  
 — aneurism of, 553.  
 — calcification of, 553.  
 — fatty degeneration of, 553.  
 — thrombosis of, 572.
- Arteritis, 551.
- Arthritis deformans, 252.  
 — senile, 248.
- Articular gout, 255.  
 — rheumatism, acute, 240; chronic, 248.
- Ascaris lumbricoides, 660.  
 — mystax, 657.
- Ascites, 28, 589; symp., *ib.*; phys. signs, 590; treat., 592.
- Asiatic cholera, 194.
- Asphyxia, 13, 370.
- Aspirateur, use of the, 470.
- Asthenia, 13.
- Asthenic fever, 82.

- Asthenic ague, 227.  
 Asthma, 426.  
   — bronchial, 427.  
   — cardiac, 479.  
   — diaphragmatic, 431.  
   — humid, 428.  
   — spasmodic, 427; symp., 428; diag., 429; treat., *ib.*  
 Atavism, 7.  
 Ataxic fever, 82.  
 Atelectasis, 432.  
 Atheroma, 552.  
 Atonic dyspepsia, 605.  
 Atrophy, 62.  
   — of arteries, 553.  
   — of the heart, 535.  
   — of the liver, 682; acute yellow, 681; chronic, 693.  
   — of the optic disc, 810.  
   — of the pancreas, 718.  
   — of skin, 987.  
   — progressive muscular, 956.  
 Attrition sounds, 366.  
 Auditory vertigo, 814.  
 Aura epileptica, 859.  
   — hysterica, 867.  
 Auscultation, 348.  
   — of the heart, 484, 489.  
   — of the respiratory organs, 360.  
  
 Bacillus anthracis, 100, 207.  
   — of cholera, 194.  
   — tuberculosis, 294.  
 Bacterium lactis, 99.  
 Basedow's disease, 796.  
 Basic impulse, 488.  
 Bell's palsy, 828.  
 Bell-sound, 368.  
 Bile-ducts, inflammation of, 679.  
 Bilharzia hæmatobia, 752.  
 Biliary calculi, 676; colic, 699.  
 Bladder, distended, 745.  
   — inflammation of, 785.  
   — worm, 657.  
 Blood, abnormal conditions of the, 561.  
   — determination of, 44.  
 Blood-concretions, intra-cardiac, 569.  
 Blood-letting, 58.  
 Blowing breathing, 362.  
 Bloody flux, 637.  
 Blue disease, 549.  
 Borborygmi, 594.  
 Bothriocephalus cordatus, 657.  
   — — latus, 659.  
 Brain, diseases of, clinical phenomena of, 848.  
   — and membranes, acute inflammations of the, 893.  
   — — adventitious growths in connection with, 920.  
   — cancer of, 920.  
   — chronic diseases of the, 918; diag., prog., and treat., 924.  
   — hypertrophy of, 924.  
   — sarcoma of, 921.  
   — sclerosis of, 943.  
   — syphilitic disease of, 921.  
  
 Brain, tubercle of, 920.  
 Breakbone fever, 213.  
 Breath-sounds, 360.  
 Bright's disease, acute, 761; treat., 765; chronic, 766; diag., prog., and treat., 774.  
   — changes in arteries in, 553.  
 Bronchi, diseases of the, 393.  
   — dilatation of, 402.  
   — inflammation of, 393.  
 Bronchial asthma, 427.  
   — breathing, 360.  
   — catarrh, acute, 393; chronic, 399.  
   — phthisis, 793.  
 Bronchiectasis, 402.  
 Bronchitis, acute catarrhal, 393; symp., 394; diag., 397, 474; treat., 397.  
   — chronic, 399; phys. signs, 400; treat., 401.  
   — capillary, 395.  
   — epidemic, 396.  
   — mechanical, 396.  
   — plastic or croupous, 402.  
   — primary, 394.  
   — secondary, 396.  
 Bronchocele, 795.  
 Bronchophony, 367.  
 Bronchorrhœa, 400.  
 Broncho-pneumonia, 414.  
 Brow-ague, 838.  
 "Bruit de pot félé," 358.  
 "Buffy-coat," 57.  
 Bulbar paralysis, 958.  
 Bulimia, 593.  
 Bullous inflammations of skin, 975.  
  
 Cachexia, 77.  
 Calcareous degeneration, 69.  
 Calcification, 69; of arteries, 553; of tubercle, 290.  
 Calculus, biliary, 697.  
   — urinary, 781.  
 Cancer, 298; anat. char., *ib.*; structure, 299; path., 301; symp., 302.  
   — of the brain, 920.  
   — of the gall-bladder, 697.  
   — of the glands, 793.  
   — of the intestines, 646.  
   — of the kidney, 777.  
   — of the liver, 688.  
   — of the lungs, 458.  
   — of the œsophagus, 339.  
   — of the peritoneum, 586.  
   — of the spleen, 715.  
   — of the stomach, 614.  
 Cancroid, 299.  
 Cancrum oris, 317.  
 Capillary bronchitis, 395.  
   — congestion, 26.  
 Carcinoma, 298.  
 Cardiac affections, chronic, general diag. of, 540; prog., 541; treat., 543.  
   — action, disordered, 509.  
   — aneurism, 539.  
   — asthma, 479.  
   — dyspnoea, 479.  
   — enlargement, 529.

- Cardiac impulse, 486.  
 — movements, 485.  
 — murmurs, 493.  
 — percussion, 488.  
 — region, changes in, 485.  
 — — peculiar sensations over, 488.  
 — sounds, 489.  
 — thrombosis, 569.  
 Cardialgia, 593.  
 Cardiogram, 485.  
 Cardiograph, the, 484, 485.  
 Cardio-sphygmograph, 484.  
 Carnification, 432.  
 Caseation, 67 ; of tubercle, 290.  
 Case-taking, method of, 13.  
 Casts, 742.  
 Catalepsy, 870.  
 Catarrh, 374.  
 — bronchial, acute, 393 ; chronic, 399.  
 — dry, 400.  
 — enteric, 633.  
 — epidemic, 191.  
 — gastric, 607, 611.  
 — intestinal, 633.  
 — — chronic, 633.  
 — laryngeal, acute, 379 ; chronic, 384.  
 — pharyngeal, acute, 326 ; chronic, 332.  
 — vesical, 785.  
 Catarrhal inflammation, 51 ; of skin, 972.  
 — bronchitis, 393.  
 — laryngitis, 376, 379.  
 — nephritis, 756.  
 — pharyngitis, 326.  
 — pneumonia, 414 ; diag., 474.  
 — stomatitis, 314.  
 — tracheitis, 376, 379.  
 Causation of disease, 4.  
 Cavernous breathing, 362.  
 — râles, 365.  
 Cell-proliferation, 46.  
 Cephalalgia, 811.  
 Cephalodynia, 249.  
 Cerebral abscess, 899.  
 — anæmia, 905.  
 — congestion or hyperæmia, 903.  
 — diseases, chronic, diag., 924 ; prog., 925 ; treat., *ib.*  
 — embolism and thrombosis, 910.  
 — hæmorrhage, 906 ; symp., 907.  
 — inflammations, acute, 899 ; diag., prog., and treat., 900.  
 — sclerosis, 943.  
 — softening, 910 ; chronic, 919.  
 — tumour, 920.  
 — vomiting, 598.  
 Cerebritis, acute, 899 ; treat., 902.  
 Cerebro-spinal meningitis, epidemic, 219 ; diag., 239.  
 — — sclerosis, 952.  
 Cervical paraplegia, 872.  
 Cestodes, description of, 657.  
 Charbon, 207.  
 Cheesy degeneration, 67.  
 Chest, physical examination of, 344.  
 — shape of, 349.  
 — deformities of, 350.  
 Chest-walls, rheumatism of, 250.  
 Cheyne-Stokes respiration, 479.  
 Chicken-pox, 169.  
 Child-crowing, 388.  
 Chloasma uterinum, 989.  
 Chlorosis, 563.  
 Cholagogues, 704.  
 Cholelithiasis, 697.  
 Cholera, Asiatic, algide, or malignant, 194 ; anat. char., 195 ; symp., 196 ; comp. and seq., 199 ; var., *ib.* ; path., *ib.* ; prog., 201 ; treat., *ib.*  
 — diag. of, 238.  
 — morbus, 194.  
 Choleraic diarrhoea, 200.  
 Choline, 200.  
 Cholesteatoma of brain, 921.  
 Chondroid cancer, 299.  
 — tissue, 273.  
 Chorea, 873.  
 Chronic ulcer of stomach, 611.  
 Chylous urine, 740, 748.  
 Chyluria, 748.  
 Circulatory organs, diseases of the, clinical characters, 478 ; physical exam. of, 484.  
 Cirrhosis of the liver, 690.  
 — of lung, 416.  
 Cirrhotic kidney, 770.  
 Clavus hystericus, 839.  
 Clergyman's sore throat, 326, 385.  
 Clinical examination, 13.  
 — history, 2.  
 — investigation of febrile diseases, 106.  
 Cocaine, 998.  
 Cœcum, inflammation of, 633, 635.  
 Cold, a, 374.  
 Colic, biliary or hepatic, 699.  
 — intestinal, 626.  
 — nephritic, 783.  
 Colitis, 634.  
 Colloid cancer, 298.  
 — degeneration, 73.  
 Colloid in the omentum, 587.  
 Coma, 13, 816.  
 Comedo, 994.  
 Complications, 15.  
 Concussion of spinal cord, 927.  
 Congenital diseases, 7.  
 — syphilis, 284.  
 Congestion, 24 ; active, *ib.* ; mechanical or venous, 25 ; passive or capillary, 26.  
 — cerebral, 903.  
 — laryngeal, 384.  
 — of the liver, 674.  
 — of the spleen, 709.  
 — pulmonary, 403.  
 — renal, 756.  
 — spinal, 935.  
 Consciousness, disorders of, 815.  
 Constipation, 827 ; symp., 628 ; treat., *ib.*  
 Constitutional diseases, 77, 240.  
 — syphilis, 281.  
 Consumption, pulmonary, 434.  
 Contagion, 94 ; origin, modes of propagation, &c., 95 ; nature of, 98 ; effects of, 100 ; elimination of, 102.  
 Contagious fevers, hygienic treatment of, 104.



- Continued fever, 81, 113.  
 — type of fever, 81.  
 Convulsions, 820.  
 Cord, diseases of. *See* Spinal cord.  
 Corn, 985.  
 Cornification, 291.  
 Coryza, 374.  
 Cough, 342, 371.  
 Coup de soleil, 892.  
 Cowpox, 165.  
 Cramp of the stomach, 597.  
 Cremasteric reflex, 804.  
 Crepitant râles, 364.  
 Crisis, 80.  
 Croup, spasmodic, 388; spurious, 377, 388; treat., 389.  
 — true, 380; membranous, *ib.*; treat., 382.  
 Croupous bronchitis, 402.  
 — inflammation, 51.  
 — laryngitis, 377, 380.  
 — pneumonia, 407; diag. of, 474.  
 — stomatitis, 316.  
 Cruveilhier's paralysis, 956.  
 Cry fremitus, 355; resonance, 366.  
 Crystalline pock, 160.  
 Cutaneous erysipelas, 173.  
 — inflammation, 50.  
 Cyanosis, 549.  
 Cynanche laryngea, 379.  
 — maligna, 330.  
 — pharyngea, 326.  
 — tonsillaris, 327.  
 — trachealis, 380.  
 Cystic cancer, 299.  
 — disease of kidney, 779.  
 Cysticercus, 657.  
 — of brain, 920.  
 Cystine, 740.  
 Cystitis, 785.  
 Dandy fever, 213.  
 Defervescence, 80.  
 Degeneration, fatty, 65; caseous, 67; mineral, 69; fibroid, 71; pigmentary, *ib.*; mucoid, 73; colloid, *ib.*; albuminoid, 74.  
 Degenerative reactions, 806.  
 Delirium, 815.  
 — tremens, 882.  
 Delitescence, 47.  
 Dengue, 213; diag., 239.  
 Derbyshire neck, 795.  
 Dermatitis, 973.  
 Dermatolysis, 986.  
 Dermatophytic affections, 991; dermatozoa ditto, 990.  
 Descending degeneration, 951.  
 Descending optic neuritis, 809.  
 Desquamative nephritis, acute, 761.  
 — chronic, 768.  
 Determination of blood, 24.  
 Diabetes insipidus, 312.  
 — mellitus, 303; symp., 307; diag., 309; treat., 310.  
 Diagnosis, 2; general remarks on, 17.  
 — of acute specific fevers, 231.  
 Diagnostic table of principal fevers, 233.  
 Diapedesis, 45.  
 Diaphragmatic asthma, 431.  
 Diarrhœa, 609; summer, 200.  
 Diathesis, 77.  
 Diathetic diseases of skin, 981.  
 Dietetic treatment, 22.  
 Digitalis in cardiac diseases, 546.  
 Dilatation of bronchi, 402.  
 — of œsophagus, 339.  
 — of stomach, 616.  
 Diphtheria, 175; symp., 178; forms of, *ib.*; complications, 180; treat., 181; diag., 238.  
 Diphtheritic inflammation, 51.  
 — laryngitis, 377.  
 — paralysis, 889.  
 — stomatitis, 316.  
 Disc, optic, ophthalmoscopic appearances of, 808.  
 Disease, causation of, 2, 4; definition, 1; mode of invasion, course, &c., 11.  
 Diseases, nomenclature of, 77.  
 Disseminated paralysis, 799.  
 — pneumonia, 414.  
 — sclerosis, 952.  
 Distoma crassum, 657.  
 — hepaticum, 669.  
 — heterophyes, 657.  
 Dochmius duodenalis, 657.  
 Dorsodynia, 250.  
 Dropsical fluid, 29.  
 Dropsy, 28; spurious, *ib.*; path. conditions producing, 29; symp., 31; diag., *ib.*; orig. seat, and extent, 32; treat., 32.  
 — of the gall-bladder, 696.  
 — of kidney, 780.  
 — ovarian, 28, 591.  
 — of the peritoneum, 589.  
 — of the pleura, 471.  
 — pericardial, 518.  
 Duchenne's paralysis, 961.  
 Duodenitis, 633.  
 Dynamometer, 803.  
 Dysæsthesia, 801, 836.  
 Dysentery, 637; symp., 640; treat., 642.  
 Dysidrosis, 994.  
 Dyspepsia, 603; symp., 604; varieties, 605; diag., 618; treat., 605, 621.  
 — acute, 605.  
 — atonic, 605.  
 — chronic, 605.  
 — irritative, 606.  
 — nervous, 606.  
 Dyspnoea, 369; inspiratory, 354; cardiac, 479.  
 Dysuria, 726.  
 Ecchymosis, 35.  
 Echinococcus hominis, 684.  
 Eclampsia, 821.  
 Ecstasy, 870.  
 Ecthyma, 977.  
 Eczema, 972.  
 Effusion, 47.  
 Electricity, uses of, 804, 832.  
 Elephantiasis, 749.

Elephantiasis arabum, 969.  
 Emboli, origin of, and anatomical changes resulting from, 572.  
 Embolic abscess, 574.  
 Embolism, 568, 572.  
 — cerebral, 910.  
 — renal, 756.  
 — splenic, 709.  
 Emesis, 598.  
 Emphysema, interstitial or interlobular, 426.  
 — of lungs, 420.  
 — vesicular, 420; anat. char., 422; symp., 424; phys. signs, *ib.*; treat., 425.  
 Empyæma, 463.  
 Encephalitis, 899.  
 Encephaloid cancer, 298.  
 Enderteritis deformans, 551.  
 Endemic disease, 102.  
 Endocardial murmurs, 484, 493.  
 Endocarditis, acute, 519; treat., 521.  
 — chronic, 523.  
 English cholera, 200.  
 Enlarged liver, 700.  
 Enteralgia, 625.  
 Enteric catarrh, 633.  
 — fever, 122.  
 Enteritis, 633.  
 Epidemic catarrh, 191.  
 — cerebro-spinal meningitis, 219.  
 — cholera, 195.  
 — roseola, 152.  
 Epidemics, 102; laws of, 103.  
 — prevention of, 104.  
 Epigastric impulse, 488.  
 — reflex, 804.  
 Epilepsy 857; symp., 858; stages of attack, 859; treat., 862.  
 — gravior, 859; mitior, 858.  
 Epileptiform hysteria, 869.  
 Epistaxis, 35.  
 Epithelioma—epithelial cancer, 299.  
 Equinia, 204.  
 — mitis, 206.  
 Erratic ague, 227.  
 — erysipelas, 173.  
 Eructation, 594.  
 Erysipelas, 170; symp., 171; var., 173; treat., 174.  
 — diagnosis of, 235, 237.  
 Erythema, 970.  
 Erythematous inflammations, 970.  
 Essential fever, 78.  
 — paralysis of children, 932.  
 Etiology. *See* Ætiology.  
 Examination, physical, general remarks on, 14.  
 Examination of patients, mode of, &c., 13.  
 Exanthemata, acute, 78.  
 Excoriation, 49.  
 Exophthalmic goitre, 796.  
 Expectoration, 342.  
 Extravasation, 35.  
 Exudation, 47.  
 Eye, paralysis of, 829.

Facial erysipelas, 173.  
 — paralysis, 828.  
 Fainting, 507.  
 Falling sickness, 857.  
 Famine fever. *See* Relapsing fever.  
 Farcy, 204; diag., 238.  
 — buttons and buds, 206.  
 Fatty degeneration, 65.  
 — of heart, 536.  
 — of arteries, 553.  
 — disease of the heart, 535, 536.  
 — infiltration, 68.  
 — of heart, 535.  
 — kidney, 772.  
 — liver, 683.  
 — pancreas, 718.  
 — metamorphosis, 65; of heart, 536.  
 Febricula, 113; diag. of, 233.  
 Febrile diseases, clinical investigation of acute, 106.  
 Ferment, 98.  
 Fever, 78; types, 81; path., 83; prog., 87; treat., *ib.*  
 — cerebro-spinal, 219.  
 — continued, 81.  
 — dandy, 213.  
 — intermittent, 225.  
 — malarial, 222.  
 — relapsing, 135.  
 — remittent, 229.  
 — rheumatic, 240.  
 — scarlet, 140.  
 — typhoid, 122.  
 — typhus, 114.  
 Fevers, diagnostic table of, 233.  
 Fibrinous exudation, 47.  
 Fibroma, 986.  
 Fibroid degeneration, 71, 416.  
 — phthisis, 416.  
 Filaria sanguinis hominis, 748.  
 Flux, bloody, 637.  
 Follicular laryngitis, 385.  
 — pharyngitis, 327.  
 — sore-throat, 330.  
 — stomatitis, 314.  
 Fomites, 96.  
 Fremitus, 355.  
 Friction-fremitus, 356.  
 — sounds, 366.  
 Front-tap contraction, 804.  
 Fungous stomatitis, 317.  
 Fungus hæmatodes, 298.  
 Gall-bladder, affections of the, 696.  
 — stones, 697; symp., 699; treat., 707.  
 Galloping consumption, 439.  
 Gangrene, 49.  
 — of the lung, 418.  
 Gangrenous erysipelas, 173.  
 — stomatitis, 317.  
 Gastralgia, 596.  
 Gastric affections, chronic, diag., 618; prog., 620; treat., 621.  
 — cancer, 614.  
 — catarrh, acute, 607; anat. char., *ib.* symp., 608; treat., 609.  
 — — chronic, 610.

- Gastric fever, 131.  
   — ulcer, 611.  
 Gastritis, acute, 607.  
   — chronic, 610.  
 Gastrodynia, 596.  
 Gastrorrhœa, 611.  
 Gelatiniform cancer, 298.  
 General paralysis, 824.  
 Germ theory of disease, 98.  
 German measles, 152.  
 Germination of cells, 46.  
 Giddiness, 812.  
 Gin-drinker's liver, 691.  
 Glanders and farcy, 204; diag., 238.  
 Glands of skin, affections of, 993.  
   — absorbent, diseases of, 786; treat., 794.  
 Glandular hypertrophy, 791.  
   — laryngitis, 385.  
 Glioma of brain, 921.  
 Glomerulo-nephritis, 141, 763.  
 Glossitis, 320; chronic, 321.  
 Glosso-labio-laryngeal paralysis, 958.  
 Glottis, œdema of, 387.  
   — spasm of, 388.  
 Gluteal reflex, 804.  
 Glycosuria, 303.  
 Gmelin's test, 736.  
 Goitre, 795; exophthalmic, 796.  
 Gonorrhœal rheumatism, 251.  
 Gout, 253; acute, 255; chronic, 257; retrocedent, *ib.*; diag., 259; treat., 260.  
 Granular kidney, 770.  
   — liver, 691.  
   — sore-throat, 332.  
 Gravel, 781.  
 Graves's disease, 796.  
 Great vessels, malformations of the, 549.  
 Grey granulations, 288.  
 Gummata, 282.  
 Gummatous hepatitis, 695.  
 Gyngivitis ulcerosa, 316.  
  
 Hæmacytometer, 713.  
 Hæmatemesis, 36, 594, 601; treat., 602.  
 Hæmatinuria, 753.  
 Hæmatoidine, 72.  
 Hæmatoma, in lungs, 462.  
 Hæmaturia, 36, 751.  
 Hæmophilia, 40.  
 Hæmoptysis, 35, 342, 372.  
 Hæmorrhage, 35; ætiol., 36; classes of, *ib.*; anat. char., *ib.*; symp., 37; treat., 39.  
   — cerebral and meningeal, 906.  
   — into the lungs, 404.  
   — into the pleura, 472.  
   — intestinal, 632.  
   — of pancreas, 718.  
   — pericardial, 518.  
   — pulmonary, 403.  
   — spinal, 936.  
   — ventricular, 909.  
 Hæmorrhagic diathesis, 40.  
   — infarct, 35, 573.  
   — infarction of spleen, 709.  
 Hæmothorax, 472.  
 Hair-headed worm, 657.  
  
 Hassall's test, 735.  
 Haut mal, 859.  
 Hay asthma, 429, 431.  
 Headache, 811.  
 Heart, acute inflammations of, 519.  
   — atrophy of, 535; brown, 72.  
   — auscultation of, 489.  
   — chronic affections of, 523; diag., 540; prog., 541; treat., 543.  
   — clinical phenomena connected with, 478.  
   — degenerations and new formations in the walls of, 538.  
   — dilatation of, 529.  
   — enlargements of the, 529; symp., 532; phys. signs, 533.  
   — examination of, 485.  
   — fatty disease of, 535.  
   — gouty, 536.  
   — hypertrophy of, 529.  
   — malformations of, 549.  
   — palpitation of, 509.  
   — parenchymatous degeneration of, 522.  
   — rupture of, 539.  
   — sounds of, 484, 489.  
   — valves, &c., of, affections of, 523.  
 Heart-burn, 593.  
 Hectic type of fever, 81, 83.  
 Helminthiasis, 656.  
 Hemi-anæsthesia, 835.  
 Hemichorea, 875.  
 Hemicrania, 855.  
 Hemiplegia, 824, 825.  
 Hepar adiposum, 683.  
 Hepatalgia, 669.  
 Hepatic abscess, 655; treat., 680.  
   — affections, chronic, general diag., 700; prog., 703; treat., *ib.*  
   — colic, 699.  
   — congestion, 675.  
   — enlargements, characters of, 701.  
 Hereditary disease, 6.  
 Hernia, 649.  
 Herpes, 975.  
 Hip-gout, 839.  
 Hippocratic succussion, 349.  
 Histology, morbid, 2.  
 Hob-nailed liver, 691.  
 Hooping-cough, 187; diag., 238.  
 Horn-pock, 160.  
 Hodgkin's disease, 711, 791.  
 Hydatid fremitus, 576.  
   — tumour of the liver, 684; anat. char., 685; symp., 687; treat., 705.  
   — tumour of brain, 920.  
   — — of kidney, 778.  
   — — of lung, 462.  
   — — of peritoneum, 586.  
   — — of spleen, 715.  
 Hydræmia, 562.  
 Hydrocele, 28.  
 Hydrocephalus, 28; acute, 896; chronic, 923.  
 Hydrometria, 28.  
 Hydronephrosis, 28; 780.  
 Hydropericardium, 28, 518.  
 Hydrophobia, 210; diag., 238.



- Hydro-pneumothorax, 472.  
 Hydrops, 28.  
   — vesicæ felleæ, 696.  
 Hydrothorax, 28, 471.  
 Hygienic treatment, 22.  
   — of contagious fevers, 104.  
 Hypæmia, 562.  
 Hypæsthesia, 801, 834.  
 Hypalbuminosis, 562.  
 Hyperæmia, 24, 562.  
   — cerebral, 903.  
   — of the optic disc, 808.  
   — of the liver, 674.  
   — of the lungs, 403.  
   — of the spleen, 709.  
   — renal, 756.  
 Hyperæsthesia, 801, 836.  
 Hyperalbuminosis, 562.  
 Hyperidrosis, 993.  
 Hyperinosis, 562.  
 Hyperplasia, 61.  
 Hyperpyrexia, 79.  
 Hypertrophy, 61; fatty, 68.  
   — of the brain, 924.  
   — of the glands, 791.  
   — of the heart, 529.  
   — of the liver, 682.  
   — of the pancreas, 718.  
   — of the spleen, 710.  
   — of the skin, 985.  
 Hypnosis, 562.  
 Hypochondriasis, 872.  
 Hysteria, &c., 864; symp., 865; charac-  
   ters of, *ib.*; treat., 871.  
 Hystero-epilepsy, 870.  
  
 Icterus, 669.  
 Ichthyosis, 985.  
 Idiopathic fever, 77.  
   — bronchitis, 394.  
   — parotitis, 186.  
 Idiosyncrasy, 6.  
 Impetigo, 973; contagiosa, 977.  
 Indigestion, 603.  
 Indigo-carmin test for sugar, 735.  
 Induration, 49.  
 Infantile paralysis, 932.  
 Infarction, hæmorrhagic, 573; of spleen,  
   709; renal, 756.  
 Infection, 96.  
 Inflammation, 42; changes in blood-  
   vessels, &c., 44; do. in affected tissues,  
   46; path. terminations and products,  
   47; gen. morbid anat., 49; path., 52;  
   symp., 56; varieties, 57; treat., 58.  
   — of arteries, 551.  
   — of the bile-ducts, 679; treat., *ib.*  
   — of the bladder, 785.  
   — of the brain and its membranes, 893;  
   diag., prog., and treat., 900.  
   — of the bronchi, 393.  
   — of the gall-bladder, 696.  
   — of the glands, 787.  
   — of the heart, 522.  
   — of the intestines, 633.  
   — of the kidney, 758.  
   — of the larynx and trachea, 376.  
  
 Inflammation of the liver, 676; treat., 680.  
   — of the lungs, 407.  
   — of the mouth, 314.  
   — of the mucous membrane, 51.  
   — of the œsophagus, 336.  
   — of the pancreas, 718.  
   — of the pericardium, 512.  
   — of the peritoneum, 580.  
   — of the pleura, 462.  
   — of the skin, 970.  
   — of the spinal cord and its membranes,  
   928, 930; diag., 941; prog., 942; treat., *ib.*  
   — of the spleen, 709.  
   — of the stomach, 607.  
   — of the throat, 325.  
   — of the tongue, 321.  
   — suppurative, 48.  
 Inflammatory diseases of brain, acute, 893;  
   general diagnosis of, 900.  
   — exudation, 47.  
 Influenza, 191; symp., 192; diag., 238;  
   treat., 193.  
 Inoculated syphilis, 281.  
 Inoculation, 96; of vaccine-lymph, 165.  
 Inorganic murmurs, 496.  
 Insensibility, 816.  
 Insolation, 892.  
 Insomnia, 819.  
 Intercostal neuralgia, 839.  
 Interlobular emphysema, 426.  
 Intermittent fever, 225; diag., 239.  
   — type of fever, 81.  
 Interstitial absorption, 49; emphysema,  
   426; hepatitis, 690; nephritis, chronic,  
   770; pneumonia, 416.  
 Intestines, accumulations in, 649, 654.  
   — albuminoid disease of, 648.  
   — cancer of, 646.  
   — compression of, 649, 654.  
   — constriction of, 649.  
   — incarceration of, 649.  
   — inflammation of, 633.  
   — intus-susception of, 650, 654.  
   — invagination of, 650.  
   — perforation of, 587.  
   — rotation of, 650.  
   — scrofulous disease of, 647.  
   — strangulation of, 649, 654.  
   — stricture of, 649, 654.  
   — torsion of, 650.  
   — tubercle of, 647.  
   — ulcers of, 644.  
 Intestinal catarrh, 633.  
   — colic, 626.  
   — hæmorrhage, 632.  
   — new formations and ulcerations, 644.  
   — obstruction, 649; anat. char., 650;  
   symp., 652; diag., 653; treat., 655.  
   — perforation, 126.  
   — worms, 656; description, 658;  
   treat., 664.  
 Introduction, 1.  
 Intus-susception, 650.  
 Invagination, 650.  
 Irregular paralysis, 827.  
 Irritative dyspepsia, 606.  
 Ischæmia of the optic disc, 809.

- Ischuria, 727.  
 Itch, the, 991.  
 Itching, 990.  
  
 Jaundice, 669; anat. char., 671; symp., *ib.*; diag., 673; prog., *ib.*; treat., 674.  
 Johnson's test for albumen, 732; for sugar, 735.  
  
 Keloid, 985.  
 Kidney, cancerous growths in, 777; diag., prog., and treat., 780.  
   — cirrhotic, 770.  
   — congestion of, 756.  
   — cystic disease of, 779; treat., 781.  
   — dropsy of, 780.  
   — fatty, 772.  
   — granular, contracted, 770.  
   — horse-shoe, 745.  
   — hydatid disease of, 744.  
   — lardaceous or albuminoid, 773.  
   — large, white, smooth, 768.  
   — movable or floating, 744.  
   — parasitic growths in, 778.  
   — pelvis of, inflammation of, 758.  
   — suppurative inflammation of, 757; general diag., prog., and treat., 760.  
   — tubercular disease of, 778; treat., 780.  
   — tumour, 744.  
 Kiesteine, 740.  
 Knee-jerk, 804, 833.  
  
 Landry's paralysis, 934.  
 Lardaceous disease, 74.  
   — — of the glands, 793.  
   — kidney, 773.  
   — liver, 683.  
   — spleen, 715.  
 Laryngeal affections, chronic, diagnosis, treatment, &c., of, 391.  
   — catarrh, 379; chronic, 384.  
   — paralysis, 390.  
   — perichondritis, 388.  
   — sounds, 356.  
 Laryngismus stridulus, 377, 388.  
 Laryngitis, acute, 376.  
   — acute catarrhal, 379.  
   — chronic, 384.  
   — croupous or plastic, 377, 380.  
   — oedematous, 376, 380.  
 Laryngoscope, uses of, 343.  
 Larynx, abscess of, 388.  
   — affections of, 376.  
   — examination of, 342.  
   — functional or nervous affections of, 388.  
   — morbid growths in, 387.  
   — necrosis of the cartilages of, 388.  
   — ulcers of, 386.  
 Lateral sclerosis, 949, 950, 951.  
 Lead-poisoning, 886.  
 Leptomenigitis spinalis, 928, 937.  
 Lesions in nervous diseases, 844.  
 Leucocytes, 45.  
 Leucocythæmia, 562, 711.  
 Leucocytosis, 562.  
  
 Leucoderma or Leucopathia, 989.  
 Leukæmia, 711.  
 Lichen, 978.  
 Liquor puris, 48.  
 Liver, acute inflammatory diseases of the, 676; diag., prog., and treat., 679.  
   — affections, chronic, general diag. of, 700; prog., 703; treat., *ib.*  
   — albuminoid disease of, 683.  
   — and appendages, diseases of, clinical characters, 667.  
   — atrophy of, 682, 692.  
   — cancerous growths of, 688.  
   — chronic diseases of, 682.  
   — cirrhosis of, 691.  
   — congestion of, 674.  
   — fatty, 683.  
   — hydatid tumour of, 684.  
   — hypertrophy of, 682.  
   — inflammation of, 676.  
   — lardaceous, 683.  
   — rickety, 277, 696.  
   — syphilitic, 695.  
   — tuberculosis of, 696.  
 Liver-fluke, 669.  
 Lobar pneumonia, 407.  
 Lobular pneumonia, 414.  
 Local diseases, 314.  
   — paralysis, 827.  
 Lock-jaw, 878.  
 Locomotor ataxy, 945.  
 Lumbago, 250.  
 Lung, brown induration of, 406.  
 Lungs, cancer of, 458.  
   — cirrhosis of, 416.  
   — emphysema of, 420.  
   — gangrene of, 418.  
   — hæmorrhage into, 404.  
   — hyperæmia of, 403.  
   — inflammation of, 407.  
   — morbid formations in, 458, 462.  
   — cedema of, 403.  
   — syphilitic disease of, 460.  
   — and pleuræ, general diagnosis of affections of the, 474.  
 Lupus, 988.  
 Lymph, 47.  
 Lymphadenoma, 791.  
 Lymphadenosis, 791.  
 Lymphangiectasis, 789.  
 Lymphangiectodes, 789.  
 Lymphangitis, 787.  
 Lymphangioma, 789.  
 Lymphatic dilatation, 789.  
   — leucocythæmia, 711.  
 Lymphorrhagia, 790.  
 Lymphorrhœa, 764, 790.  
 Lysis, 81.  
  
 Maculæ, 966.  
 Malaria or marsh-miasm, 222; origin and propagation, *ib.*; nature, 224; mode of entrance, *ib.*  
 Malignant disease, 298; of lungs, 458.  
   — pustule, 207; diag., 238.  
 Malignant type of fever, 82.  
 Marasmus, 63.

Maxillary nerve, paralysis of, 830.  
 Measles, 148; symp., *ib.*; varieties, 150;  
   treat., 151.  
   — diag. of, 235, 237.  
 Mediastinal tumours, 560.  
 Medullary cancer, 298.  
 Melæna, 36, 594, 632.  
 Melanæmia, 72, 563.  
 Melanin, 72.  
 Melanoderma, 989.  
 Melanotic cancer, 299.  
 Melasma, 989.  
 Membranous laryngitis, 377, 380.  
 Menière's disease, 812, 814.  
 Meningeal hæmorrhage, 906.  
 Meningitis, cerebral, simple or primary, 893.  
   — cerebro-spinal, 219.  
   — chronic, 918.  
   — rheumatic, 899.  
   — spinal, 928, 937.  
   — tubercular, 896.  
 Menorrhagia, 36.  
 Mensuration of chest, 347.  
 Mercurial poisoning, 889.  
   — stomatitis, 318.  
   — tremors, 889.  
 Metallic poisoning, 886.  
   — tinkling, 368.  
 Metamorphosis, 65; fatty, *ib.*  
 Metastasis, 47.  
 Metastatic erysipelas, 173.  
 Miasmatic diseases, 102.  
 Microsporon furfur, 992.  
 Migraine or megrim, 855.  
 Migration of blood corpuscles, 45.  
 Miliaria, 993.  
 Miliary erysipelas, 173.  
   — tubercle, 288.  
 Milium, 995.  
 Mineral degeneration, 69.  
 Mitral murmurs, 494.  
   — obstruction, 525.  
   — regurgitation, 524.  
 Mogigraphia, 959.  
 Molluscum contagiosum vel sebaceum, 995.  
   — fibrosum, 986.  
 Moore's test, 735.  
 Morbid anatomy, 2.  
 Morbilli, 148.  
 Morphea, 986.  
 Mortification, 49.  
 Motor disorders, 821.  
   — paralysis, 824.  
 Mouth and tongue, diseases of, 314.  
   — ulcers of, 321.  
 Mucin, 47, 48.  
 Muco-enteritis, 633.  
 Mucoid degeneration, 73.  
 Mucous inflammation, 51.  
   — membrane of throat, relaxed, 332.  
   — râles, 365.  
 Mulberry calculus, 781.  
   — rash, 117.  
 Multilocular hydatid cyst, 687.  
 Mumps, 186; diag., 238.  
 Murmurs, 483, 485, 493.  
 Muscles, tests applied to, 803.

Muscular and tendinous rheumatism, 249.  
   — atrophy, progressive, 956.  
   — palsy, 824.  
   — paralysis, pseudo-hypertrophic, 961.  
 Myalgia, 249; varieties, *ib.*; treat., 250.  
 Myelitis, acute, 930; chronic, 938.  
 Myelogenic leucocythæmia, 711.  
 Myo-carditis, acute, 522.  
 Myxœdema, 31, 798.  
 Myxoma of brain, 921.  
 Nails, diseases of, 997.  
 Nasal diphtheria, 179.  
 Necrobiosis, 62.  
 Necrosis of cartilages of larynx, 388.  
 Nematodes, characters of, 660.  
 Nephritis, desquamative or tubal, acute,  
   761; chronic, 768.  
   — catarrhal, 756.  
   — interstitial, chronic, 770.  
   — suppurative, 757.  
 Nerve-centres, sclerosis of, 943; treat.,  
   955.  
 Nervous diseases, localization of, 847.  
   — — secondary atrophic lesions in,  
   844.  
   — dyspepsia, 606.  
   — system, diseases of the, clinical exam-  
   ination, 800; phys. exam., 802.  
   — uses of electricity in diagnosis of, 804.  
 Neuralgia, 836; varieties, 838; treat.,  
   839.  
 Neurasthenia spinalis, 927.  
 Neuritis, optic, 809.  
 Noma, 317.  
 Numerical hypertrophy, 61.  
 Nutmeg liver, 675.  
 Nystagmus, 802.  
 Obsolescence of tubercle, 291.  
 Obstruction, intestinal, 649.  
   — of the absorbent vessels, 789.  
   — pyloric, 616.  
 Œdema, 28.  
   — of the disc, 809.  
   — glottidis, 387.  
   — of the lungs, 403.  
 Œdematous erysipelas, 173.  
   — laryngitis, 376, 380.  
 Œgophony, 367.  
 Œsophagus, diseases of, 334.  
   — chronic diseases of, 336; functional  
   derangements of, 337; organic diseases  
   of, *ib.*; diag. and treat., 340.  
 Œsophagismus, 337.  
 Œsophagitis, acute, 336.  
 Oidium albicans, 317.  
 Oinomania, 882.  
 Oligæmia, 562.  
 Oligochromæmia, 562.  
 Oligocythæmia, 562.  
 Oliguria, 727.  
 Omentum, colloid in, 587.  
 Omodynia, 250.  
 Ophthalmoscope, uses of, 808.  
 Optic disc, hyperæmia, &c., 808.  
   — neuritis, 809.



- Opisthotonos, 878.  
 Organs, inflammation of, 51.  
 Orthotonos, 878.  
 Ossiform tissue, 273.  
 Osteoid cancer, 299.  
   — tissue, 274.  
 Ovarialgia, 867.  
 Ovarian dropsy, 28.  
   — tumour, physical signs of, 591.  
 Oxyuris vermicularis, 660.
- Pachymeningitis, spinal, 928, 937.  
 Palpitation, 509; symp., 510; treat., 512.  
 Palsy, muscular, 824; wasting, 956;  
   — Scrivener's, 959; shaking, 963.  
 Paludal fevers, 222.  
 Pancreas, diseases of the, 717.  
 Pancreatitis, 718.  
 Pandemic wave, 104.  
 Papillary stomatitis, 316.  
 Papular inflammations of skin, 978.  
 Paracentesis, 34.  
   — abdominis, 592; thoracis, 469.  
 Paræsthesia, 801.  
 Paralysis agitans, 963.  
   — ascendens acuta, 934.  
   — bulbar, 958.  
   — Cruveilhier's, 956.  
   — of children, essential, 932.  
   — diphtheritic, 889.  
   — disseminated, 827.  
   — Duchenne's, 961.  
   — facial, 828.  
   — general, 824.  
   — glosso-labio-laryngeal, 958.  
   — in connection with the eye, 829.  
   — infantile, 932.  
   — intestinal, 655.  
   — Landry's, 934.  
   — laryngeal, 390.  
   — motor or muscular, 824.  
   — of inferior maxillary nerve, 830.  
   — of œsophagus, 337.  
   — of pharynx, 830.  
   — of tongue, 830.  
   — pseudo-hypertrophic muscular, 961.  
   — sensory, 834.  
   — spinal, 932, 933.  
   — treatment of, 830.  
   — uses of electricity in, 832.  
 Paraplegia, 824, 827; spastic, 949.  
 Parasitic diseases of the skin, 990.  
   — growths in kidney, 778.  
   — stomatitis, 317.  
 Parenchymatous inflammation, 46.  
   — — of tongue, 320.  
 Paresis, 824.  
 Parotid bubo, 321.  
   — chronic enlargement of, 322.  
 Parotitis, idiopathic, 186; symptomatic, 321.  
 Paroxysmal hæmatinuria, 753.  
 Patellar reflex, 833.  
 Pathognomonic symptoms, 11.  
 Pathology, definition, &c., 2.  
 Pectoriloquy, 367.  
 Pediculus; pediculosis, 990.
- Peliosis rheumatica, 971.  
 Pemphigus, 976.  
 Percussion, 348, 356, 484, 488.  
 Perforating ulcer of stomach, 611.  
 Perforation, abdominal, 587.  
   — of the œsophagus, 339.  
   — in typhoid fever, 131.  
 Peri-arteritis, 551.  
 Pericardial dropsy, 518.  
   — hæmorrhage, 518.  
   — murmurs, 485, 488, 497.  
 Pericarditis, acute, 512; symp., 513;  
   — phys. signs, 514; treat., 516.  
   — chronic, 517.  
 Pericardium, diseases of, 512.  
   — adherent, 517.  
 Perichondritis, laryngeal, 388.  
 Peri-hepatitis, 679; treat., 680.  
 Perinephritis, 760.  
 Perineuritis, 810.  
 Peripheral nervous diseases, clinical phenomena of, 849.  
 Peritoneum, diseases of, 580.  
   — cancer of, 586.  
   — dropsy of, 581.  
   — hydatids of, 586.  
   — morbid growths in, 586.  
   — tubercle of, 586.  
 Peritonitis, acute, 580; anat. char., *ib.*;  
   — symp., 581; var., 582; treat., 583.  
   — chronic, 585; treat., 586.  
 Perityphlitis, 633.  
 Pertussis, 187.  
 Pestis, 214.  
 Petechiæ, 35.  
 Petechial hæmorrhage, 35.  
 Petit mal, 858.  
 Petrification, 69.  
 Pettenkofer's test, 736.  
 Pharyngitis, catarrhal, 326.  
   — follicular, 327.  
 Pharynx, paralysis of the, 830.  
 Phlebectasis laryngea, 384.  
 Phlebitis, 571.  
 Phlebolith, 571.  
 Phlegmasia dolens, 571.  
 Phlegmonous erysipelas, 173.  
 Phlyctenous erysipelas, 173.  
 Phtheiriasis, 990.  
 Phthisis, 434; ætiol., *ib.*; path., 436.  
   — acute, 439; treat., 440; diag., 474.  
   — bronchial, 793.  
   — chronic, 441; symp., 443; phys. signs, 447; var., 449; diag., 451; prog., *ib.*; treat., 452.  
   — fibroid, 416.  
   — inflammatory forms of, 436.  
 Physical examination explained, 14.  
   — — of the abdomen, 575.  
   — — of the chest, 343.  
   — — of circulatory organs, 484.  
 Piarhæmia, 750.  
 Pica, 593.  
 Picric acid as test for albumen, 732.  
 Pigmentary degeneration, 71.  
   — changes in skin, 989.  
 Pityriasis, 980; rubra, 981; versicolor, 992.

- Plague, 214; diag., 239.  
 Plantar reflex, 804.  
 Plaques molles, 125; dures, *ib.*  
 Plastic bronchitis, 402.  
 — laryngitis, 380.  
 Plethora, 562.  
 Pleura, diseases of, 462.  
 — dropsy of the, 471.  
 — diag. of affections of, table, 474.  
 — hæmorrhage into the, 472.  
 Pleurisy, 462; anat. char., 463; symp., 464; phys. signs, 465; treat., 467; diag., 474.  
 Pleuritic friction-fremitus, 356; sounds, 366.  
 Pleurodynia, 250.  
 Pleurosthotonos, 878.  
 Pleximeter, 348.  
 Pneumonia, 407.  
 — acute croupous, 407; anat. char., 408; symp., 409; phys. signs, 411; term. and dur., 412; prog., *ib.*; diag., *ib.* and 474; treat., 413.  
 — catarrhal, 414; diag., 474; treat., 416.  
 — chronic or interstitial, 416.  
 — disseminated or lobular, 414.  
 — hypostatic, 408.  
 — intermittent, 410.  
 — latent, 410.  
 — lobar, 407.  
 — typhoid, 411.  
 Pneumo-pericardium, 518.  
 Pneumothorax, 472.  
 Podagra, 253.  
 Polio-myelitis anterior acuta, 932; sub-acuta, 934.  
 Polyæmia, 562.  
 Polycythæmia, 562.  
 Polydipsia, 312.  
 Polyuria, 312, 727.  
 Portable tests for urine, 732.  
 Portal congestion, 675.  
 Pouching of œsophagus, 339.  
 Prognosis, 2, 19.  
 Prolapsus ani, 650.  
 Prophylaxis, 21.  
 Prosopalgia, 838.  
 Prurigo, 979.  
 Psoriasis, 980.  
 Ptyalism, 322.  
 Pulmonary artery and branches, thrombosis in, 570.  
 — — murmurs, 497.  
 — apoplexy, 404.  
 — breathing, 360.  
 — collapse, 432.  
 — compression, 432.  
 — congestion, 403; symp., 406; phys. signs, *ib.*; prog., *ib.*; treat., *ib.*  
 — consumption, 434.  
 — diseases, acute, diagnostic table of, 474.  
 — obstruction and regurgitation, 529.  
 — orifice, disease at, 529.  
 — percussion-sound, 556; changes in, 557.  
 Pulse, the, 499.  
 Purpura, 267.  
 Purulent urine, 747.  
 Pus, formation of, 48; kinds of, 49.  
 Pustule, malignant, 207; diag., 238.  
 Pustular inflammations of skin, 977.  
 Putrid fever, 83.  
 Pyæmia, 566.  
 Pyæmic hepatic abscess, 676.  
 Pyelitis, 758.  
 Pyelo-nephritis, 759.  
 Pyle-phlebitis, 676.  
 Pyloric obstruction or incompetence, 616.  
 Pyonephrosis, 758.  
 Pyo-pneumothorax, 472.  
 Pyrexia, 78.  
 Pyrosis, 594.  
 Pythogenic fever, 122.  
 Pyuria, 747.  
 Quartan ague, 227.  
 Quinsy, 327.  
 Quotidian ague, 227.  
 Rabies, 210.  
 Rachitis, 269; symp., 278; treat., 280.  
 Râles, 349, 363; classification of, *ib.*  
 Reactions of degeneration, 806.  
 Red softening of brain, 911.  
 Reflexes, exam. of, 803.  
 — superficial and deep, 833.  
 Regurgitation of food, 594.  
 Relapsing fever, 135; symp., 137; relapse, 138; comp. and seq., 139; treat., *ib.*  
 — — diagnosis of, 234, 236.  
 Relapsing type of fever, 81.  
 Remittent fever, 229; diag., 239.  
 — type of fever, 81.  
 Renal abscess, 757.  
 — calculus, 781.  
 — casts, 742.  
 — congestion, 756.  
 — embolism, 756.  
 — infarction, 756.  
 — tumour, 744.  
 — (*See also* Kidneys).  
 Resolution, 47.  
 Respiration, movements of, 352; sounds of, 349.  
 Respiratory organs, diseases of the, clinical characters, 341.  
 Retching, 594.  
 Retinitis, 810.  
 Retrocedent gout, 257.  
 Retro-pharyngeal abscess, 334.  
 Rheumatism, 240; diag., 244, 259.  
 — acute articular, 240.  
 — chronic articular, 248.  
 — gonorrhœal, 251.  
 — muscular, 249.  
 Rheumatic fever, 240; ætiol., *ib.*; anat. char., 241; symp., 242; comp., 243; treat., 245.  
 — gout, 252.  
 Rheumatic meningitis, 899.  
 Rheumatoid arthritis, 252; diag., 253, 259.

- Rhonchal fremitus, 356.  
 Rhonchi, 349, 363.  
 Rickets, 269; in glands, 794.  
 Rickety liver, 696.  
 Ringworm, 992.  
 Rosacea, 995.  
 Rose, the, 170.  
 Roseola, 970.  
 Rotation of intestine, 650.  
 Rötheln, 152.  
 Round worm, 658, 663.  
 Rubeola, 148.  
 — notha, 152.  
 Rupia, 984.  
 Ruptures, abdominal, 587.  
 — of heart, 539.  
  
 St. Anthony's fire, 170.  
 St. Vitus's dance, 873.  
 Salivary glands, diseases of, 314.  
 Salivation, 322.  
 Sanguineous apoplexy, 906.  
 Sarcina ventriculi, 594.  
 Sarcoma of brain, 921.  
 Saturnism, 886.  
 Scabies, 991.  
 Scalp, rheumatism of, 249.  
 Scapular reflex, 804.  
 Scapulodynia, 250.  
 Scarification, 35.  
 Scarletina, Scarlet fever, 140; symp., 142; stages, *ib.*; var., 144; comp. and seq., 145; prog., 146; treat., *ib.*; diag. of, 236, 334.  
 Sciatica, 839.  
 Scirrhus cancer, 298.  
 — of pancreas, 719.  
 Scleroderma, 986.  
 Sclerosis of nerve-centres, 943; treat., 955.  
 Scorbutus, 263.  
 Scrivener's palsy, 959.  
 Scrofulosis, 287.  
 Scrofulous diathesis, 295.  
 — disease of the glands, 793.  
 — — of the intestines, 647.  
 Scurvy, 263.  
 Sea-sickness, 598.  
 Seat-worm, 657, 663, 941.  
 Sebaceous glands, diseases of, 994.  
 Seborrhœa, 995.  
 Secondary fever, 78.  
 Semeiology, 2, 10.  
 Senile arthritis, 248.  
 Sensory paralysis, 834.  
 Septic fever, 83.  
 Septicæmia, 566.  
 Sequelæ, 12.  
 Serous inflammations, 50.  
 Serum, 47.  
 Shaking palsy, 963.  
 Sick-headache, 855.  
 Simple erysipelas, 173.  
 Skin, diseases of the, 965.  
 — diathetic diseases of, 981.  
 — hypertrophies and atrophies of, 985.  
 — inflammation of, 50.  
  
 Skin, inflammatory diseases of, 970; bulbous, 975; catarrhal, 972; erythematous 970; papular, 978; pustular, 977 squamous, 980.  
 — new formations of, 987.  
 — pigmentary changes of, 989.  
 — parasitic diseases of, 990.  
 Sleep, disorders of, 818.  
 Small-pox, 154; anat. char., 155; symp., 156; eruption, 157; secondary fever, 158; varieties, 159; inoculated, 160; after vaccination, *ib.*; comp. and seq., 161; prog., *ib.*; treat., 162.  
 — diagnosis of, 235, 237.  
 Soft cancer, 298.  
 Softening of the brain, 910; acute, 912; chronic, 919.  
 — of spinal cord, 938.  
 — of heart, 538.  
 Somnambulism—somniloquism, 819.  
 Somnolence, 818.  
 Sore-throat, relaxed, 326; hospital, 329; sloughing, 330; granular, 332; follicular, *ib.*  
 Sounds of the heart, 484, 490.  
 Spanæmia, 563.  
 Spasms, 821.  
 Spasm of the glottis, 388.  
 — of intestines, 626, 650.  
 — of œsophagus, 337.  
 — of the stomach, 597.  
 Spasmodic asthma, 427.  
 — croup, 388.  
 — wry-neck, 964.  
 Spastic paraplegia, 945.  
 Specific fevers, acute, 78.  
 — diseases, 4; diagnosis of, 231.  
 Sphygmograph, description of the, 499; uses, 502.  
 Sphygmographic tracings, 500.  
 Sphygmomanometer, 484.  
 Spinal cord, diseases of, clinical phenomena, 848.  
 Spinal cord and membranes, diseases of, 926; diag., 941; treat. of, 942.  
 — adventitious growths in, 940.  
 — concussion of, 927.  
 — congestion, 935.  
 — hæmorrhage, 936.  
 — inflammation, 930.  
 — meningitis, acute, 928.  
 — — chronic, 937.  
 — paralysis, 932, 933, 949.  
 — sclerosis, 945, 952; treat., 955.  
 — softening, 938.  
 Spirillum, 100, 135.  
 Spleen, diseases of the, 707; general diag., prog., and treat., 716.  
 — congestion of, 709.  
 — hypertrophy of, 710.  
 — rare morbid conditions of, 715.  
 Splenic cachexia, 708.  
 — leucocythemia, 711.  
 — tumour, 708.  
 Splenification of lung, 404.  
 Splenitis, 709.  
 Sporadic cholera, 200.



- Sporadic diseases, 102.  
 Sputa, 342.  
 Squamous inflammations of skin, 980.  
 Sthenic ague, 227.  
 Stiff-neck, 249.  
 Stigmata, 35.  
 Stomach, cancer of, 614; treat., 621.  
 — chronic diseases of, 610.  
 — dilatation of, 616.  
 — and intestines, diseases of the, clinical characters, 593.  
 — inflammation of, acute, 607; chronic, 610.  
 — spasm or cramp of, 597.  
 — ulcer of, 611; diag., 619; treat., 621.  
 Stomatitis, 314; treat., 319.  
 Stricture of œsophagus, 338.  
 — of intestines, 649.  
 Strumous diathesis, 295.  
 Stupor, 816.  
 Subcuticular mottling in typhus, 116.  
 Succussion, 368.  
 Sudamina, 987.  
 Suffocative breast-pang, 504.  
 Sugar, tests for, 733.  
 Summer diarrhoea, 200.  
 Sun-stroke, 892.  
 Suppuration, 48.  
 — of gall-bladder, 696.  
 Suppurative inflammation of liver, 676.  
 — of kidney, 757.  
 — nephritis, 757.  
 — phlebitis, 571.  
 — pyle-phlebitis, 676.  
 Supra-renal capsules, diseases of, 720.  
 Sweat-glands, diseases of, 993.  
 Sycosis, 996.  
 Symptomatology, 2, 10.  
 Symptomatic fever, 78.  
 — parotitis, 321.  
 Symptoms, mode of examination for, 17.  
 Syncope, 507; treat., 508.  
 Synocha, 113.  
 Syphilides, 981.  
 Syphilis, acquired, skin eruptions in, 982.  
 — congenital, 284.  
 — constitutional, 281; diag., 286; treat., 287.  
 — hereditary, 284.  
 — inoculated, 281.  
 Syphilitic acne, 984.  
 — disease of arteries, 553; of lungs, 460; of liver, 695; of brain, 921; of spleen, 715.  
 — skin eruptions, 981.  
 Systemic veins, thrombosis in the, 571.  
 Tabes dorsalis, 945.  
 — mesenterica, 793.  
 Tâches cerebrales, 811.  
 Tænia Egyptica, 657.  
 — elliptica, 657.  
 — flavo-punctata, 657.  
 — medio-canellata, 659.  
 — nana, 657.  
 — solium, 659.  
 Tape-worms, 657, 658.  
 Temperaments, 6.  
 Temperature, 109.  
 Tendinous rheumatism, 249.  
 Tendon reflexes, 803, 833.  
 Tenesmus, 596.  
 Tertian ague, 227.  
 Tetanus, 878.  
 Tetany, 879.  
 Therapeutics, 2, 20.  
 Therapeutic treatment, 22.  
 Thermo-anæsthesia, 801.  
 Thermometer, mode of use, &c., 108.  
 Thoracic aneurism, 555; symp., 556; phys. signs, *ib.*; diag., 557; treat., 558.  
 Thorax, divisions of, 345.  
 Thread-worm, 657, 658.  
 Thrill, 488, 498.  
 Throat, diseases of, 324; chronic, 332.  
 — ulceration of, 330.  
 Thrombosis, 568.  
 — arterial, 572.  
 — cardiac, 569.  
 — cerebral, 910.  
 — in the pulmonary artery, 570.  
 — in the systemic veins, 571.  
 Thrush, 317.  
 Thyroid gland, diseases of the, 795.  
 Tic-douloureux, 838.  
 Tinea, 991.  
 “Tissu spongoïde,” 273.  
 Tongue, characters of, 594.  
 — diseases of, 314.  
 — paraiysis of, 830.  
 — parenchymatous inflammation of, 320.  
 — ulcers of, 321.  
 Tonsillitis, acute, 327; treat., 328.  
 Tonsils, chronic enlargement of, 333.  
 Torsion, 650.  
 Torticollis, 249, 964.  
 Torula cerevisiæ, 99.  
 — ventriculi, 594.  
 Trachea, affections of, 376; exam. of, 343.  
 Tracheal catarrh, 379.  
 — sounds, 356.  
 Tracheitis, 376.  
 Trance, 870.  
 Traumatic hæmorrhage, 36.  
 Treatment, 2, 20; therapeutic, 22.  
 Tremor, purring, 488.  
 Trichina spiralis, 666.  
 Trichinosis, 665.  
 Tricocephalus dispar, 657.  
 Tricophyton, 991.  
 Tricuspid murmurs, 496.  
 — obstruction, 529.  
 — regurgitation, 528.  
 Tropical hepatic abscess, 676.  
 Tubal nephritis, acute, 761.  
 — — chronic, 768.  
 Tubercle, 288.  
 — bacilli, detection of, 444.  
 — of brain, 920.  
 — of glands, 793.  
 — of intestines, 647.  
 — of kidney, 778.  
 — of liver, 696.

- Tubercle of lungs, 438.  
 — of peritoneum, 586.  
 — of spleen, 715.  
 Tubercular disease of the glands, 793.  
 — diathesis, 295.  
 — meningitis, 896.  
 — phthisis, 438.  
 — ulceration of the intestines, 647.  
 Tuberculosis, 287; path., 291; symp., 295; diag., 296; treat., 297.  
 — acute forms of, 296.  
 — of liver, 696.  
 Tubular breathing, 362.  
 — cancer, 299.  
 Tumours, mediastinal, 560.  
 Tumour, cerebral, 920; renal, 744.  
 Tussive fremitus, 356; resonance, 349, 368.  
 Tympanic sound, 356.  
 Typhlitis, 633.  
 Typhoid fever, 122; predisposing causes, 123; anat. char., 124; stages, *ib.*; symp., 127; varieties, 130; comp. and seq., 131; duration and term., *ib.*; prog., 132; treat., *ib.*  
 — diagnosis of, 234, 236.  
 — type of fever, 81.  
 Typhus fever, 114; anat. char., 115; symp., 116; varieties, 119; sequelæ, *ib.*; term. and duration, *ib.*; treat., 120.  
 — — diagnosis of, 234, 236.  
 Ulceration, 49.  
 Ulcerative stomatitis, 316.  
 Ulcers of the intestines, 644.  
 — of the larynx, 386.  
 — of the mouth and tongue, 321.  
 — of the œsophagus, 337.  
 — of the stomach, 611.  
 — of the throat, 330.  
 — typhoid, 125.  
 Unilateral paralysis, 824, 825; sensory, 835.  
 Uræmia, 727, 754.  
 Urinary calculus, 781.  
 — — deposits, 737.  
 — organs, diseases of the, 726.  
 Urine, abnormal conditions of, 746.  
 — chylous, 740, 748.  
 — examination of, 727.  
 — purulent, 747.  
 — tests for, 728.  
 Urticaria, 971.  
 Vaccination, Vaccinia, 165.  
 Vacuolation, 302.  
 Valves and orifices of heart, affections of, 523.  
 Valvulitis, 519.  
 Varicella, 169; diag., 235, 237.  
 Varicelloid, 160.  
 Variola. *See* Small-pox.  
 Varioloid, 160.  
 Veins, clinical phenomena connected with the, 484.  
 — exam. of, 502.  
 Venesection in inflammation, 58.  
 Venous hyperæmia, 24.  
 — murmurs, 485, 503.  
 Ventricular hæmorrhage, 909.  
 Verruca, 985.  
 Vertige épileptique, 858.  
 Vertigo, 812.  
 Vesical catarrh, 785.  
 Vesicular breathing, 360.  
 — emphysema, 420.  
 Vibices, 35.  
 Villous cancer, 299.  
 Virus, 98.  
 Vitiligoidea, 701.  
 Vocal cords, paralysis of muscles of, 390.  
 — fremitus, 355; resonance, 345, 349, 366.  
 Volvulus, 650, 651.  
 Vomiting, 598; treat., 600.  
 — of blood, 601.  
 Wart, 985.  
 Wart-pock, 160.  
 Wasting palsy, 956.  
 Water-brash, 594.  
 Water canker, 317.  
 Waxy disease, 74.  
 Waxy liver, 688.  
 Whip worm, 657, 662.  
 White leg, 572.  
 — mouth, 317.  
 — softening, 938.  
 Whooping cough, 187.  
 Worms, intestinal, 656.  
 Writer's cramp, 959.  
 Wry neck, 249, 964.  
 Xanthelasma, 996.  
 Xanthopsys, 672.  
 Yellow atrophy of liver, acute, 681.  
 Yellow fever, 216; anat. char., *ib.*; symp. 217; var., 218; treat., *ib.*  
 — — diagnosis of, 239.  
 — tubercle, 289.  
 Zona or Zoster, 975.  
 Zyme, 98.  
 Zymotic diseases, 102.



February, 1885.

# CATALOGUE OF WORKS

PUBLISHED BY

## H. K. LEWIS

136 GOWER STREET, LONDON, W.C.

---

G. GRANVILLE BANTOCK, M.D., F.R.C.S. EDIN.  
*Surgeon to the Samaritan Free Hospital for Women and Children.*

I.  
ON THE USE AND ABUSE OF PESSARIES. Second Edition,  
with Illustrations, 8vo, 5s.

II.  
A PLEA FOR EARLY OVARIOTOMY. Demy 8vo, 2s.

---

FANCOURT BARNES, M.D., M.R.C.P.  
*Physician to the Chelsea Hospital for Women; Obstetric Physician to the Great Northern Hospital, &c.*

A GERMAN-ENGLISH DICTIONARY OF WORDS AND  
TERMS USED IN MEDICINE AND ITS COGNATE SCIENCES.  
Square 12mo, Roxburgh binding, 9s.

---

ASHLEY W. BARRETT, M.B. LOND., M.R.C.S., L.D.S.  
*Dental Surgeon to the London Hospital, &c.*

DENTAL SURGERY FOR GENERAL PRACTITIONERS  
AND STUDENTS OF MEDICINE. With Illustrations, crown 8vo,  
3s. [Now ready.  
*Lewis's Practical Series*].

---

ROBERTS BARTHOLOW, M.A., M.D., LL.D.  
*Professor of Materia Medica and Therapeutics, in the Jefferson Medical College of Philadelphia, &c., &c.*

I.  
A TREATISE ON THE PRACTICE OF MEDICINE, FOR  
THE USE OF STUDENTS AND PRACTITIONERS. Fifth  
Edition, with Illustrations, large 8vo, 21s. [Just published.

II.  
A PRACTICAL TREATISE ON MATERIA MEDICA AND  
THERAPEUTICS. Fifth Edition, Revised and Enlarged, 8vo, 18s.  
[Just published.

---

GEO. M. BEARD, A.M., M.D.  
*Fellow of the New York Academy of Medicine; Member of the American Academy of Medicine, &c.*

AND  
A. D. ROCKWELL, A.M., M.D.  
*Fellow of the New York Academy of Medicine; Member of the American Academy of Medicine, &c.*

A PRACTICAL TREATISE ON THE MEDICAL AND  
SURGICAL USES OF ELECTRICITY. Including Localized and  
General Faradization; Localized and Central Galvanization; Frank-  
linization; Electrolysis and Galvano-Cautery. Fourth Edition. With  
nearly 200 Illustrations, roy. 8vo, 28s. [Just published.



**A. HUGHES BENNETT, M.D.**

*Member of the Royal College of Physicians of London; Physician to the Hospital for Epilepsy and Paralysis, Regent's Park, and Assistant Physician to the Westminster Hospital.*

**I.**  
**A PRACTICAL TREATISE ON ELECTRO-DIAGNOSIS**  
**IN DISEASES OF THE NERVOUS-SYSTEM.** With Illustrations,  
8vo, 8s. 6d.

**II.**  
**ILLUSTRATIONS OF THE SUPERFICIAL NERVES AND**  
**MUSCLES, WITH THEIR MOTOR POINTS,** A knowledge of  
which is essential in the Art of Electro-Diagnosis. (Extracted from the  
above). 8vo, paper cover, 1s. 6d.; cloth, 2s.

**III.**  
**ON EPILEPSY: ITS NATURE AND TREATMENT.** 8vo, 2s. 6d.

**DR. THEODOR BILLROTH.**

*Professor of Surgery in Vienna.*

**GENERAL SURGICAL PATHOLOGY AND THERA-**  
**PEUTICS.** In Fifty-one Lectures. A Text-book for Students and  
Physicians. With additions by Dr. ALEXANDER VON WINIWARTER, Pro-  
fessor of Surgery in Luttich. Translated from the Fourth German edi-  
tion with the special permission of the Author, and revised from the  
Tenth edition, by C. E. HACKLEY, A.M., M.D. Copiously illustrated,  
8vo, 18s.

**G. H. BRANDT, M.D.**

**I.**  
**ROYAT (LES BAINS) IN AUVERGNE, ITS MINERAL**  
**WATERS AND CLIMATE.** With Frontispiece and Map. Second  
edition, crown 8vo, 2s. 6d.

**II.**  
**HAMMAM R'IRHA, ALGIERS. A Winter Health Re-**  
**sort and Mineral Water Cure Combined.** With Frontispiece and Map,  
crown 8vo, 2s. 6d.

**GURDON BUCK, M.D.**

**CONTRIBUTIONS TO REPARATIVE SURGERY; SHOW-**  
ing its Application to the Treatment of Deformities, produced by De-  
structive Disease or Injury; Congenital Defects from Arrest or Excess  
of Development; and Cicatricial Contractions from Burns. Illustrated  
by numerous Engravings, large 8vo, 9s.

**ALFRED H. CARTER, M.D. LOND.**

*Member of the Royal College of Physicians; Physician to the Queen's Hospital, Birmingham;  
Examiner in Medicine for the University of Aberdeen, &c.*

**ELEMENTS OF PRACTICAL MEDICINE.** Third Edition,  
crown 8vo, 9s. [Now ready.]

**P. CAZEAUX.**

*Adjunct Professor in the Faculty of Medicine of Paris, &c.*

**AND**  
**S. TARNIER.**

*Professor of Obstetrics and Diseases of Women and Children in the Faculty of Medicine of  
Paris.*

**OBSTETRICS: THE THEORY AND PRACTICE;** including  
the Diseases of Pregnancy and Parturition, Obstetrical Operations, &c.  
Seventh Edition, edited and revised by ROBERT J. HESS, M.D., with  
twelve full-page plates, five being coloured, and 165 wood-engravings,  
1081 pages, roy. 8vo, 35s. [Now ready.]

---

JOHN COCKLE, M.A., M.D.

*Physician to the Royal Free Hospital.*

ON INTRA-THORACIC CANCER. 8vo, 4s. 6d.

---

W. H. CORFIELD, M.A., M.D. OXON.

*Professor of Hygiene and Public Health in University College, London.*

DWELLING HOUSES: Their Sanitary Construction and Arrangements. Second Edit., with Illustrations. Cr. 8vo, 3s. 6d. [*Just ready.*]

---

J. THOMPSON DICKSON, M.A., M.B. CANTAB.

*Late Lecturer on Mental Diseases at Guy's Hospital.*

THE SCIENCE AND PRACTICE OF MEDICINE IN RELATION TO MIND, the Pathology of the Nerve Centres, and the Jurisprudence of Insanity, being a course of Lectures delivered at Guy's Hospital. Illustrated by Chromo-lithographic Drawings and Physiological Portraits. 8vo, 14s.

---

HORACE DOBELL, M.D.

*Consulting Physician to the Royal Hospital for Diseases of the Chest, &c.*

I.  
ON DIET AND REGIMEN IN SICKNESS AND HEALTH, and on the Interdependence and Prevention of Diseases and the Diminution of their Fatality. Seventh Edition, 8vo, 10s. 6d.

II.  
AFFECTIONS OF THE HEART AND IN ITS NEIGHBOURHOOD. Cases, Aphorisms, and Commentaries. Illustrated by the heliotype process. 8vo, 6s 6d.

---

JOHN EAGLE.

*Member of the Pharmaceutical Society.*

A NOTE-BOOK OF SOLUBILITIES. Arranged chiefly for the use of Prescribers and Dispensers. 12mo, 2s. 6d.

---

JOHN ERIC ERICHSEN.

*Holme Professor of Clinical Surgery in University College; Senior Surgeon to University College Hospital, &c.*

MODERN SURGERY; Its Progress and Tendencies. Being the Introductory Address delivered at University College at the opening of the Session 1873-74. Demy 8vo, 1s.

---

DR. FERBER.

MODEL DIAGRAM OF THE ORGANS IN THE THORAX AND UPPER PART OF THE ABDOMEN. With Letter-press Description. In 4to, coloured, 5s.

AUSTIN FLINT, JR., M.D.

*Professor of Physiology and Physiological Anatomy in the Bellevue Medical College, New York; attending Physician to the Bellevue Hospital, &c.*

I.

**A TEXT-BOOK OF HUMAN PHYSIOLOGY; DESIGNED**  
for the Use of Practitioners and Students of Medicine. New edition,  
Illustrated by plates, and 313 wood engravings, large 8vo, 28s.

II.

**THE PHYSIOLOGY OF THE SPECIAL SENSES AND**  
**GENERATION; (Being Vol. V. of the Physiology of Man).** Roy. 8vo,  
18s.

J. MILNER FOTHERGILL, M.D.

*Member of the Royal College of Physicians of London; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park, &c.*

I.

**THE HEART AND ITS DISEASES, WITH THEIR TREAT-**  
**MENT; INCLUDING THE GOUTY HEART.** Second Edition,  
entirely re-written, copiously illustrated with woodcuts and litho-  
graphic plates. 8vo. 16s.

II.

**INDIGESTION, BILIOUSNESS, AND GOUT IN ITS PRO-**  
**TEAN ASPECTS.**

**PART I.—INDIGESTION AND BILIOUSNESS.** Post 8vo, 7s. 6d.

**PART II.—GOUT IN ITS PROTEAN ASPECTS.** Post 8vo, 7s. 6d.

III.

**HEART STARVATION.** (Reprinted from the Edinburgh Medical  
Journal), 8vo, 1s.

ERNEST FRANCIS, F.C.S.

*Demonstrator of Practical Chemistry, Charing Cross Hospital.*

**PRACTICAL EXAMPLES IN QUANTITATIVE ANALYSIS,**  
forming a Concise Guide to the Analysis of Water, &c. Illustrated,  
fcap. 8vo, 2s. 6d.

HENEAGE GIBBES, M.D.

*Lecturer on Physiology and Histology in the Medical School of Westminster Hospital; late  
Curator of the Anatomical Museum at King's College.*

**PRACTICAL HISTOLOGY AND PATHOLOGY.** Second Edit.  
revised and enlarged. Crown 8vo, 5s.

C. A. GORDON, M.D., C.B.

*Deputy Inspector General of Hospitals, Army Medical Department.*

**REMARKS ON ARMY SURGEONS AND THEIR WORKS.**  
Demy 8vo, 5s.



W. R. GOWERS, M.D., F.R.C.P. M.R.C.S.

*Physician to University College Hospital, &c.*

**DIAGRAMS FOR THE RECORD OF PHYSICAL SIGNS.**

In books of 12 sets of figures, 1s. Ditto, unbound, 1s.

SAMUEL D. GROSS, M.D., LL.D., D.C.L., OXON.

*Professor of Surgery in the Jefferson Medical College of Philadelphia.*

**A PRACTICAL TREATISE ON THE DISEASES, INJURIES, AND MALFORMATIONS OF THE URINARY BLADDER, THE PROSTATE GLAND; AND THE URETHRA.** Third Edition, revised and edited by S. W. GROSS, A.M., M.D., Surgeon to the Philadelphia Hospital. Illustrated by 170 engravings, 8vo, 18s.

SAMUEL W. GROSS, A.M., M.D.

*Surgeon to, and Lecturer on Clinical Surgery in, the Jefferson Medical College Hospital, and the Philadelphia Hospital, &c.*

**A PRACTICAL TREATISE ON TUMOURS OF THE MAMMARY GLAND:** embracing their Histology, Pathology, Diagnosis, and Treatment. With Illustrations, 8vo, 10s. 6d.

WILLIAM A. HAMMOND, M.D.

*Professor of Mental and Nervous Diseases in the Medical Department of the University of the City of New York, &c.*

**I.**  
**A TREATISE ON THE DISEASES OF THE NERVOUS SYSTEM.** Seventh edition, with 112 Illustrations, large 8vo, 25s.

**II.**  
**A TREATISE ON INSANITY.** Large 8vo, 25s. [Just published.]

**III.**  
**SPIRITUALISM AND ALLIED CAUSES AND CONDITIONS OF NERVOUS DERANGEMENT.** With Illustrations, post 8vo, 8s. 6d.

ALEXANDER HARVEY, M.A., M.D.

*Emeritus Professor of Materia Medica in the University of Aberdeen; Consulting Physician to the Aberdeen Royal Infirmary, &c.*

**FIRST LINES OF THERAPEUTICS;** as based on the Modes and the Processes of Healing, as occurring Spontaneously in Disease; and on the Modes and the Processes of Dying, as resulting Naturally from Disease. In a series of Lectures. Post 8vo, 5s.

ALEXANDER HARVEY, M.D.

*Emeritus Professor of Materia Medica in the University of Aberdeen, &c.*

AND

ALEXANDER DYCE DAVIDSON, M.D.

*Professor of Materia Medica in the University of Aberdeen.*

**SYLLABUS OF MATERIA MEDICA FOR THE USE OF TEACHERS AND STUDENTS.** Based on a selection or definition of subjects in teaching and examining; and also on an estimate of the relative values of articles and preparations in the British Pharmacopœia with doses affixed. Seventh Edition, 32mo.

[In preparation]

**GRAILY HEWITT, M.D.**

*Professor of Midwifery and Diseases of Women in University College, Obstetrical Physician to University College Hospital, &c.*

**OUTLINES OF PICTORIAL DIAGNOSIS OF DISEASES OF WOMEN.** Fol. 6s.

**BERKELEY HILL, M.B. LOND., F.R.C.S.**

*Professor of Clinical Surgery in University College; Surgeon to University College Hospital and to the Lock Hospital.*

**THE ESSENTIALS OF BANDAGING.** For Managing Fractures and Dislocations; for administering Ether and Chloroform; and for using other Surgical Apparatus. Fifth Edition, revised and much enlarged, with Illustrations, fcap. 8vo, 5s.

**BERKELEY HILL, M.B. LOND., F.R.C.S.**

*Professor of Clinical Surgery in University College; Surgeon to University College Hospital and to the Lock Hospital.*

AND

**ARTHUR COOPER, L.R.C.P., M.R.C.S.**

*Late House Surgeon to the Lock Hospital, &c*

I.

**SYPHILIS AND LOCAL CONTAGIOUS DISORDERS.**

Second Edition, entirely re-written, royal 8vo, 18s.

II.

**THE STUDENT'S MANUAL OF VENEREAL DISEASES.** Being a Concise Description of those Affections and of their Treatment. Third Edition, post 8vo, 2s. 6d.

**HINTS TO CANDIDATES FOR COMMISSIONS IN THE PUBLIC MEDICAL SERVICES, WITH EXAMINATION QUESTIONS, VOCABULARY OF HINDUSTANI MEDICAL TERMS, ETC.** 8vo, 2s.

**SIR W. JENNER, Bart., M.D.**

*Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales.*

**THE PRACTICAL MEDICINE OF TO-DAY: Two**

Addresses delivered before the British Medical Association, and the Epidemiological Society, (1869). Small 8vo, 1s. 6d.

**C. M. JESSOP, M.R.C.P.**

*Associate of King's College, London; Brigade Surgeon H.M. British Forces.*

**ASIATIC CHOLERA,** being a Report on an Outbreak of Epidemic Cholera in 1876 at a Camp near Murree in India. With map, demy 8vo, 2s. 6d.

---

GEORGE LINDSAY JOHNSON, M.A., M.B., B.C. CANTAB.  
*Clinical Assistant, late House Surgeon and Chloroformist, Royal Westminster Ophthalmic Hospital; Medical and Surgical Registrar, &c.*

**A NEW METHOD OF TREATING CHRONIC GLAUCOMA**, based on Recent Researches into its Pathology. With Illustrations and coloured frontispiece, demy 8vo, 3s. 6d.

---

NORMAN W. KINGSLEY, M.D.S., D.D.S.  
*President of the Board of Censors of the State of New York; Member of the American Academy of Dental Science, &c.*

**A TREATISE ON ORAL DEFORMITIES AS A BRANCH OF MECHANICAL SURGERY.** With over 350 Illustrations, 8vo, 16s.

---

E. A. KIRBY, M.D., M.R.C.S. ENG.  
*Late Physician to the City Dispensary.*

I.  
**A PHARMACOPŒIA OF SELECTED REMEDIES, WITH THERAPEUTIC ANNOTATIONS**, Notes on Alimentation in Disease, Air, Massage, Electricity and other Supplementary Remedial Agents, and a Clinical Index; arranged as a Handbook for Prescribers. Sixth Edition, enlarged and revised, demy 4to, 7s.

II.  
**ON THE VALUE OF PHOSPHORUS AS A REMEDY FOR LOSS OF NERVE POWER.** Fifth Edition, 8vo, 2s. 6d.

---

J. WICKHAM LEGG, F.R.C.P.  
*Assistant Physician to Saint Bartholomew's Hospital, and Lecturer on Pathological Anatomy in the Medical School.*

I.  
**ON THE BILE, JAUNDICE, AND BILIOUS DISEASES.** With Illustrations in chromo-lithography, 719 pages, roy. 8vo, 25s.

II.  
**A GUIDE TO THE EXAMINATION OF THE URINE;** intended chiefly for Clinical Clerks and Students. Fifth Edition, revised and enlarged, with additional Illustrations, fcap. 8vo, 2s. 6d.

III.  
**A TREATISE ON HÆMOPHILIA, SOMETIMES CALLED THE HEREDITARY HÆMORRHAGIC DIATHESIS.** Fcap. 4to, 7s. 6d.

---

DR. GEORGE LEWIN.  
*Professor at the Fr. Wilh. University, and Surgeon-in-Chief of the Syphilitic Wards and Skin Disease Wards of the Charité Hospital, Berlin.*

**THE TREATMENT OF SYPHILIS WITH SUBCUTANEOUS SUBLIMATE INJECTIONS.** Translated by DR. CARL PRÆGLE, and DR. E. H. GALE, late Surgeon United States Army. Small 8vo, 7s.



**LEWIS'S PRACTICAL SERIES.**

Under this title Mr. LEWIS purposes publishing a complete Series of Monographs, embracing the various branches of Medicine and Surgery.

The volumes, written by well-known Hospital Physicians and Surgeons recognized as authorities in the subjects of which they treat, are in active preparation. The works are intended to be of a THOROUGHLY PRACTICAL nature, calculated to meet the requirements of the general practitioner, and to present the most recent information in a compact and readable form; the volumes will be handsomely got up, and issued at low prices, varying with the size of the works.

**BODILY DEFORMITIES AND THEIR TREATMENT: A HANDBOOK OF PRACTICAL ORTHOPÆDICS.** By H. A. REEVES, F.R.C.S. Edin., Senior Assistant Surgeon and Teacher of Practical Surgery at the London Hospital; Surgeon to the Royal Orthopædic Hospital, &c. With numerous Illustrations, cr. 8vo, 8s. 6d. [Now ready.]

**DENTAL SURGERY FOR GENERAL PRACTITIONERS AND STUDENTS OF MEDICINE.** By ASHLEY W. BARRETT, M.B. Lond., M.R.C.S., L.D.S., Dental Surgeon to, and Lecturer on Dental Surgery and Pathology in the Medical School of, the London Hospital. With Illustrations, cr. 8vo, 3s. [Now ready]

Further volumes will be announced in due course.

\* \* \* Prospectus of the Series with specimen pages, &c., on application.

**LEWIS'S POCKET MEDICAL VOCABULARY.**

[In the Press.]

**J. S. LOMBARD, M.D.**

*Formerly Assistant Professor of Physiology in Harvard College.*

I.  
**EXPERIMENTAL RESEARCHES ON THE REGIONAL TEMPERATURE OF THE HEAD,** under Conditions of Rest, Intellectual Activity and Emotion. With Illustrations, 8vo, 8s.

II.  
**ON THE NORMAL TEMPERATURE OF THE HEAD.**  
8vo, 5s.

**WILLIAM THOMPSON LUSK, A.M., M.D.**

*Professor of Obstetrics and Diseases of Women in the Bellevue Hospital Medical College, &c.*

**THE SCIENCE AND ART OF MIDWIFERY.** Second Edition, with numerous Illustrations, 8vo, 18s.

**JOHN MACPHERSON, M.D.**

*Inspector-General of Hospitals H.M. Bengal Army (Retired).*

*Author of "Cholera in its Home," &c.*

**ANNALS OF CHOLERA FROM THE EARLIEST PERIODS TO THE YEAR 1817.** With a map. Demy 8vo, 7s. 6d.

**DR. V. MAGNAN.**

*Physician to St. Ann Asylum, Paris; Laureate of the Institute.*

**ON ALCOHOLISM, the Various Forms of Alcoholic Delirium and their Treatment.** Translated by W. S. GREENFIELD, M.D., M.R.C.P. 8vo, 7s. 6d.

---

A. COWLEY MALLEY, B.A., M.B., B.CH. T.C.D.

**PHOTO-MICROGRAPHY**; including a description of the Wet Collodion and Gelatino-Bromide Processes, together with the best methods of Mounting and Preparing Microscopic Objects for Photo-Micrography. Second Edition, with Photographs and Illustrations, crown 8vo, 7s. 6d. [Now ready.]

---

PATRICK MANSON, M.D., C.M.  
*Amoy, China.*

**THE FILARIA SANGUINIS HOMINIS; AND CERTAIN NEW FORMS OF PARASITIC DISEASE IN INDIA, CHINA, AND WARM COUNTRIES.** Illustrated with Plates and Charts. 8vo, 10s. 6d.

---

PROFESSOR MARTIN.

**MARTIN'S ATLAS OF OBSTETRICS AND GYNÆCOLOGY.** Edited by A. MARTIN, Docent in the University of Berlin. Translated and edited with additions by FANCOURT BARNES, M.D., M.R.C.P., Physician to the Chelsea Hospital for Women; Obstetric Physician to the Great Northern Hospital; and to the Royal Maternity Charity of London, &c. Medium 4to, Morocco half bound, 31s. 6d. net.

---

WILLIAM MARTINDALE, F.C.S.

*Late Examiner of the Pharmaceutical Society, and late Teacher of Pharmacy and Demonstrator of Materia Medica at University College.*

AND

W. WYNN WESTCOTT, M.B. LOND.

*Deputy Coroner for Central Middlesex.*

**THE EXTRA PHARMACOPŒIA of Unofficial Drugs** and Chemical and Pharmaceutical Preparations, with References to their Use abstracted from the Medical Journals and a Therapeutic Index of Diseases and Symptoms. Third Edition, revised with numerous additions, limp roan, med. 24mo, 7s. [Now ready.]

---

J. F. MEIGS, M.D.

*Consulting Physician to the Children's Hospital, Philadelphia.*

AND

W. PEPPER, M.D.

*Lecturer on Clinical Medicine in the University of Pennsylvania.*

**A PRACTICAL TREATISE ON THE DISEASES OF CHILDREN.** Seventh Edition, revised and enlarged, roy. 8vo, 28s.

---

Wm. JULIUS MICKLE, M.D., M.R.C.P. LOND.

*Member of the Medico-Psychological Association of Great Britain and Ireland; Member of the Clinical Society, London; Medical Superintendent, Grove Hall Asylum, London.*

**GENERAL PARALYSIS OF THE INSANE.** 8vo, 10s.

KENNETH W. MILLICAN, B.A. CANTAB., M.R.C.S.

**THE EVOLUTION OF MORBID GERMS: A Contribution to Transcendental Pathology.** Cr. 8vo, 3s. 6d.

E. A. MORSHEAD, M.R.C.S., L.R.C.P.

*Assistant to the Professor of Medicine in University College, London.*

**TABLES OF THE PHYSIOLOGICAL ACTION OF DRUGS.** Fcap. 8vo, 1s.

A. STANFORD MORTON, M.B., F.R.C.S. ED.

*Senior Assistant Surgeon, Royal South London Ophthalmic Hospital.*

**REFRACTION OF THE EYE: Its Diagnosis, and the Correction of its Errors, with Chapter on Keratoscopy.** Second edit., with Illustrations, small 8vo, 2s. 6d.

WILLIAM MURRELL, M.D., F.R.C.P.

*Lecturer on Materia Medica and Therapeutics at Westminster Hospital; Examiner in Materia Medica, University of Edinburgh.*

**WHAT TO DO IN CASES OF POISONING.** <sup>I.</sup> Fourth Edition, revised and enlarged, royal 32mo, 3s. 6d.

**NITRO-GLYCERINE AS A REMEDY FOR ANGINA PECTORIS.** <sup>II.</sup> Crown 8vo, 3s. 6d.

WILLIAM NEWMAN, M.D. LOND., F.R.C.S.

*Surgeon to the Stamford Infirmary.*

**SURGICAL CASES: Mainly from the Wards of the Stamford, Rutland, and General Infirmary,** 8vo, paper boards, 4s. 6d.

DR. FELIX von NIEMEYER.

*Late Professor of Pathology and Therapeutics; Director of the Medical Clinic of the University of Tübingen.*

**A TEXT-BOOK OF PRACTICAL MEDICINE, WITH PARTICULAR REFERENCE TO PHYSIOLOGY AND PATHOLOGICAL ANATOMY.** Translated from the Eighth German Edition, by special permission of the Author, by GEORGE H. HUMPHREY, M.D., and CHARLES E. HACKLEY, M.D., Revised Edition, 2 vols., large 8vo, 36s.



C. F. OLDHAM, M.R.C.S., L.R.C.P.

*Surgeon H.M. Indian Forces; late in Medical charge of the Dalhousie Sanitarium.*

**WHAT IS MALARIA?** and why is it most intense in hot climates? An explanation of the Nature and Cause of the so-called Marsh Poison, with the Principles to be observed for the Preservation of Health in Tropical Climates and Malarious Districts. Demy 8vo, 7s. 6d.

G. OLIVER, M.D., M.R.C.P.

**THE HARROGATE WATERS:** <sup>I.</sup> Data Chemical and Therapeutical, with notes on the Climate of Harrogate. Addressed to the Medical Profession. Crown 8vo, with Map of the Wells, 3s. 6d.

**ON BEDSIDE URINE TESTING:** <sup>II.</sup> including Quantitative Albumen and Sugar. Third edition, revised and enlarged, fcap. 8vo. [In the press.]

JOHN S. PARRY, M.D.

*Obstetrician to the Philadelphia Hospital, Vice-President of the Obstetrical and Pathological Societies of Philadelphia, &c.*

**EXTRA-UTERINE PREGNANCY;** Its Causes, Species, Pathological Anatomy, Clinical History, Diagnosis, Prognosis and Treatment. 8vo, 8s.

E. RANDOLPH PEASLEE, M.D., LL.D.

*Late Professor of Gynæcology in the Medical Department of Dartmouth College; President of the New York Academy of Medicine, &c., &c.*

**OVARIAN TUMOURS:** Their Pathology, Diagnosis, and Treatment, especially by Ovariectomy. Illustrations, roy. 8vo, 16s.

G. V. POORE, M.D., F.R.C.P.

*Professor of Medical Jurisprudence, University College; Assistant Physician to, and Physician in charge of the Throat Department of University College Hospital.*

**LECTURES ON THE PHYSICAL EXAMINATION OF THE MOUTH AND THROAT.** With an Appendix of Cases. 8vo, 3s. 6d.

R. DOUGLAS POWELL, M.D., F.R.C.P., M.R.C.S.

*Physician to the Middlesex Hospital, and Physician to the Hospital for Consumption and Diseases of the Chest at Brompton.*

**DISEASES OF THE LUNGS AND PLEURÆ.** Third Edition, rewritten and enlarged. With Illustrations, 8vo.

[In the press.]

AMBROSE L. RANNEY, A.M., M.D.

*Adjunct Professor of Anatomy in the University of New York, etc.*

**THE APPLIED ANATOMY OF THE NERVOUS SYSTEM**, being a study of this portion of the Human Body from a standpoint of its general interest and practical utility, designed for use as a Text-book and a Work of Reference. With 179 Illustrations, 8vo, 20s.

H. A. REEVES, F.R.C.S. ED.

*Senior Assistant Surgeon and Teacher of Practical Surgery at the London Hospital;  
Surgeon to the Royal Orthopædic Hospital, &c.*

**BODILY DEFORMITIES AND THEIR TREATMENT:**  
A Handbook of Practical Orthopædics. With numerous Illustrations, crown 8vo, 8s. 6d. [Now ready.  
*Lewis's Practical Series*].

RALPH RICHARDSON, M.A., M.D.

*Fellow of the College of Physicians, Edinburgh.*

**ON THE NATURE OF LIFE: An Introductory Chapter** to Pathology. Second Edition, revised and enlarged. Fcap. 4to, 10s. 6d.

W. RICHARDSON, M.A., M.D., M.R.C.P.

**REMARKS ON DIABETES, ESPECIALLY IN REFERENCE TO TREATMENT.** Demy 8vo, 4s. 6d.

SYDNEY RINGER, M.D.

*Professor of the Principles and Practice of Medicine in University College; Physician to, and Professor of Clinical Medicine in, University College Hospital.*

**A HANDBOOK OF THERAPEUTICS.** Tenth Edition, 8vo, 15s.

II.

**ON THE TEMPERATURE OF THE BODY AS A MEANS OF DIAGNOSIS AND PROGNOSIS IN PHTHISIS.** Second Edition, small 8vo, 2s. 6d.

FREDERICK T. ROBERTS, M.D., B.SC., F.R.C.P.

*Examiner in Medicine at the Royal College of Surgeons; Professor of Therapeutics in University College; Physician to University College Hospital; Physician to Brompton Consumption Hospital, &c.*

**A HANDBOOK OF THE THEORY AND PRACTICE OF MEDICINE.** Fifth Edition, with Illustrations, in one volume, large 8vo, 21s.

II.

**NOTES ON MATERIA MEDICA AND PHARMACY.** Fcap. 8vo, 7s. 6d. [Now ready.]

D. B. St. JOHN ROOSA, M.A., M.D.

*Professor of Diseases of the Eye and Ear in the University of the City of New York; Surgeon to the Manhattan Eye and Ear Hospital; Consulting Surgeon to the Brooklyn Eye and Ear Hospital, &c., &c.*

**A PRACTICAL TREATISE ON THE DISEASES OF THE EAR**, including the Anatomy of the Organ. Sixth Edition, Illustrated by wood engravings and chromo-lithographs, large 8vo.

[Just ready.

J. BURDON SANDERSON, M.D., LL.D., F.R.S.

*Jodrell Professor of Physiology in University College, London.*

**UNIVERSITY COLLEGE COURSE OF PRACTICAL EXERCISES IN PHYSIOLOGY.** With the co-operation of F. J. M. PAGE, B.Sc., F.C.S.; W. NORTH, B.A., F.C.S., and AUG. WALLER, M.D. Demy 8vo, 3s. 6d.

W. H. O. SANKEY, M.D. LOND., F.R.C.P.

*Late Lecturer on Mental Diseases, University College and School of Medicine for Women, London; Formerly Medical Superintendent (Female Department) of Hanwell Asylum; President of Medico-Psychological Society, &c.*

**LECTURES ON MENTAL DISEASE.** Second Edition, with coloured plates, 8vo, 12s. 6d.

[Now ready.

ALDER SMITH, M.B. LOND., F.R.C.S.

*Resident Medical Officer, Christ's Hospital, London.*

**RINGWORM: Its Diagnosis and Treatment.**

Third Edition, rewritten and enlarged. With Illustrations, fcap. 8vo.

[In the press.

J. LEWIS SMITH, M.D.

*Physician to the New York Infants' Hospital; Clinical Lecturer on Diseases of Children in Bellevue Hospital Medical College.*

**A TREATISE ON THE DISEASES OF INFANCY AND CHILDHOOD.** Fifth Edition, with Illustrations, large 8vo, 21s.

FRANCIS W. SMITH, M.B., B.S.

**THE LEAMINGTON WATERS; CHEMICALLY, THERAPEUTICALLY AND CLINICALLY CONSIDERED;** with observations on the climate of Leamington. With Illustrations, crown 8vo, 2s. 6d.

JAMES STARTIN, M.B., M.R.C.S.

*Surgeon and Joint Lecturer to St. John's Hospital for Diseases of the Skin.*

**LECTURES ON THE PARASITIC DISEASES OF THE SKIN. - VEGETOID AND ANIMAL.** With Illustrations, Crown 8vo, 3s. 6d.

HENRY R. SWANZY, A.M., M.B., F.R.C.S.I.

*Examiner in Ophthalmic Surgery, University of Dublin; Surgeon to the National Eye and Ear Infirmary, Dublin; Ophthalmic Surgeon at the Adelaide Hospital, Dublin.*

**HANDBOOK OF DISEASES OF THE EYE AND THEIR TREATMENT.** Illustrated with wood-engravings, colour tests, etc., large post 8vo, 10s. 6d.

[Now ready.



LEWIS A. STIMSON, B.A., M.D.

*Surgeon to the Presbyterian Hospital; Professor of Pathological Anatomy in the Medical Faculty of the University of the City of New York.*

**A MANUAL OF OPERATIVE SURGERY.** With three hundred and thirty-two Illustrations. Post 8vo, 10s. 6d.

HUGH OWEN THOMAS, M.R.C.S.

I.

**DISEASES OF THE HIP, KNEE, AND ANKLE JOINTS**, with their Deformities, treated by a new and efficient method. With an Introduction by RUSHTON PARKER, F.R.C.S, Lecturer on Surgery at the School of Medicine, Liverpool. Second Edition, 8vo, 25s.

II.

**CONTRIBUTIONS TO MEDICINE AND SURGERY:—**

PART I.—Intestinal Obstruction; with an Appendix on the Action of Remedies. 10s.

„ 2.—The Principles of the Treatment of Joint Disease, Inflammation, Anchylosis, Reduction of Joint Deformity, Bone Setting. 5s.

„ 5.—On Fractures of the Lower Jaw. 1s.

„ 8.—The Inhibition of Nerves by Drugs. Proof that Inhibitory Nerve-Fibres do not exist. 1s.

(Parts 3, 4, 6, 7, 9, 10, are expected shortly).

J. ASHBURTON THOMPSON, M.R.C.S.

*Late Surgeon at King's Cross to the Great Northern Railway Company.*

**FREE PHOSPHORUS IN MEDICINE WITH SPECIAL REFERENCE TO ITS USE IN NEURALGIA.** A contribution to Materia Medica and Therapeutics. An account of the History, Pharmaceutical Preparations, Dose, Internal Administration, and Therapeutic uses of Phosphorus; with a Complete Bibliography of this subject, referring to nearly 200 works upon it. Demy 8vo, 7s. 6d.

J. C. THOROWGOOD, M.D.

*Assistant Physician to the City of London Hospital for Diseases of the Chest.*

**THE CLIMATIC TREATMENT OF CONSUMPTION AND CHRONIC LUNG DISEASES.** Third Edition, post 8vo, 3s 6d.

EDWARD T. TIBBITS, M.D. LOND.

*Physician to the Bradford Infirmary; and to the Bradford Fever Hospital.*

**MEDICAL FASHIONS IN THE NINETEENTH CENTURY**, including a Sketch of Bacterio-Mania and the Battle of the Bacilli. Crown 8vo, 2s. 6d.

LAURENCE TURNBULL, M.D., PH.G.

*Aural Surgeon to Jefferson Medical College Hospital, &c., &c.*

**ARTIFICIAL ANÆSTHESIA: A Manual of Anæsthetic Agents, and their Employment in the Treatment of Disease.** Second Edition, with Illustrations, crown 8vo, 6s.

---

W. H. VAN BUREN, M.D., LL.D.

*Professor of Surgery in the Bellevue Hospital Medical College.*

**DISEASES OF THE RECTUM: And the Surgery of the Lower Bowel.** Second Edition, with Illustrations, 8vo, 14s.

---

RUDOLPH VIRCHOW, M.D.

*Professor in the University, and Member of the Academy of Sciences of Berlin, &c., &c.*

**INFECTION-DISEASES IN THE ARMY, Chiefly** Wound Fever, Typhoid, Dysentery, and Diphtheria. Translated from the German by JOHN JAMES, M.B., F.R.C.S. Fcap. 8vo, 1s. 6d.

---

ALFRED VOGEL, M.D.

*Professor of Clinical Medicine in the University of Dorpat, Russia.*

**A PRACTICAL TREATISE ON THE DISEASES OF CHILDREN.** Translated and Edited by H. RAPHAEL, M.D. From the Fourth German Edition, illustrated by six lithographic plates, part coloured, large 8vo, 18s.

---

A. DUNBAR WALKER, M.D., C.M.

**THE PARENT'S MEDICAL NOTE BOOK.** Oblong post 8vo, cloth, 1s. 6d.

---

W. SPENCER WATSON, F.R.C.S. ENG., B.M. LOND.

*Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital.*

I.  
**DISEASES OF THE NOSE AND ITS ACCESSORY CAVITIES.** Profusely Illustrated. Demy 8vo, 18s.

II.  
**EYEBALL-TENSION: Its Effects on the Sight and its Treatment.** With woodcuts, p. 8vo, 2s. 6d.

III.  
**ON ABSCESS AND TUMOURS OF THE ORBIT.** Post 8vo, 2s. 6d.

---

A. DE WATTEVILLE, M.A., M.D., B.SC., M.R.C.S.

*Physician in Charge of the Electro-therapeutical Department at St. Mary's Hospital.*

**A PRACTICAL INTRODUCTION TO MEDICAL ELECTRICITY.** Second Edition, re-written and enlarged, copiously Illustrated, 8vo, 9s. [Just published.]

---

FRANCIS H. WELCH, F.R.C.S.

*Surgeon Major, A.M.D.*

**ENTERIC FEVER: as Illustrated by Army Data at Home and Abroad, its Prevalence and Modifications, Ætiology, Pathology and Treatment.** 8vo, 5s. 6d. [Just published.]

---

DR. F. WINCKEL.

*Formerly Professor and Director of the Gynæcological Clinic at the University of Rostock.*

**THE PATHOLOGY AND TREATMENT OF CHILD-BED:** A Treatise for Physicians and Students. Translated from the Second German edition, with many additional notes by the Author, by J. R. CHADWICK, M.D., 8vo, 14s.

**EDWARD WOAKES, M.D. LOND.**

*Senior Aural Surgeon and Lecturer on Aural Surgery at the London Hospital; Senior Surgeon to the Hospital for Diseases of the Throat.*

**ON DEAFNESS, GIDDINESS AND NOISES IN THE HEAD.**

**VOL. I.—POST-NASAL CATARRH, AND DISEASES OF THE NOSE CAUSING DEAFNESS.** With Illustrations, cr. 8vo, 6s. 6d.

**VOL. II.—ON DEAFNESS, GIDDINESS AND NOISES IN THE HEAD.** Third Edition, with Illustrations, cr. 8vo. [*In preparation.*]

**E. T. WILSON, B.M. OXON., F.R.C.P. LOND.**

*Physician to the Cheltenham General Hospital and Dispensary.*

**DISINFECTANTS AND HOW TO USE THEM. In**  
Packets of one doz. price 1s.

**Clinical Charts For Temperature Observations, etc.**

Arranged by W. RIGDEN, M.R.C.S. 7s. per 100, or 1s. per dozen.

Each Chart is arranged for four weeks, and is ruled at the back for making notes of cases; they are convenient in size, and are suitable both for hospital and private practice.

**PERIODICAL WORKS PUBLISHED BY H. K. LEWIS.**

**THE NEW SYDENHAM SOCIETY'S PUBLICATIONS.** Annual Subscription, One Guinea.

(Report of the Society, with Complete List of Works and other information, gratis on application.)

**ARCHIVES OF PEDIATRICS.** A Monthly Journal, devoted to the Diseases of Infants and Children. Annual Subscription, 12s. 6d., post free.

**THE NEW YORK MEDICAL JOURNAL.** A Weekly Review of Medicine. Annual Subscription, One Guinea, post free.

**THE THERAPEUTIC GAZETTE.** A Monthly Journal, devoted to the Science of Pharmacology, and to the introduction of New Therapeutic Agents. Edited by Drs. H. C. Wood and R. M. Smith. Annual Subscription, 10s., post free.

**MEDICAL BULLETIN.** A Monthly Journal of Medicine and Surgery. Edited by Dr. J. V. Shoemaker. Annual Subscription, 5s.

**THE GLASGOW MEDICAL JOURNAL.** Published Monthly. Annual Subscription, 20s., post free. Single numbers, 2s. each.

**LIVERPOOL MEDICO-CHIRURGICAL JOURNAL,** including the Proceedings of the Liverpool Medical Institution. Published twice yearly, 3s. 6d. each.

**THE INDIAN MEDICAL JOURNAL.** A Journal of Medical and Sanitary Science specially devoted to the Interests of the Medical Services. Annual Subscription, 24s., post free.

**THE MIDLAND MEDICAL MISCELLANY AND PROVINCIAL MEDICAL JOURNAL.** Annual Subscription, 7s. 6d., post free.

**TRANSACTIONS OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA.** Volumes I to VI., now ready, 8vo, 10s. 6d. each.

**\*\* MR. LEWIS** has transactions with the leading publishing firms in America for the sale of his publications in that country. Arrangements are made in the interests of Authors either for sending a number of copies of their works to the United States, or having them reprinted there, as may be most advantageous.

Mr. Lewis's publications can be procured of any bookseller in any part of the world.











